CASE REPORT

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Aesthetic management of severely fluorosed incisors in an adolescent female

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Abstract

Background: Dental fluorosis is a condition of enamel hypomineralization due to the effects of excessive fluoride on ameloblasts during enamel formation. Delayed degradation of enamel matrix proteins or inhibited protein removal results in impaired and incomplete crystal growth, producing hypomineralized and porous enamel. Severely fluorosed teeth may undergo post-eruptive surface breakdown and post-eruptive dark brown to black staining.

Methods: A 13 year old girl presented with severely discoloured maxillary central incisors. Initial aesthetic management of these teeth was conservative, including in-office tooth whitening, microabrasion and take-home whitening.

Results: Dark brown to black staining of the teeth was reduced successfully without the need for gross mechanical preparation of the enamel. Further improvement of aesthetics was achieved with composite veneers.

Conclusions: Conservative treatment options such as tooth whitening and microabrasion can dramatically improve severely discoloured fluorosed teeth. This can provide a satisfactory interim outcome or minimize the removal of discoloured enamel and dentine prior to the provision of composite veneers. The use of casein phosphopeptide-amorphous calcium phosphate (CPP-ACP™) may enhance remineralization and decrease post-operative sensitivity following tooth whitening and microabrasion procedures in hypomineralized teeth.

Key words: Whitening, microabrasion, CPP-ACP, fluorosis, aesthetics.

Abbreviations and acronyms: CP = carbamide peroxide; CPP-ACP = casein phosphopeptide-amorphous calcium phosphate; FPM = first permanent molars; HP = hydrogen peroxide; PEB = post-eruptive surface breakdown.

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INTRODUCTION

Dental fluorosis is a condition of enamel hypomineralization due to the effects of excessive fluoride on ameloblasts during enamel formation. Interactions between developing enamel mineral, matrix and ameloblasts lead to the subsequent changes.[1,2] In fluorosed enamel, fluoride disturbs mineralization by decreasing free calcium ion concentrations in the mineralizing matrix, indirectly interfering with the proteinases which degrade matrix proteins during the maturation phase of amelogenesis.[1,3] This results in delayed enzymatic degradation of enamel matrix proteins or inhibited removal of degraded enamel matrix proteins.[1] The fluoride-induced retention of enamel matrix proteins such as proline-rich amelogenins, ameloblastins, tuftelins, enamelins and high molecular weight sulphated proteins, is responsible for impaired and incomplete crystal growth.[1,4] Poor interlocking of crystals caused by peri-prismatic gaps and greater intercrystalline spaces occupied by water and protein account for the increased porosity of fluorosed enamel, and the subsequent optical and physical changes.[1,4]

Chemical aspects of dental fluorosis

Fluoride levels in developing enamel are directly related to plasma fluoride levels, and the fluoride content of fluorosed enamel prior to tooth eruption represents the fluoride acquired during the early formative stages of tooth development.[1] Fluorosed enamel has higher fluoride levels and greater protein content than normal enamel.[2,5-7] Normal enamel protein content ranges from 0.07 to 0.14 per cent, while fluorosed enamel protein content ranges from 0.03 to 0.56 per cent.[2]

The high fluoride concentrations in outermost enamel decrease markedly to a depth of approximately 100µm, thereafter levelling out in the innermost enamel.[5,6] Fluoride concentrations at specific depths within fluorosed enamel increase with increasing severity of fluorosis.[5,6] Extremely high concentrations (up to 4000ppm F) can be found in the outermost enamel of unerupted severely fluorosed teeth (Thylstrup & Fejerskov Index > 6).[5] Due to post-eruptive uptake of

fluoride from the oral environment, the outermost layer of erupted fluorosed enamel may exceed 5000ppm F.[6] The clinical relevance of increased fluoride levels (as found in fluorosed enamel) in caries reduction is unreported, and the presence of increased enamel fluoride in communities with water fluoridation should not be used as an argument for the existence of beneficial pre-eruptive effects of fluoride.

Clinical features of dental fluorosis

In its mildest forms, enamel fluorosis appears as loss of marginal translucency, poorly demarcated opacities, faint white flecks, spots or striations.[4] The white striations reflect accentuated striae of Retzius and von Ebner lines. With increasing severity, white flecks or striations enlarge and may merge.[4] The classical appearance of fluorosis is characterized by banding following the developmental lines of enamel and by substantial symmetry on homologous teeth.[8] In severely fluorosed teeth, hypomineralization extends towards the dentinal-enamel junction,[5] and may be subject to extensive post-eruptive surface breakdown (PEB) and post-eruptive dark brown to black staining.[1,3]

Risk periods

The effects of fluoride on enamel formation depend on the total fluoride intake, timing and duration of fluoride exposure.[1,9,10] There is no broadly identifiable threshold dose below which the effect of fluoride on enamel will not manifest clinically and a linear relationship exists between total fluoride dose and subsequent enamel fluorosis.[1] The most critical period for susceptibility to fluorosis in human maxillary central incisors corresponds to the first 24 months of age,[9] or more precisely, the age range of 15 to 24 months for females and 21 to 30 months for males.[11] A meta-analysis of available data confirmed that the duration of total cumulative fluoride exposure during the period of amelogenesis, rather than during specific risk periods, explains the development of fluorosis in the permanent maxillary central incisors.[12]

Implications for management

The main consequence of dental fluorosis is compromised aesthetics, from white spots, striations or opacities at lower severity, to post-eruptive dark brown to black staining with increasing severity of fluorosis.[13-15] With increasing severity, PEB and exposure of subsurface hypomineralized enamel, fluorosis can increase caries risk.[16,17]

CASE REPORT

Presentation and clinical examination

A 13 year old girl was referred for evaluation and management of a "hypoplastic dentition" following the extraction of teeth 75, 85 and 46 at the Emergency Department of the Royal Dental Hospital of Melbourne. The patient had migrated from Ethiopia eight months prior to presentation and was concerned about the severe discolouration of her maxillary central incisors (Fig 1).

Fig 1. Labial view showing the severely discoloured maxillary central incisors.

The patient was born at full term following an uneventful pregnancy in the Wonji Shoa Sugar Estate in the Ethiopian Rift Valley. She resided in this area until 4 years of age, then moved to the Ethiopian capital Addis Ababa where she lived until migrating to Australia. Her medical history was non-contributory. There was no history of trauma. She was the oldest of three siblings and there was no family history of the dental condition of concern. Her mother was born and raised in a different part of Ethiopia and her siblings aged 7 and 9 were born and raised in Melbourne. Prior to arrival in Melbourne, the patient had not received any dental care nor had she been exposed to any fluoride-containing dental products or supplements. A wooden stick was used for tooth cleaning purposes while living in Ethiopia.

Intra-oral examination revealed a generalized hypomineralized and severely carious permanent dentition with edentulous spaces in the mandible. The mandibular central incisors, maxillary and mandibular lateral incisors, canines, premolars and second molars appeared chalky white. The maxillary central incisors and all remaining first permanent molars (FPM) had post-eruptive dark brown to black discolouration. Oral hygiene was poor and generalized plaque and supra-gingival and subgingival calculus deposits were present. The gingival margins were rolled, erythematous and oedematous. Radiographic examination showed dental caries, agenesis of teeth 35 and 45, and a peg-shaped 22. All developing third molars were present. Cephalometric analysis indicated a Class I skeletal pattern and a normal-angle profile. The maxillary and mandibular incisors were slightly proclined with respect to the skeletal base.

The symmetrical and bilateral pattern of enamel hypomineralization and the presence of a contributory fluoride history suggest a putative diagnosis of dental fluorosis. The aetiology is attributed to early exposure

to high concentrations of fluoride ingested from drinking water. Between 1977 and 1985, the fluoride content of drinking water collected from wells in villages in the Ethiopian Rift Valley ranged from 1.2ppm to 36ppm.[18] Due to the severely affected FPMs and central incisors, there is possible overlap of molar-incisor-hypomineralization. Although differential diagnosis of enamel opacities is an incomplete science, the suggested diagnosis is plausible.[19]

A problem list was developed as follows: severely hypomineralized dentition, dental caries, PEB and discolouration of maxillary central incisors and all remaining FPMs, cariogenic and erosive diet, poor oral hygiene and gingivitis, agenesis of 35 and 45, peg-shaped 22, proclined incisors and increased overjet.

Treatment

Various treatment options addressing the patient's presenting complaint were discussed with the patient and her mother. These included the use of in-office and take-home tooth whitening systems, enamel micro-abrasion and composite veneers. Porcelain veneers were contraindicated at this time due to the patient's immature gingival contour and pulpal size. In view of these reasons, the patient and her mother chose the more conservative treatment options of tooth whitening and microabrasion. It was agreed to use tooth whitening techniques and enamel microabrasion to improve the appearance of the teeth initially and to review the effectiveness and need for further treatment after three months. A comprehensive treatment plan was developed including preventive and periodontal therapy, restoration of carious lesions, full coverage cast crowns, a mandibular partial denture, interim reconstruction of the peg-shaped 22 and a mouthguard. The treatment objectives were to improve the patient's oral health awareness and to provide her with a healthy, functional and aesthetic dentition. This is likely to improve her self-esteem and contribute to healthy psychological development.

A preventive oral health regimen began with scaling and root planing, oral hygiene instruction on brushing and flossing, and advice on dietary modification. A 0.05% (w/w) neutral sodium fluoride mouthrinse (Colgate Neutrafluor 220, Colgate-Palmolive Pty Ltd, NSW, Australia) was prescribed for daily use. Remineralization therapy was instituted with daily use of 10% (w/v) casein phosphopeptide-amorphous calcium phosphate (CPP-ACP™) (Tooth Mousse™, GC Corp, Tokyo, Japan). Fissure sealants (Conseal, SDI Ltd, Vic, Australia) were placed on all newly-erupted premolars and second molars.

Small carious lesions were restored with composite (Filtek Supreme, 3M ESPE Dental Products, St. Paul, MN, USA). Teeth 26 and 36 exhibited extensive PEB and were restored with full coverage cast gold crowns. A survey crown was fabricated for tooth 36 prior to fabrication of the chrome-cobalt mandibular partial denture. Interim reconstruction of 22 was achieved with composite.

Fig 2. Intra-oral view of the maxillary central incisors after two treatments with GC Tion™ in-office tooth whitening.

Dark brown to black staining of the maxillary central incisors was treated initially with an in-office tooth whitening system GC TiON™ (GC America Inc, Alsip, IL, USA). This system features a titanium dioxide photo-initiator, putatively enabling increased whitening effect with lower hydrogen peroxide (HP) concentration (20 per cent) than other in-office tooth whitening systems. The maxillary central incisors were isolated with rubber dam and gingival protector (TiON™, GC America Inc, Alsip, IL, USA) was applied around the gingival cuff to prevent gel leakage. The GC TiON™ "reactor" was applied to the surfaces to be whitened; the "Whitening Gel" was then mixed with the "Whitening Liquid" and dispensed onto the isolated teeth. Visible light irradiation was applied with the Demetron LC light curing unit (Kerr Corp, CA, USA) for one minute per tooth. The gel was then removed with gauze and the procedure was repeated thrice. Immediately after treatment, Tooth Mousse™ was applied to the teeth to enhance remineralization and decrease postoperative sensitivity. One month later, the patient received a second treatment with the same system; a marked reduction in staining intensity was evident. However, despite marked improvement, some brown staining persisted and the patient requested further treatment (Fig 2).

One month later, the teeth were microabraded with Prema™ microabrasion paste (Premier Dental Products Co, Plymouth Meeting, PA, USA) following the technique of the UK National Clinical Guidelines in Paediatric Dentistry.[20] The maxillary central incisors were isolated with rubber dam and Vaseline (Elida Faberge, Vienna, Austria) was applied around the cervical portion of the teeth to prevent leakage of hydrochloric acid. The paste was applied to the teeth with the mandrel and mandrel tip provided, in a slow-speed rotary handpiece, for five seconds. A high-volume aspirator was placed on the lingual surfaces of the teeth being treated to minimize paste splattering. The tooth surface was then washed for five seconds directly into the aspirator. Ten "five second" applications of the microabrasion paste were delivered

Fig 3. Intra-oral view of the maxillary central incisors after two treatments with GC Tion™ in-office tooth whitening, microabrasion and take-home whitening gel (15% carbamide peroxide) for a month.

to each tooth. Immediately after treatment, Tooth Mousse™ was applied to enhance remineralization. On review two weeks later, the stained area was reduced in size but small brown areas persisted and the patient requested further treatment.

The patient was provided with custom-fabricated trays and take-home whitening gel containing 15% carbamide peroxide (CP) as the active ingredient (Opalescence, Ultradent, Utah, USA), and instructed on gel usage. Due to her hypomineralized dentition, precautions were taken to prevent excessive enamel demineralization and the patient was instructed to alternate use of the take-home gel with Tooth Mousse™ for each night of the week. To prevent gel overuse, she was given only one syringe of gel at a time and her mother closely supervised the procedures.

Review

The patient was reviewed after one month and the reduction in brown stain intensity was remarkable (Fig 3). The patient continued using the take-home whitening system for three months. No tooth sensitivity or gingival irritation was reported. Vitality testing of the maxillary central incisors undertaken at each monthly review appointment indicated no change in pulpal status. After three months using the take-home whitening system and Tooth Mousse™, the patient was pleased with the aesthetic improvement but requested further treatment to improve the uniformity of tooth colour. This was achieved with direct composite veneers.

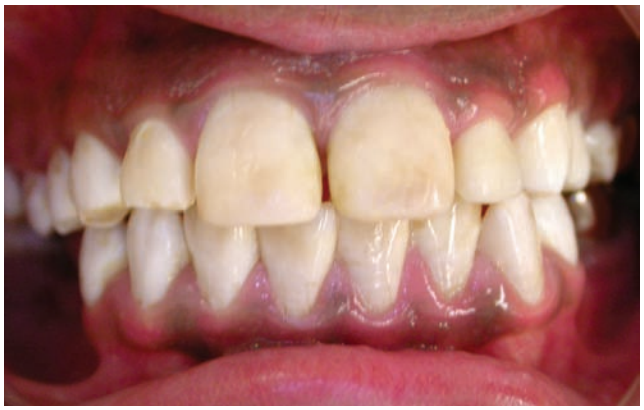

Fig 4. Post-treatment intra-oral view illustrating the improved gingival condition, improved aesthetics following tooth whitening and microabrasion, composite veneers, and restoration of the peg lateral incisor.

Fig 5. Post-treatment intra-oral view illustrating the improved gingival condition and the full coverage cast crown on 26.

Post-treatment photographs showed the improved gingival condition and confirmed the treatment objectives were achieved (Figs 4-6). The patient was delighted with the aesthetic improvement and reported smiling more and feeling more confident amongst her peers. Compliance with her preventive oral health regimen has remained excellent. Her mandibular partial denture is comfortable and functional.

DISCUSSION

The clinical management of tooth discolouration aims to produce an acceptable cosmetic result as conservatively as possible.[14] Conservative treatment options such as tooth whitening and/or microabrasion

Fig 6. Post-treatment intra-oral view illustrating restoration of 36 with a cast survey crown and restoration of edentulous spaces with a chrome cobalt partial denture.

can produce dramatic improvements in brown and yellow discolouration, providing a satisfactory interim result before more invasive procedures are considered, if necessary.[14,21-25] Although safety concerns including localized adverse effects and potential for toxicity surround the use of peroxide-based whitening products in children and adolescents,[26] it was considered in the present case that the benefits of improved aesthetics outweighed the risks; although measures were put in place to limit the dose, duration and frequency of exposure to the whitening product. The initial use of GC TiON™ to achieve gross changes in-office was an attempt to obviate or reduce the need for lengthy take-home whitening procedures at a stage when patient compliance was uncertain. Although the latter was eventually used, the patient was closely monitored to prevent gel over-use and ensure early diagnosis of localized adverse effects, if any.

Contemporary tooth whitening systems are based on HP or CP. Hydrogen peroxide is an oxidizing agent which breaks down into free radicals, eventually combining to form oxygen and water. Chromophores are complex organic molecules possessing extended conjugated chains of single or double bonds and are responsible for the tooth discolouration.[27] The HP oxidizes, carboxylates and lightens chromophores, particularly within the dentine.[27] Carbamide peroxide dissociates to form HP and urea on contact with water. The clinical efficacy of products containing CP is similar to that of HP containing products of equivalent or similar HP content and delivered using similar techniques and formulations.[27]

Most current in-office whitening systems are based on HP solutions of 25 to 35 per cent. Most take-home whitening systems are based on CP solutions of 5 to 22 per cent, which equate to 1.5 to 6.6 per cent of available HP. In addition to patient factors such as the initial tooth colour, type of intrinsic stain and subject age, the efficacy of tooth whitening is determined by the concentration of HP or CP used, and the duration of application.[27] The relationship between the concentration of HP and the number of application times required to produce an optimal shade outcome is exponential, and higher concentrations of HP need fewer applications to produce a whitening effect.[27,28] Tooth sensitivity is a common adverse effect of vital bleaching as peroxide can penetrate enamel and dentine and enter the pulp chamber.[29] The degree of penetration increases with increasing concentrations of HP and hypomineralized enamel may allow greater penetration of HP.[29] The patient in the present case did not report sensitivity or gingival irritation with the in-office whitening system containing 20% HP, or with the take-home whitening system containing 15% CP. This may have been because of the relatively low concentrations of HP and CP used, or the conscientious use of remineralizing products such as CPP-ACP™ and fluoride mouthrinse. Topical fluorides may be used for post-eruptive remineralization of fluorosed teeth as fluorosis is a condition of enamel hypomineralization due to pre-eruptive insult to ameloblasts.

Enamel microabrasion corrects surface enamel hypomineralization and colouration defects by removing superficial enamel.[14,30] Microabrasion may be achieved by the use of up to 18% hydrochloric acid and pumice slurry, producing dramatic improvement in brown and yellow discolouration, and removing approximately 100µm of enamel in 10 applications.[20-25] The Prema™ microabrasion paste contains 10% hydrochloric acid in a prophylaxis paste of distilled water, silicon dioxide and silicon carbide. The Opalustre™ microabrasion slurry (Ultradent Products Inc, Utah, USA) contains 6.6% hydrochloric acid and silicon carbide microparticles.

If the discoloured defect is superficial and micro-abrasion exposes underlying enamel of normal quality, the tooth acquires a glassy lustrous quality due to changes in the intrinsic properties of enamel following simultaneous abrasion and erosion of the surface.[14] This is known as the "abrosion effect".[21] In fluorosed enamel where hypomineralization may extend from close to the surface toward the dentinal-enamel junction, the subsurface hypomineralized enamel may become exposed. Therefore, if a satisfactory clinical outcome is not achieved after 10 "five second" applications, an alternative treatment option should be selected.[20] In this case report, microabrasion was used successfully to reduce the area and intensity of the black and brown stain. However, small brown areas persisted and it was decided to cease microabrasion in order to avoid exposing more subsurface hypo-mineralized enamel. The patient was offered the alternative of a take-home whitening system.

CONCLUSION

This report illustrates a clinical case in which a combination of microabrasion, in-office tooth whitening and take-home tooth whitening techniques improved the appearance of a young patient's discoloured anterior teeth. Dark brown to black staining outside the range of any available shade guide was reduced successfully without the need for gross mechanical tooth preparation. Further improvement in aesthetics was achieved readily with composite veneers.

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