The Report of One Case of the Developmental Gingival Hyperplasia

WANG Da-shan¹, YANG Jian-jun¹, WANG Ling-xiang², ZHANG Yan¹, WU Pin-lin¹

(1 Department of Oral and Maxillofacial Surgery, Affiliated Hospital of Qingdao University Medical College.

2 Department of Stomatology, Affiliated Hospital of Qingdao University Medical College. Shandong Qingdao 266003)

ABSTRACT Objective: To investigate the clinical and pathological features of developmental gingival hyperplasia, and to provide a theoretical basis for the diagnosis and treatment of the disease. Methods: The detailed disease history and clinical examination of 11 years old girl were collected, whose mandibular anterior teeth gingival mucosa abnormal grew for 8 years, and who has no family history of similar disease. Dental examination showed a prominent ribbon gingival hyperplasia in the labial side of the mandibular anterior region. It was slightly red, about 2.5 CM× 1.0 CM in size, and grew from the labial side of mucogingival junction to dental crown. The hyperplastic gingival was like a flower petal and covered the middle 1/3 of the anterior teeth. Opened the flower petal could find that the marginal gingiva and gingival papilla of the 41-32 region were almost normal. There were little swollen, almost no tenderness, no erosion of the surface, no bleeding while probing on the hyperplastic gingival. Resected the hyperplastic gingiva by conventional surgical, repaired the local mucosal, and sent the specimen to pathological examination. Results: After the first healed period, there was no scar formation, and the vestibular sulcus and lower labial frenum had a good form. There was no follow 2-year later. The vestibular sulcus, gingiva papillae and lower labial frenum were the normal form. The patient and their families were satisfied. Conclusion: The surgical treatment was an effective treatment for developmental of the gingival hyperplasia.

Key Words: Gingival hyperplasia; Periodontal disease; Developmental Chinese Library Classification (CLC): R781.4 Document code: A Article ID:1673-6273(2011)04-753-04

Introduction

The gingival hyperplasia (GH) is a common disease in clinic of stomatology. The local irritation is the main fashion and the clinical manifestations are inflammatory hyperplasia of the gingival tissue, local pain, bleeding, and often occur in the gingival papilla and the marginal gingival. The base of the hyperplastic gingiva often occurs in the attached gingiva, with occlude difficulty and affect the appearance. The main pathological feature is the fibrous connective tissue proliferation ^[1]. The development of gingival hyperplasia which presents a ribbon and sheet gingival hyperplasia of the attached gingiva is very uncommon, and has not been reported in literatures at home and abroad. The author received a patient of the developmental gingival hyperplasia, and got a satisfactory treatment result.

1 Material and method

uan × was 11-year-old, female, whose gum of mandibular anterior were slight red in 2-3years of age, and the local was ridge-like protrusions, no treatment. In mixed dentition, when the mandibular central incisor erupted, the local protrusive mucosal of ridge-like started to grow with a ribbon and sheet. At 7-year-old, the lower 1/3 of the labial side of mandibular anterior teeth had been covered with a ribbon and sheet gingival mucosa, which with the attached gingival round to an unclosed pocket. At the

△Correspoonding author: YANG Jian-jun, E-mail: qdyjj@126.com (Received:2010-11-03 Accepted:2010-11-30) 9-year-old, the mucosal flap had covered with the mandibular anterior teeth junction of the middle and lower 1/3 of the labial side. Stirred up the mucosal flap could see the consecutive gingival papilla with normal shape. There were no redness, swelling or pressing pain. After one week of using anti-inflammatory by herself (drug names were unknown), the effect was poor. At July 2006, she went to the oral medicine of our hospital for treatment, and she was diagnosed as "the hyperplasia of mandibular anterior ".At that time, her parents opposed to perform operation and only accepted supragingival scaling. At the 11-year-old, when found the mucosal flap of hyperplastic grew faster, her parent took her to the department of oral and maxillofacial surgery of our hospital for treatment again.

Past history and Family history: She was well before, denied any history of hepatitis and tuberculosis disease, and had no history of acute and chronic rhinitis and tonsillitis, no long-term medication history, no past history of allergy to drugs (food), no mouth breathing and other bad habits, no history of special and related genetic disease in the family.

Physical examination (PE): She was general in good condition. Maxillofacial was elementary symmetric and no development malformation. The oral hygiene was good and the lateral incisor in the fourth quadrant was absent. A ribbon and sheet gingival hyperplasia was showed on the labial side of the mandibular 41-32 area. It grew from the mucogingival junction to dental crown and the top was free. The hyperplastic gingival was like a flower petal and covered the middle 1/3 of the anterior teeth, about 2.5CM × 1.0CM in size (Fig.1). There were little swollen, almost no tenderness, no erosion of the surface, no bleeding while probing on the

Author introduction: WANG Da-shan, (1983-), man, master,

Department of Oral and Maxillofacial Surgery

^{13583290867,} E-mail: wdsapple@163.com

hyperplastic gingival. Opened the flower petal could find that the marginal gingiva and gingival papillae of the 41-32 region were almost normal (Fig.2). There were a little of salivary calculus in this



Fig.1 In Pre-Operation, front view

In the submaxillay area and double-neck, swollen lymph nodes did not be touched. It was normal by other examinations of oral and maxillofacial region. The panoramic radiograph showed mixed dentition, 55, 85 roots were completely absorbed, 15, 45 teeth were developing under the root. The other deciduous teeth were lost, with corresponding permanent teeth had erupted. The lateral incisor germ in the fourth quadrant was absent. The alveolar bone of 41-32 area developed normally, no bone absorbent image (Fig.3).



Fig.3 The panoramic radiograph in pre-operation

Treatment: The maxillofacial and oral was disinfected by 1% Povidone-iodine, put asepsis towel, performed mucogingival surgery and gingival revision on the 2% Lidocaine local anesthesia. The hyperplasia was resected very completely (Fig 4), and the wound was covered with periodontal pack, the specimens were sent to pathology. 0.02% Hibitane mouth rinse had been used for two weeks and the cephalexin had been used for 3 days after surgery.

2 Results

Pathology showed "submucosal fibrous tissue hyperplasia with collagen deposition, blood vessels expanded, Congo red stain (-) (Fig 5)". After one week for reexamination, periodontal pack had already peeled, surgical wound had been healing by first inarea and the stippling of the attached gingiva was disappeared, but there was no periodontal pocket.



Fig.2 In Pre-Operation, turned up the hyperplastic tissue

tention, there were no oozy and mild red, and the shape of gingival was favorable (Fig 6). Respectively reexamined on 3, 6, 12, 24 months, the oral mucosa of anterior teeth area was smooth and had no cicatrix. The oral vestibule, gingiva papilla and lower-labial frenum were normal in shape (Fig.7). There was no recurrence. The patient and her families were satisfied.



Fig.4 Excised hyperplastic gingiva

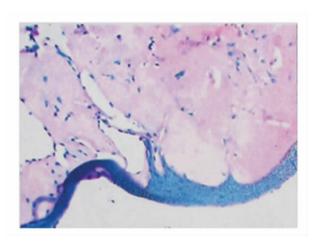


Fig.5 The pathology in post-operation



Fig.6 After one week for reexamination in post-operation



Fig.7 After 24 weeks for reexamination in post-operation

3 Discussion

Gingival hyperplasia as a common clinical disease has several causes, including local stimulations (dental calculus, denture, Mouth-breathing), corticosteroid, medications and hereditary [24]. GH caused by local stimulations, often happened in free gingiva of mandibular anterior region, and accompanied by local pain, bleeding and so on. Increasingly became bulbous, soft and lacked stippling, at last formed gingival pocket. Pathological showed a lot of capillary expansion, heavy congestion, a large number of inflammatory cells infiltration, serious form inflammatory granulation tissue in subepithelial connective tissue but collagen fiber hyperplasia was not obvious ^[5,6]. GH caused by hormonal imbalance tend to occur in puberty and trimester of pregnancy, based on local chronic stimulations and with systemic sex hormone levels of significant changed , hyperplasia involved the whole gingival. Clinical manifestation was mainly inflammatory performance such as bleeding, pain and swelling; Histology showed capillary hyperplasia, inflammatory cells infiltration and inflammation granulation tissue forms as characteristics while collagen fiber hyperplasia was not obvious ^[5,7]. GH caused by medicine was outstanding with the changes of gingival shape, during the early hyperplasia period it was small globular protuberant or nodules, later became mulberry. Hyperplastic gingival tissue grew to crown to form pseudo gingival packet which could easily accumulate plaque and calculus, thus gingival inflammation was aggravated and made hyperplasia intensified [8-11]. Hereditary gingival fibromatosis (HGF) was extremely rare in clinical and few cases had been reported in the literature. It had family history and often happened during the eruption of permanent teeth. The clinical manifestations were gingival hyperplasia gradually spread the whole gingival, even could involve the attached gingiva and mucogingival junction. Hyperplastic gingival had normal colors, smooth surface, obvious stipple, little bleeding and painless, but at the later stage, the gingival obstructed chew for covering all teeth ^[12]. HGF histopathologic performance confirmed gingival epithelial were thickened, spikes were deep into connective tissue connective which were full of thick collagen fiber bunches and less fibroblasts. A small amount of inflammatory cells were also visible in subepithelial connective tissue ^[13-15].

11-year-old girl, the appearance of the asymptomatic mass in the mandibular anterior region at 3 years old, and the lesion had gradually grown since then. Her period of morbidity was earlier than that of other girl adolescence (10-20 years old) [16]. There were not inflammation performances such as bleeding, pain, dull-red gingival and so on in clinical examination. The Subgingival calculus was a little. Periodontal foundation treatment had negligible curative effect so that the causes of local irritation and hormone change could be eliminated. Although the hyperplastic gingival grew towards to dental crowns and covered partial teeth, the way of hyperplasia was different from that of the drugs and hereditary caused GH. The clinical manifestation of the latter showed that the gingiva tissues were more extensive and expansile growth, the texture was hard and the normal shape of gingival papillae was disappeared. In this case, the gingival hyperplasia grew outward from the mucogingival junction presented flake protuberant coverage which could be turned on, it could be seen that the normal gingival no inflammation, texture toughening below it. The patient had not taken these drugs which had been known to cause GH, such as Nifedipine, Dilantin sodium, Cyclosporine. She denied the bad habit such as mouth breathing or family hereditary disease. The hyperplastic gingival were excided, and pathology showed "submucosal fibrous tissue hyperplasia with collagen deposition", did not see the inflammatory cells, thick collagen fiber bunches or similar scar organization structure. Therefore the causes of drug and hereditary factor could be eliminated. The significant changes of gingival hyperplasia were happened in mixed dentition period, when the eruption of permanent anterior teeth stopped, the growth of hyperplasia had obviously slow down. According to the patient's history, clinical manifestation and postoperative pathologic reports, the author thinks the gingival hyperplasia of the patient was may be related to developmental anomalies. So it was diagnosed as the developmental of gingival hyperplasia.

There are many ways to cure gingival hyperplasia, mainly including initial therapy and periodontal surgical phase. Initial therapy which includes oral hygiene instructions, drug therapy, supragingival scaling and subgingival scaling, are suitable for the gingival hyperplasia of obvious local inflammatory reaction and can use for pre-surgical adjunctive treatment. Periodontal surgical which include gingivectomy, flap surgery, mucogingival surgery and so on, apply to the gingival hyperplasia caused by drugs and hereditary factor. According to the patient's clinical manifestation, a systematic treatment plan was drawn up. Due to the hyperplastic gingival grew from mucogingival junction to dental crown with the top was free, the shape of the marginal gingiva and gingival papilla were normal, and there was no periodontal pocket, so the mucogingival surgery and gingival revision were chose after initial therapy. The hyperplastic gingival was integrally cut off from the root, lower labial frenum and vestibular groove were repaired to recover the normal shapes of the gingival and mucosa. At present, many scholars believed that the mainly cause of recurrence was the early accumulation of bacteria ^[17]. So, periodontal pack had been used to cover the surface of the wound for seven days in order to promote the blood circulation and accelerate wound healing. 0.02% Hibitane mouth rinse which could kill bacteria of local tissue and the wound was also cooperated to keep the wound sterile for two weeks. Follow-up 24 months showed that the oral mucosa of anterior teeth area of the patient was smooth and no cicatrix, the shape of the gingiva papilla and lower labial frenum was normal. It wasn't relapse, the patient and her parents were all satisfied. The author thinks that surgical excision associate periodontal pack and hibitane mouth rinse to cure gingival hyperplasia caused by developmental anomalies is an effective method.

References

- Yu SF. The oral histopathology[M]. The fifth edition. Beijing: People's Medical Publishing House, 2006, 173-177
- [2] Anil S, Smaranayake LP, Nair RG, et al. Gingival enlargement as a di agnostic indicator in leukaemia: Case report [J]. Aust Dent J, 1996,41:235-237
- [3] Long RG, Hlousek L, Doyle JL. Oral manifestations of systemic dis-

eases[J]. Mt Sinai J Med, 1998,65:309-315

- [4] Serhat D, Hakan Ö, Mehmet Ş, et al. Gingival Hyperplasia as an Early Diagnostic Oral Manifestation in Acute Monocytic Leukemia: A Case Report[J]. Eur J Dent, 2007, 1(2): 111-114
- [5] Cao CF. Periodontology [M]. The second edition. Beijing: People's Medical Publishing House, 2003, 109-115, 117-120, 179-233
- [6] Fanibunda KB, Soames JV. Odontodysplasia, gingival manifestations, and accompanying abnormalities [J]. Oral surg oral med oral pathol oral radiol endod, 1996, 81(1): 84-88
- [7] Lee W, O'Donnell D. Severe gingival hyperplasia in a child with I-cell disease[J]. Int J Paediatr Dent, 2003, 13(1): 41-45
- [8] Brunet L, Niranda J, Farre N, et al. Aingival enlarge-ment induced by drugs[J]. Drug Safety, 1996, 15(3): 219
- [9] Sun AY. Gingival hyperplasia of calcium antagonists[J]. Foreign Medical Sciences(Section of Pharmarcy), 1995, 22 (1): 43
- [10] Josef Z, Pavel H, Jana Z, et al. Improvement of cardiovascular risk factors and cosmetic side effects in kidney transplant recipients after conversion to tacrolimus[J]. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub, 2009,153(1):67-74
- [11] Chae HJ, Ha MS, Yun DH, et al. Mechanism of Cyclosporine-induced overgrowth in Gingiva[J]. J Dent Res, 2006; 85: 515-519
- [12] Peng QF, Tang LY. Hereditary gingival fibromatosis: Report of two case[J]. Stomatology, 2002, 22 (3) :156
- [13] Ramer M, Marrone J, Stahl B, et al. Hereditary gingival fibromatosis: Identification, treatment controls [J]. J Am Dent Ass oc, 1996, 127 (4): 493-495
- [14] Martelli-Junior H, Lemos DP, Sliva CO, et al. Hereditary gingival fibromatosis: Report of a five-generation family with cellular proliferation analysis[J]. J Periodontol, 2005, 76 (6): 2229-2305
- [15] Brannon RB, Pousson RR. Gingival fibrous nodule--anomaly or pathology[J]. J Dent Hyg, 2003, 77(1): 50-52
- [16] Shen XM, Wang WP. Pediatrics [M]. The seventh edition. Beijing: People's Medical Publishing House, 2008, 3-4
- [17] Coletta RD, Graner E. Hereditary gingival fibromatosis: A systematic review[J]. J Periodontol, 2006, 77 (5):753 - 764

发育性牙龈增生一例报告

王大山¹ 杨建军¹△ 王玲香¹ 张 艳¹ 吴品林¹

(1青岛大学医学院附属医院口腔颌面外科2青岛大学医学院附属医院口腔内科 山东青岛266003)

摘要 目的:探讨发育性牙龈增生的临床及病理特点,并进行回顾性总结,为该疾病的临床诊断和治疗提供理论依据。方法:详细 采集病史和细致的临床检查。患者女性,11周岁,下颌前牙区牙龈粘膜异常生长8年,家族中无类似疾病史;检查见下颌41-32区 唇侧牙龈粘膜自膜龈联合处向冠方可见一带片状牙龈增生物,上端游离至下前牙唇面中1/3处,约2.5×1.0cm大小,颜色略红, 翻起增生物可见41-32处边缘龈及龈乳头形态正常,略有红肿,增生物无压痛,表面无糜烂,探诊无出血。常规手术切除增生龈组 织,局部粘膜修整,标本送病检。结果:术后创口一期愈合,粘膜平整无瘢痕,前庭沟及下唇系带形态良好。术后追踪2年无复发, 口腔前庭及牙龈乳头和下唇系带形态正常,患者及家属满意。结论:手术治疗是对发育性牙龈增生的有效治疗方法。 关键词:牙龈增生;牙周病;发育性

中图分类号:R781.4 文献标识码:A 文章编号:1673-6273(2011)04-753-04

作者简介:王大山,(1983-),男,硕士,口腔颌面外科 E-mail:wdsapple@163.com △通讯作者:杨建军,E-mail:qdyjj@126.com (收稿日期:2010-11-03 接受日期:2010-11-30)