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CASE REPORT

Periodontal disease in adolescent Down' syndrome patients. Clinical case presentation

Enfermedad periodontal en pacientes adolescentes con síndrome de Down. Presentación de caso

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ABSTRACT

Down's syndrome or trisomy 21 is man's most frequent genetic disorder. It affects one out of 600-700 live births. It is characterized by the presence of an additional chromosome at the 21 pair which elicits generalized decrease of growth as well as intellectual disability. In scientific literature there are many reports of cranio-facial and oral manifestations of this disorder. Chief oral condition reported in this group of patients is periodontal disease. **Objective:** Establishment of preventive oral care for the development of periodontal disease in adolescent patients afflicted with Down's syndrome. **Case presentation:** 15 year old Mexican-Indian male patient with trisomy 21. The patient was bilingual, afflicted with congenital heart disease, pulmonary hypertension and nephrotic syndrome. The patient equally presented ectopic dental eruption as well as congenital dental absences, Angle's class III occlusion, anterior and posterior unilateral crossbite, oral respiration, labial eversion, fissured tongue and lips, clinodactyly and localized gingivitis. Behavior management was achieved with the «show-tell-do» technique alongside with voice control. Initial treatment consisted on sweeping brushing technique with parental assistance, use of dental floss (Dento Bacterial Plaque Control [DBPC]) (100%) as well as topical application of fluoride varnish. It was recommended to the parents they should use home applications of bioadhesive gel (Perioxidin®). After these measures, oral rehabilitation was undertaken. **Conclusions:** The most relevant aspect of the treatment was the decrease (40%) in bacterial plaque reported in later control visits.

RESUMEN

El síndrome de Down o trisomía 21 es la alteración genética más frecuente en el ser humano; uno de cada 600-700 recién nacidos vivos es afectado por este síndrome. Éste se caracteriza por la presencia de un cromosoma adicional en el par 21, el cual provoca disminución generalizada en el crecimiento y discapacidad intelectual. Diversas manifestaciones craneofaciales y bucales han sido descritas en la literatura. El principal padecimiento bucal reportado en este grupo de pacientes es la enfermedad periodontal. **Objetivo:** Establecer cuidados bucales preventivos en el desarrollo de la enfermedad periodontal en pacientes adolescentes con síndrome de Down. **Presentación del caso:** Paciente indígena masculino de 15 años de edad con trisomía 21, bilingüe. Presenta cardiopatía congénita, hipertensión pulmonar y síndrome nefrótico, así como erupción ectópica y ausencias congénitas dentales, oclusión clase III de Angle, mordida cruzada unilateral anterior y posterior, respiración bucal, eversión labial, lengua y labios fisurados, clinodactilia y gingivitis localizada. El manejo de conducta se llevó a cabo a través de la técnica «decir-mostrar-hacer» y control de voz. El tratamiento inicial consistió en técnica de cepillado de barrido asistido por los padres, uso del hilo dental, CPDB (100%) y aplicación tópica de barniz de fluoruro. Se recomendó a los padres la aplicación en el hogar de un gel bioadhesivo (Perioxidin®) para la rehabilitación oral. **Conclusiones:** El aspecto más relevante fue la disminución de la cantidad de placa dentobacteriana reportada en controles posteriores (40%).

Key words: Down's syndrome, trisomy 21, periodontal disease, gingivitis.
Palabras clave: Síndrome de Down, trisomía 21, enfermedad periodontal, gingivitis.

INTRODUCTION

Down's syndrome (DS) or trisomy 21 (G-1 Trisomy) is the most frequent genetic disorder afflicting human beings. It is the result of an additional chromosome in the 21st pair. One out of 600-700 live births is affected by it.[1] This genetic disorder is characterized by mental disability in variable degrees, generalized growth decrease, muscular hypotonia and syndrome-characteristic

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craniofacial aspects as well as susceptibility to infections.[2] Among oral and craniofacial conditions related to this syndrome, scientific literature reports the following characteristics: flat occiput, hypoplasia of the mid-facial third, muscular hypotonia, pseudo-prognathism, as well as diastases and lingual protrusion, relative macroglossia, fissured tongue, stepped palate, velar insufficiency, protracted eruption, microdontia, anodontia, taurodontism, cone-shaped teeth, tooth fusion and germination respectively.[3] Other observed features are cross-bite, open bite, labial eversion, fissured lips and oral breathing.[2]

Dental caries does not represent a serious oral health problem for this type of patients.[4] This has been substantiated by a study conducted by Dr. Amano,[5,6] where he reported low numbers of aerobic bacteriae in the saliva, which in turn caused low incidence of *Streptococcus mutans*, and a more alkaline salivary pH. Nevertheless this is not the case for gingivitis[7] and periodontal disease.[8]

DS subjects exhibit great prevalence of periodontal disease, when compared to same-age healthy control groups, as well as with groups of individuals afflicted by other mental disabilities.[2,9-12]

Bone loss has been detected in a high percentage of DS children, as well as gingival bleeding, calculi and deep periodontal pockets.[2,8] Severe periodontal destruction cannot only be explained by poor oral hygiene.[2,9] Dr. Lopez et al.[7] showed there was no statistically significant relationship between plaque extension and calculus and gingivitis severity.

Dr. Lopez et al.[14] conducted a study where they studied 32 DS subjects alongside 32 control subjects in order to compare and determine gingivitis and periodontitis prevalence, severity and extension in both groups. These authors found that the relationship between plaque presence and gingivitis severity was moderate in DS patients. Periodontium health status characteristics were not very different between both groups. Gingivitis extension and severity and periodontitis extension were more prevalent in the group of DS patients than in the control group.

With respect to environmental surroundings, some studies concur in stating that DS patients attending institutions presented worse oral hygiene conditions and greater degree of periodontal disease when compared to patients who stayed at home, even when compared to non DS patients presenting intellectual disabilities.[11,15,16] This is due to the fact that professionals working at the institutions must become case-sensitive in order to face the challenge that involves providing services to this type of patients.[17] Nowadays, subjects afflicted with disabilities are beginning to request dental treatment. Percentages of institutionalized subjects as well as stay-at-home subjects are on the rise.[17]

In DS patients, periodontal disease is not solely caused by poor oral hygiene. Dr. Curess[16], in 1971 found that prevalence of periodontal disease is due to the immunological factor inherent to DS as well as to environmental factors.

Several studies[2,8,10,12,13,18] have revealed an important change in the host's immune response, where many factors are altered such as chemotaxis, phagocytosis, oxidative response as well as an abnormal bactericidal activity of polymorphonuclear leucocytes (PMN). Another proposed situation would be deficient function and low amounts of T cells.[2,13] Barr-Agholme et al.[19] demonstrated that E_2 prostaglandin levels were increased in DS patients. A year later, a new study[13] revealed the fact that there was an altered IgC distribution in the saliva of these patients. Dr. Halinene et al.,[15] in their study, found increased amounts of molecules derived from neutrophils (MMP-8, NGAL) in the crevicular gingival fluid. This results in an inappropriate enzyme regulation, related to DS.

Dr. Armano et al.[6] incorporated other factors to the cause, such as weak periodontal tissue, microrizosis, unfavorable root-crown relationship and poor masticatory function. It has been suggested that certain local factors exert influence on periodontal disease: these could be macroglossia, malocclusion, and bruxism, all of which are frequently exhibited by DS patients.[16]

With respect to progress and destruction severity, oral manifestations of DS patients are similar in pattern to those of juvenile periodontitis cases since they have their onset at early stages as is the case of DS patients.[2,6,7]

Other periodontal disease similarities found between DS patients and juvenile periodontitis (JP) patients are the following: 1) both diseases involve immunological mechanisms (increase of immunoglobulins selective cells-mediated immune- deficiencies, with uneven stimulation of lymphocytic transformation, 2) monocyte and neutrophil defective chemotaxis, 3) similar clinical characteristics: periodontal pockets and severe bone loss in first molars and central incisors, as well as all the remaining teeth.[20]

Recent studies[6,8,21] have revealed the fact that periodontal disease increases with age. Bradley et al.[22] reported the age of 5 years as a starting point for

gingivitis development, and consequently, periodontal disease. Hernandez et al.[23] verified the fact that the greatest degrees of plaque accumulation and gingival infiltration were observed in 14 to 16 year old patients. Zaldivar-Chapa et al.[18] asserted that DS patients developed chronic marginal periodontitis at early adult stages.

The American Academy of Periodontics[24] found that many periodontal bacteriae colonize in early childhood in DS patients. They found that *Porphyromonas gingivalis* and *Actinobacillus actinomycetemcomitans* which are bacteriae strongly related to severe periodontal disease, increase in prevalence alongside with age in the same type of patients, playing thus an important role in the early establishment of periodontal disease.

Gender, age, oral hygiene, type of chromosomal variation, level of special educational needs as well as type of respiration are all factor which failed to exhibit any association with gingivitis severity, as was shown by Lopez et al.[7] and Halinenen.[15]

Dr. Shaw et al.[20] reported that early-diagnosed periodontal disease initiated at lower incisors and first molars, since these teeth are the first to erupt and thus, are in longer contact with local destructive factors.

Periodontal treatment protocol proposed by the American Academy of Periodontics[24] is the following:

1. Patient motivation.
2. Odontoxesis and prophylaxis.
3. Instruction on brushing techniques.
4. Diet control.
5. Weekly therapy with fluoride.
6. Maintenance phase.

Certain cases are complemented with topical application of antiseptic agents such as 2% chlorhexidine, fluoride or antiseptic oral rinses.[25,26]

Zaldiva-Chapa et al.[18] showed that, in the long run, there were no differences between conducting surgical or non surgical treatment since, after a year, results obtained with both treatment types were similar.

Drugs such as chlorhexidine exert a favorable influence in periodontal disease prevention and treatment in patients with mental disabilities. This groups of patients lacks the motor skills needed to manipulate a dental brush, likewise, they are unable to understand the objectives of this activity.[27] Dr. Francis et al.[28,29] as well as Dr. Usher[30] and Dr. Panutti et al.[31] concurred in stating that chlorhexidine gel was a significantly effective agent against plaque and gingivitis. Furthermore, they suggested treatment with chlorhexidine as an alternative to tooth brushing, since the former elicited similar or even better results in the prevention of periodontitis.

In 2005, Dr. Yoshinara et al.[32] agreed on stating that a preventive, continuous, systematic and individualized dental program with available methods, can suppress the progress of periodontal disease in DS subjects. Consequently, in these patients, unsuitable oral hygiene or the interruption of the preventive program results in changes in supra and sub gingival plaque composition which will result in increase of periodontal pocket as well as alveolar bone loss.

OBJECTIVE

To establish preventive oral care in the development of periodontal disease in teen-age patients afflicted with Down's syndrome.

CASE PRESENTATION

15 year old native Indian Mexican patient, with trisomy 21, no karyotype. Patient was born in the city of Oaxaca, was presently living in Mexico City, was bilingual (Spanish and Zapoteca language). Patient attended the Pedodontics Clinic of the Graduate and Research School National School of Dentistry, National University of Mexico, on March 14th, 2004. The parents requested treatment due to observed dental malocclusion in their son.

Medical history taken revealed the following data:

Non-pathological personal history: The patient was the product of a second pregnancy, and was delivered full-term with a caesarean section at the General Hospital in Oaxaca.

Pathological personal history: The patient was afflicted with several congenital conditions: Heart disease (heart murmur), pulmonary disease (pulmonary arterial hypertension) and renal disease (nephrotic syndrome). The patient was pharmacologically controlled with the following drugs: digoxin (Lanoxin), furosemide (Lasix), spironolactone (Aldactone) and captopril (Capotene).

Pathological familial history: Patient's parents did not report pathological data, nevertheless, the father was 36 years old and the mother 38 years old at their son s birth.

Clinical examination: Many general characteristics of Down's syndrome were observed: nasal bridge hypoplasia, marbling complexion,

flat occiput, low-implanted, cup-shaped external ear, clinodactyly (permanent curvature of one or more fingers) and acropachy («drumstick-shaped» fingers). Face analysis revealed a diminished middle third with respect to the other two thirds, straight profile and asymmetric bi-pupilary line *(Figure 1)*.

Intra-oral examination: Clinical oral examination revealed upper and lower labial hypotonicity, hypohydrotic, thickened, fissured upper lip, oral breathing, halitosis, lingual protrusion, lingual diastasis, relative macroglossia, narrow and oval-shaped upper alveolar process, stepped palate, triangular-shaped lower alveolar process, red, poorly hydrated mucosae, suitable vascularization, adequate implantation of upper frena(vestibular) and lower frena (vestibular and lingual) *(Figure 2)*.

Generalized and localized dental status: The patient exhibited permanent dentition, abundant plaque located at the supra-gingival region, gingivitis, canine and molar Angle Class III occlusion, diastema, left unilateral posterior crossbite, ectopic eruption of tooth number 23, rotated teeth 11, 12, 21, 31, missing teeth 13, 32 and 42, resin fillings in teeth 16, 26, 36 and 46 *(Figure 3)*.

DENTAL TREATMENT AND EVOLUTION

After recording medical history, dental treatment was initiated. Prevention was the first measure taken, to that effect, the technique «say-show-do» was used as well as voice control. Parents were instructed on dental floss use as well as «sweeping» brushing technique. The first plaque control was undertaken with a 100% index. Probing and modified Löe and Silness periodontal index revealed a 2.4 score (severe gingivitis). Once prophylaxis was completed, fluoride varnish was applied (Fluor Protector®) in all teeth *(Figure 4)*.

It was recommended to the parents to apply a bio-adhesive gel (Perioxidin®) twice a day, topical application along with a light massage in the gums. «Sweeping» tooth brushing technique was insisted upon.

Operative treatment was conducted in teeth number 36 and 46. Resins were placed after an antibiotic prophylaxis (one single 2 gr dose of amoxicillin one hour before dental treatment).

Periodic fortnightly reviews were needed in order to reaffirm the aforementioned preventive measures. Likewise, in the month of April of the same year, the second plaque control procedure was undertaken.

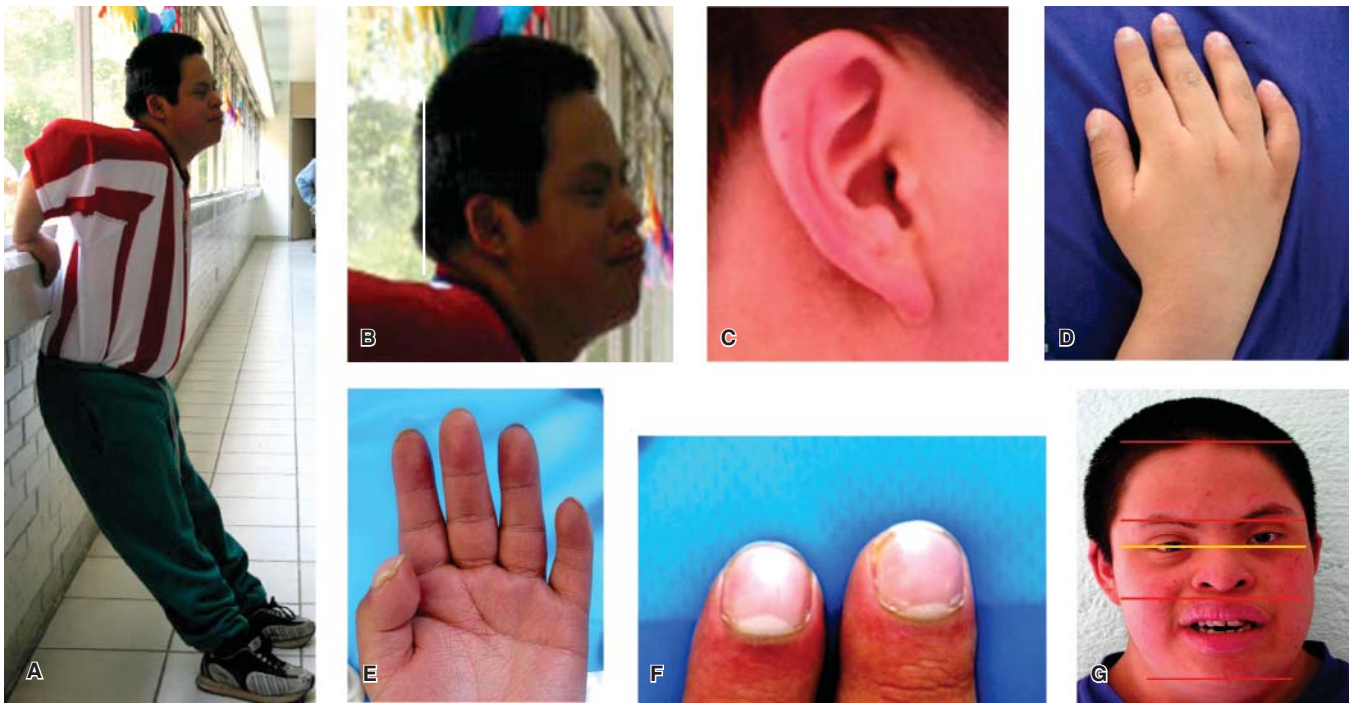

Figure 1. A) Generalized hypotonia. **B)** Flat occiput. **C)** Lw placement of cup-shaped external ear. **D and E)** Clinodactyly. **F)** Acropachy. **G)** diminished middle third and asymmetric bi-pupillary line. Source: Direct.

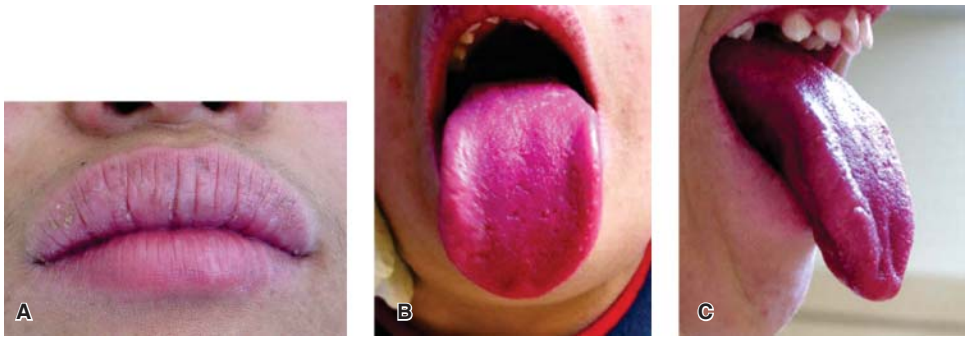

Figure 2.

A) Hipohidrotic and thick fissured upper lip. **B)** Lingual diastases. **C)** Relative macroglossia. Source: Direct.

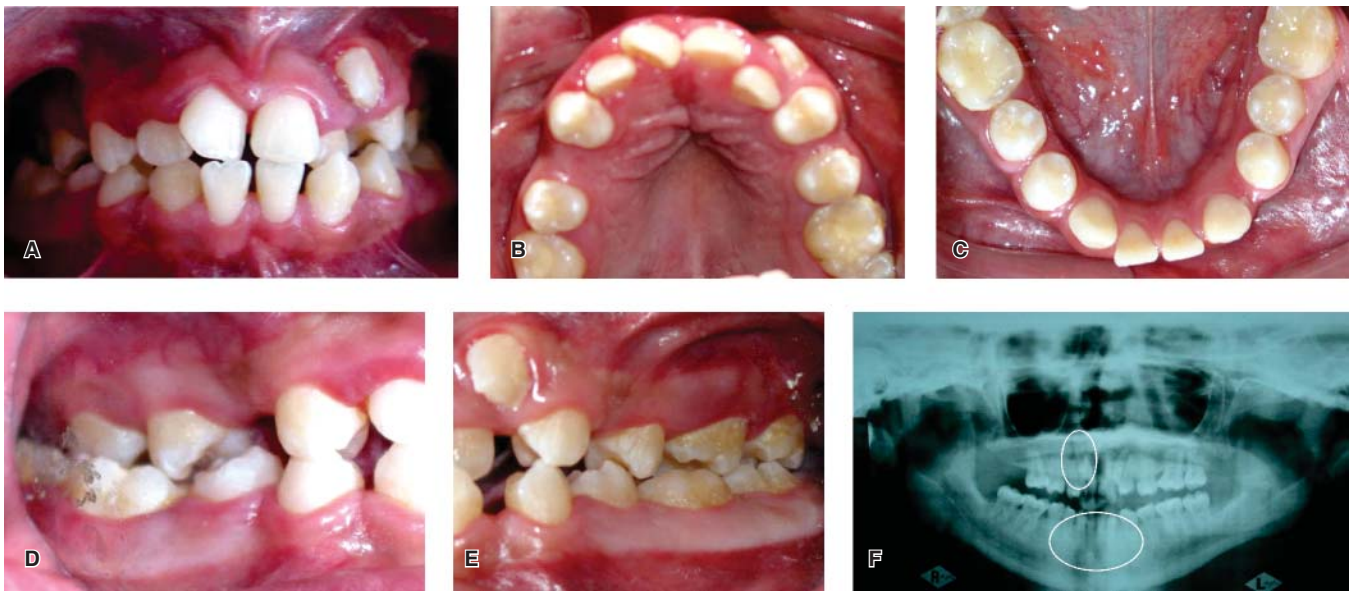

Figure 3. A) Front view: dental malocclusion, abundant plaque, supra-lingually located gingivitis. **B)** Upper occlusion: Stepped palate, resin fillings in teeth 16 and 26, rotated teeth number 12, 11, 21, ectopic eruption of tooth 23, congenital absence of tooth 13, diastema. **C)** Lower occlusal view: congenital absences of teeth 32 and 42, rotation of tooth 41, diastema, resin fillings in teeth 36 and 46. **D)** Right lateral view: crossbite. **E)** Left lateral view. **F)** Orthopantomography. Congenital absence of teeth 13,32 and 42 are observed. Source: Direct.

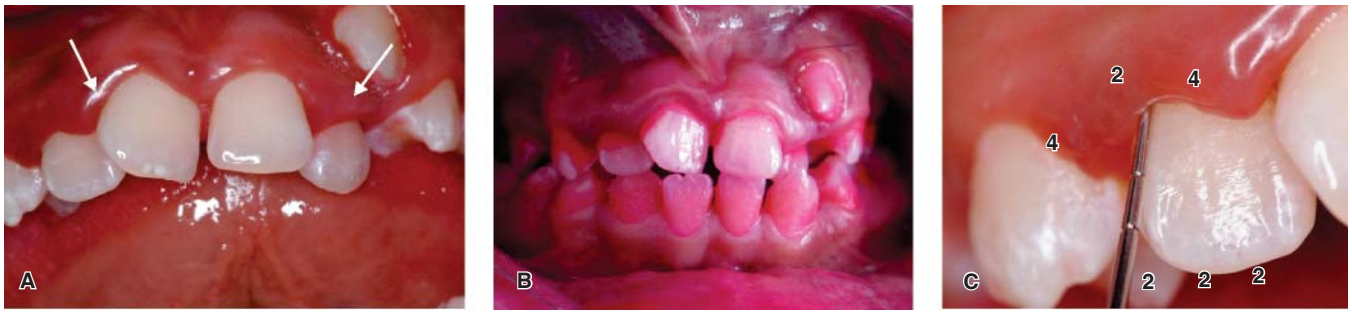

Figure 4. A) Periodontal status of patient at beginning of treatment. Marginal gingivitis in teeth 12 and 22. **B)** March 2004: First personal control of dental plaque. **C)** March 2004: First probing and periodontal index (modified Löe y Silness).
Source: Direct.

Obtained result was 85%. The third plaque control procedure was undertaken in May; obtained index was 41%. The second probing and modified Löe and Silness periodontal index revealed a 1.9 result (moderate gingivitis). First results were obtained in June 2004 in them, decreased periodontal disease was observed *(Figures 5, 6 and 7)*.

An orthopedic-orthodontic assessment was contemplated, due to malocclusion problems presented by the patient (which were the initial cause for his visit to the Clinic). Nevertheless, it was decided to first address and give precedence to the periodontal treatment, and later proceed with the required orthopedic-orthodontic treatment.

The patient missed control visits for 14 months, treatment was re-initiated in August 2005. At that point a fourth personal plaque control was achieved, as well as a third modified Löe and Silness gingival index. Obtained results were 82% and 2.7 (severe gingivitis) respectively *(Figure 8)*.

An inter-consultation with the Periodontics Clinic proved to be necessary due to the periodontal characteristics exhibited by the patient. The patient is presently being treated at that clinic.

The importance of oral hygiene control for this type of patients was stressed with his parents, since periodontal disease can be progressive with age.

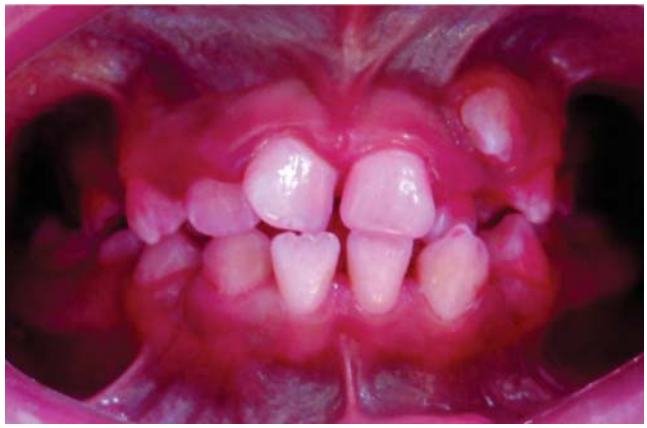

Figure 5. April 2004: Second personal control of plaque. Source: Direct.

CONCLUSIONS

In these special-requirements patients it is of the utmost importance to conduct a follow-up on the periodontal disease treatment, since its etiology not only affects environmental factors but immunological factors as well.

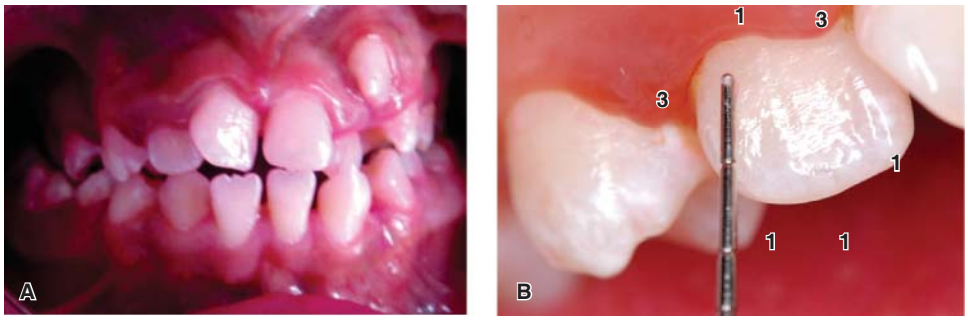

Figure 6.

A) May 2004: Third personal plaque control. **B)** May 2004: second probing and periodontal index (modified Löe y Silness).

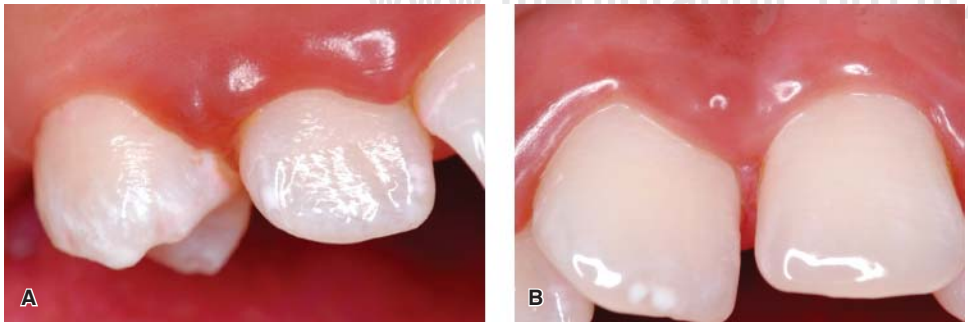

Figure 7.

A and B) June 2004: First obtained results.
Source: Direct.

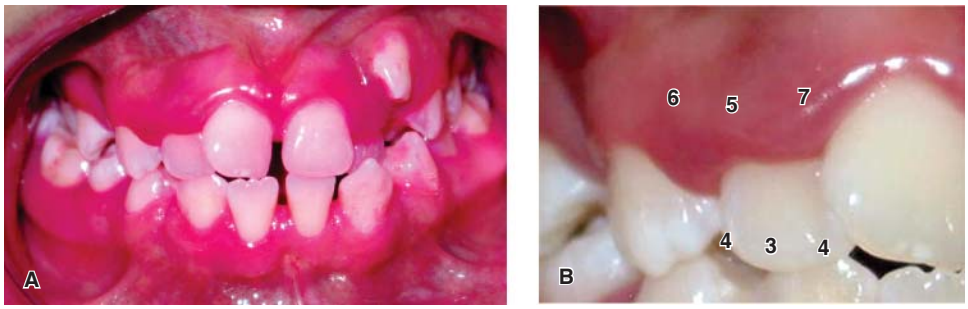

Figure 8.

A) August 2005: Fourth personal plaque control. **B)** August 2005: third probing and periodontal index (modified Löe y Silness). Source: Direct.

There is no doubt in stating that periodontal disease is inherent to Down's syndrome patients, and that it can be controlled with a preventive oral program. A team effort of both the patient's parents or caregivers with dental health professionals is required.

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