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CLINICAL SECTION

Idiopathic gingival hyperplasia and orthodontic treatment: a case report

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Abstract

There are many reasons for gingival hyperplasia. Mostly, proper oral hygiene is sufficient to achieve normal healthy gingiva. In some situations, however, gingival hyperplasia is drug-induced or can be a manifestation of a genetic disorder. In the latter, it may exist as an isolated abnormality or as part of a syndrome. If orthodontic treatment is needed in patients with gingival hyperplasia, both orthodontic and periodontal aspects need to be considered. Extreme hereditary gingival fibromatosis was periodontally treated, by removal of all gingival excess using flaps and gingivectomies. After a follow-up period, the orthodontic treatment started with fixed appliances. Monthly periodontal check-ups (scaling and polishing) were scheduled to control the gingival inflammation. After the orthodontic treatment, permanent retention was applied, once more followed by a complete gingivectomy in both maxilla and mandible. One of the most important keys to successful treatment of hyperplasia patients is the cooperation between the periodontist and the orthodontist.

Index words: idiopathic gingival hyperplasia, orthodontics, periodontal surgery.

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Introduction

Gingivitis is the most common inflammatory reaction of the gingiva and is caused by bacterial plaque colonization on tooth surfaces and subsequent invasion of the micro-organisms into the gingival sulcus. Affected gingival tissues are oedematous, soft in consistency, and may bleed when gently probed.[1,2] In some pathological conditions, gingivitis caused by plaque accumulation can be more severe. Erythematous gingival enlargement is, for example, frequently found in subjects with uncontrolled diabetes. During puberty and pregnancy, hyperplasia of the gingival tissues, also known as puberty or pregnancy epulides, may be due to poor oral hygiene, inadequate nutrition, or systemic hormonal stimulation.[3–5]

Gingival overgrowth occurs in about 50 per cent of persons taking phenytoin (Dilantin®).[6–10] During the past few years, the list of the medications causing a similar gingival overgrowth condition has increased. The gingival tissues are affected through different mechanisms.[6,11–15] Nevertheless, the hyperplasia involves the gingival margin with extension to the inter-dental papilla. The lesion may involve the inter-proximal spaces, and become so extensive that the teeth are displaced and their crowns covered.

Gingival enlargements are also seen in several blood dyscrasias. This form of gingival dysplasia is seen in acute monocytic, lymphocytic, or myelocytic leukaemia. The gingival tissues are enlarged, oedematous, soft, and tender to touch, with a tendency to bleed easily. The gingiva is bluish-red with some pseudo-membrane plaques covering ulcerated surfaces.[16] Thrombocytopenia and thrombocytopathy also can cause gingival enlargement and spontaneous bleeding. In some conditions, gingival enlargement can progress rapidly into destructive periodontal diseases, as a result of the altered immune response of the gingiva to the bacterial plaque.

A slowly progressive fibrous enlargement of the maxillary and mandibular gingiva is a feature of idiopathic fibrous hyperplasia of the gingiva. Characteristically, this massive gingival enlargement appears to cover the tooth surfaces and displace the teeth, whilst the cause of the disease is unknown, there appears to be a genetic predisposition.[17,18] On histopathological examinations, the diseased tissue contains an increased amount of mature collagenous connective tissue and a mild hyperplasia of the overlying epithelium. Most idiopathic

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hyperplasias are probably caused by a genetic disorder and should, therefore, not be called idiopathic, as the cause is 'a genetic disorder'. However, for the moment it has not been proven on which gene this genetic disorder is located, so all hyperplasia without cause should still be described as idiopathic, even if the hyperplasia is seen in the same family without any other symptoms. Table 1 gives an overview of the causes of gingival hyperplasia.

Gingival fibromatosis may exist as an isolated abnormality or as part of a syndrome.[19,20] As an isolated finding, it is mostly sporadic, but an autosomal dominant inheritance pattern is also possible. Rarely, autosomal recessive inheritance is found. The gingival enlargement may delay the eruption of teeth and make cleaning of the teeth virtually impossible. Involvement of the molar regions only has been suggested to be a partial manifestation of the condition. While the gingiva may be the only tissue involved, other cases display gingival fibromatosis in association with hypertrichosis, and/or mental retardation, and/or epilepsy. The association of gingival fibromatosis and corneal dystrophy is recognized as an autosomal dominant trait known as the Rutherfurd syndrome.[19] Cross syndrome is, almost certainly, an autosomal recessive disorder characterized by gingival fibromatosis, microphthalmia, mental retardation, and pigmentary defects.[19] Ramon syndrome is another, probably autosomal recessive, condition involving gingival fibromatosis, as well as hypertrichosis, mental retardation, delayed development, epilepsy and cherubism.[21] Laband syndrome features gingival fibromatosis, syndactily, nose and ear abnormalities, and hypoplasia of the nails and terminal phalanges. Table 2 gives an overview of syndrome related gingival overgrowth.

Table 1 Causes of gingival hyperplasia

Visuals aspect	Cause
Gingivitis	Bacterial plaque[1,2]
More severe gingivitis	Bacterial plaque and uncontrolled diabetes[3–5]
Puberty or pregnancy epulides	Bacterial plaque and puberty or pregnancy[3–5]
Drug-induced gingival over-growth	Bacterial plaque and medicine (phenytoin Dilantin)[6–10]
Enlarged, oedematous, soft and tender, easily bleeding gingivitis	Acute monocytic, lymphocytic, or myelocytic leukaemia[16]
Gingival enlargement and spontaneous bleeding	Thrombocytopenia and thrombocytopathy
Part of a syndrome	Cfr Table 2

An extreme hereditary fibromatosis

A 7-year-old Caucasian boy presented complaining of poor aesthetics due to gingival overgrowth, delayed eruption of the permanent teeth, and a reduced number of deciduous teeth. His medical history involved a cyst of the head at the age of 6 months and hereditary gingival overgrowth (fibromatosis). The cyst was not related to the fibromatosis and disappeared a few months later. There is a family history of hereditary gingival overgrowth as his father presents the same condition. There were no other significant medical findings.

At the age of 15 years and 11 months, the first orthodontic records were taken. At that time, all the permanent teeth had erupted to the occlusal plane. Before any orthodontic treatment, a gingivectomy was performed in the upper jaw.

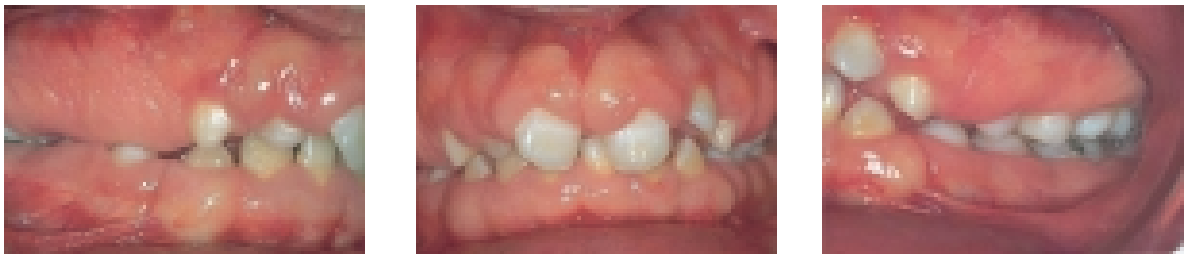
Diagnostic features at the time of presentation prior to the start of the orthodontic treatment

1. Besides the hereditary gingival overgrowth, the patient has a good general health (Figure 1).
2. Convex profile, high stomion to incisors ratio, anterior and posterior gummy smile of more than 10 mm, pronounced plica mentalis (Figure 2).
3. Class II jaw relationship (slight protrusion of the maxilla and slight retroposition of the mandible).

Table 2 An overview of gingival overgrowth related with a syndrome

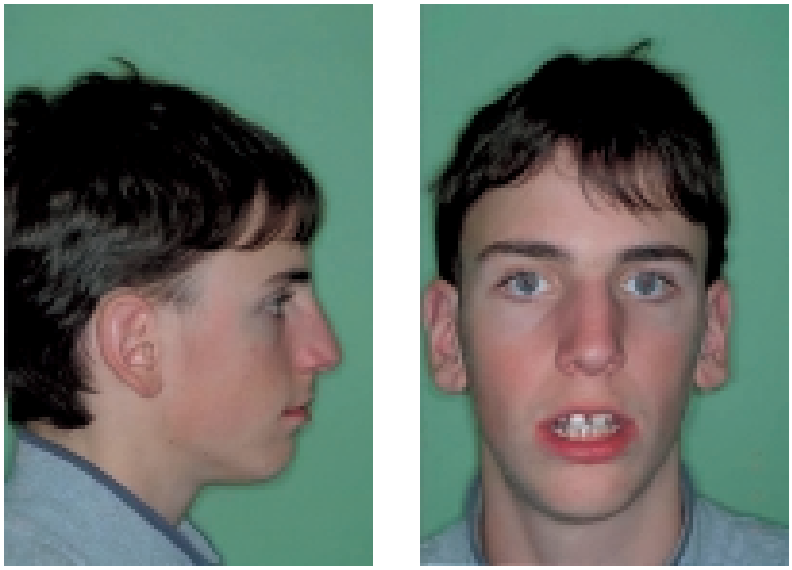
Syndrome	Symptoms other than gingival overgrowth	Autosomal dominance of recessive
Rutherfurd Syndrome[19]	Corneal dystrophy	Dominant
Cross Syndrome[19]	Microphthalmia, mental retardation, pigmentary defects	Recessive
Ramon Syndrome[21]	Hypertrichosis, mental retardation, delayed development, epilepsy, cherubism	Recessive
Laband Syndrome	Syndactily, nose and ear abnormalities, hyperplasia of the nails and terminal phalanges	Dominant

4. An asymmetrical molar occlusion of half a unit disto occlusion on the right and neutral occlusion on the left, increased overjet and overbite, deep bite with palatal gingival trauma.
5. Over-eruption of the lower incisors, retained deciduous upper canines, malposition of upper lateral incisors
6. No functional problems are noted.

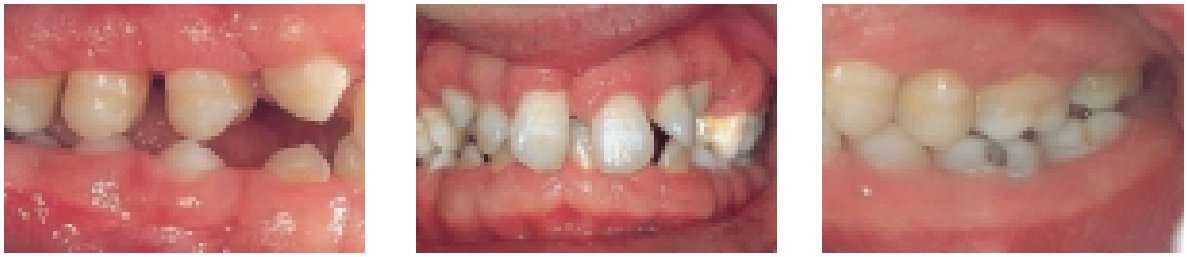


(a) (b) (c)

Fig. 1 (a–c) Orthodontic records prior to periodontal surgery.



(a) (b)



(c) (d) (e)

Fig. 2 (a–e) Orthodontic records after periodontal surgery.

Sequence of treatment

Pronounced gingival fibromatosis was present at the age of 7 years, completely covering the occlusal planes of all the deciduous teeth present. The patient was advised to wait for further dental development and was instructed how to maintain good oral hygiene.

Because the patient didn't have any medical problems, no diagnostic tests were performed to exclude the other possibilities of gingival enlargement as described in the introduction. The same can be said for the father.

At the age of 9 years and 9 months, the lower deciduous incisors were extracted to facilitate the emergence of the lower permanent incisors. The patient was followed yearly, and at the age of 12 years and 10 months, the first right upper deciduous molar and both first lower deciduous molars were extracted. At the age of 13 years and 7 months the deciduous lower canines were extracted, followed by both deciduous upper second molars and the lower left deciduous second molar, 7 months later. At this age the gingival hyperplasia totally covered the occlusal surfaces of the posterior teeth. Bleeding on probing was present in all areas. Numerous probing depths were measured from 6 to 10 mm. Moderate levels of plaque and calculus were found.

At the age of 15 years and 2 months the hypertrophic gingiva of the lower incisors was surgically removed by means of a gingivectomy, uncovering well-preserved normal teeth. The tissue was histologically analysed. The epithelium showed a reticular acanthosis with normal development, while the lamina propria consisted of extended fibrosis with focally little bone lamellas between the blood vessels. There was no evidence of dysplasia. The tissue was histologically similar to a fibrous epulis. The absence of any other medical disorders, like blood dyscrasias, lysosomal storage diseases or drug use and the presence of a similar situation in the father, suggests a genetic and hereditary form of gingival fibromatosis.

The patient was followed yearly in the periodontic department with a joint consultation of the general dentist, periodontist and orthodontist. At the age of 15, the patient was referred to the department of orthodontics as the tooth development was nearly at its final stage.

At the age of 15 years and 10 months an orthopantomogram was taken to evaluate the anterior teeth and to determine if they had 'erupted' (descended) to their normal position, as this was difficult to determine intra-orally because the teeth were still completely covered by the gingiva (Figure 3). Because this was achieved, the hypertrophic gingiva was surgically removed in the upper jaw posteriorly and anteriorly, with high risk of recurrence, uncovering normal teeth. Orthodontic records were taken 1 month later. At the age of 16 years, upper molar bands were cemented and a palatal bar was engaged to maintain the current occlusion. A Ricketts utility arch was used for intrusion of the upper central incisors. Three months later, the upper lateral incisors were bracketed in order to close the frontal diastema and start the alignment of the front teeth. Two months later, an upper biteplate was placed to enable lower fixed appliance to be bonded. Before placement of the lower fixed appliance, a gingivectomy was performed on the lower canines and premolars to uncover the crowns of these teeth. After a healing period of 2 months, the lower brackets were placed. Sectional archwires were also placed in the upper buccal segments. At the age of 17 years and 3 months the papillae of the upper lateral incisors were removed again to facilitate the closure of the diastema. Three months later, the lower right second molar was uncovered and after 2 months healing time the bracket was placed.

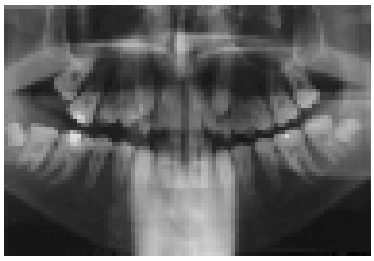


Fig. 3 OPG to evaluate the anterior teeth and to determine if they had erupted to their normal position.

At the age of 18 years and 6 months, the fixed appliances were removed and permanent retention was placed (Figure 4). The periodontal follow-up was performed monthly, during the orthodontic treatment. All teeth were polished and oral hygiene was reinforced. During treatment the gingival fibromatosis recurred and, to create a more stable result, gingivectomies were performed in the months following the start of the retention (Figure 5).

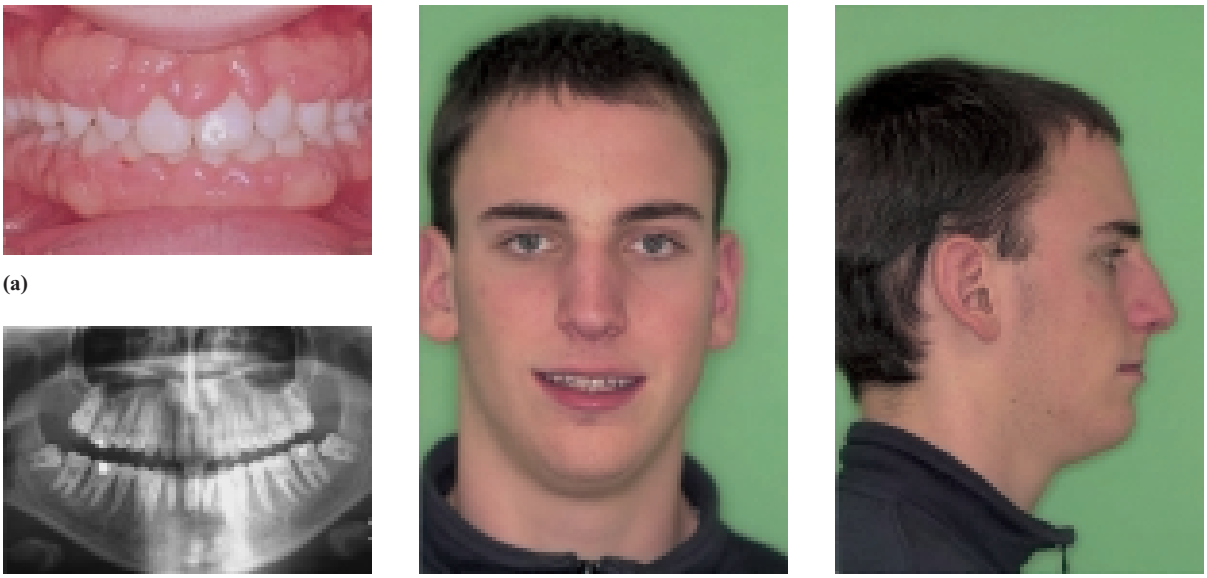
From the first day the patient visited the clinic, he was treated by the different disciplines within dentistry. All extractions where performed by a general dentist. One and the same person at the department of orthodontics performed the orthodontic treatment. The periodontal examinations and gingivectomies where always performed by the same periodontist, creating a good work-

ing environment for patient, orthodontist, and periodontist.

Discussion

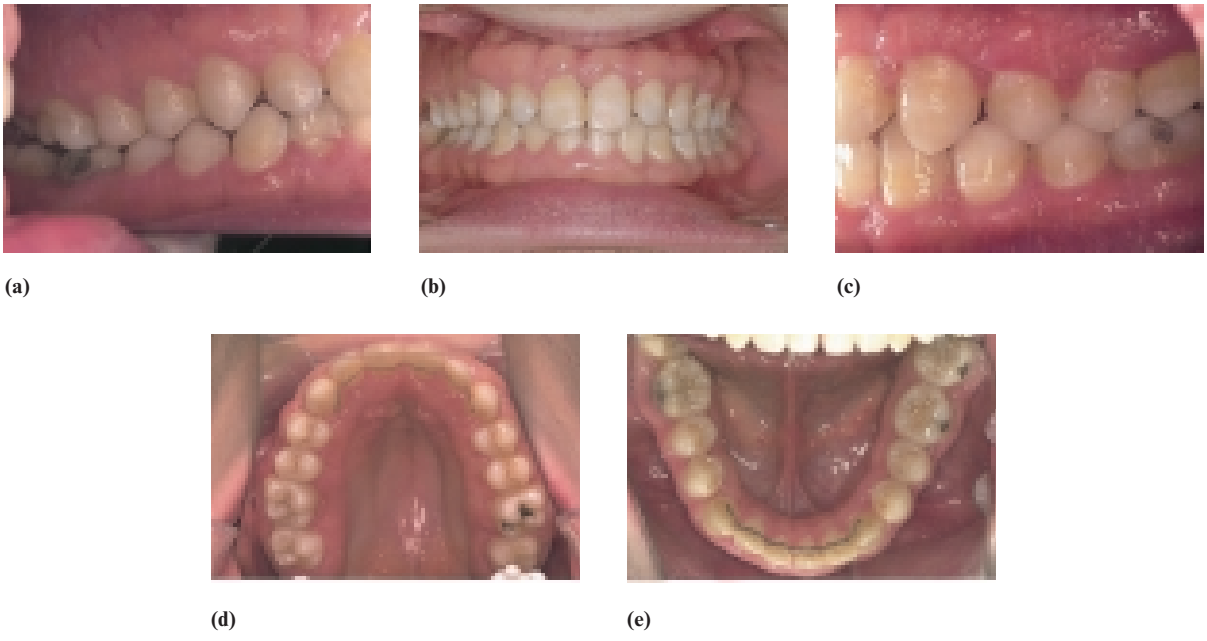
As there are few reports on the orthodontic treatment in patients with hereditary gingival hypertrophy no standard protocol can be found. In fact, following discussion with the general practitioner, most orthodontic treatments are discontinued in patients with drug-induced gingival hypertrophy.

To facilitate bracket positioning, a total gingivectomy was considered to be the first step in the orthodontic treatment. A period of 2 months should be allowed to



(a) (b) (c) (d)

Fig. 4 (a–d) Orthodontic records after orthodontic treatment prior to periodontal surgery.



(a) (b) (c) (d) (e)

Fig. 5 (a–e) Orthodontic records after orthodontic treatment and after periodontal surgery.

evaluate healing and the stability of the gingivectomy, as well as the oral hygiene of the patient. The first gingivectomy to evaluate the stability was performed in the lower jaw, although the treatment with fixed appliances started in the upper jaw. Luckily the hypertrophy didn't recur before bracket placement in the lower anterior region. Additionally, the gingivectomy should only be performed in the areas where the orthodontic treatment is started and this to reduce the chance of recurrence of the gingival hypertrophy. When the treatment in started in another area of the dentition, a gingivectomy was previously carried out providing easy accessible teeth for bracket placement.

Orthodontic treatment started with fixed appliances in the upper arch. At every orthodontic visit, the wires were removed and a periodontal examination followed. Each time the teeth were scaled and polished. At the beginning of the treatment the gingival hypertrophy recurred a little, but remained stable during the rest of the treatment. It is our opinion that the brackets and wires provide a mechanical barrier to the recurrence of the gingival proliferation.

At bracket removal, a total gingivectomy was planned. Permanent fixed retention should be considered in these patients not only as a standard retention after orthodontic treatment, but also as precaution against re-opening of inter-dental spaces due to gingival hypertrophy. Although total treatment time was not excessively long the authors believe that the gingival overgrowth did significantly interfere with general tooth movement. Waiting for 2 months after each gingivectomy and due to the gingival thickness encountered in this patient, which acted as a physical barrier to tooth movement, resulted in a somewhat longer treatment time than initially expected. Perhaps the treatment time is not longer in other patients with this problem, but as the idiopathic hyperplasia is rare, no other information is available at this moment.

Multiple surgical procedures, monthly periodontal examinations and prophylaxis are an essential part of the treatment protocol. However, additional effort is needed from the patient. Hopefully, the final treatment result makes it all worthwhile.

Conclusion

A good occlusal and aesthetic result was achieved, and took 2 years and 6 months. Recurrence of the gingival overgrowth was minimal due to the good oral hygiene achieved by monthly examinations, cleaning, and oral hygiene instructions at the Department of Periodontics. After treatment, regular recalls are necessary in order to evaluate oral hygiene, and the stability of the orthodontic and periodontal treatment.

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