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Aggressive Periodontitis Associated With Gingival Tuberculosis: What Diagnosis and Therapeutic Features?

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Abstract

Aggressive periodontitis is a destructive disease involving multiple teeth with a distinctive pattern of periodontal tissue loss; a high rate of disease progression. As long as it is explained as a destructive disease, the presence of a gingival increase cannot be considered as a constant sign of this disease, and can signal the coexistence of other infections or other diseases which may be more important. Screen in the patient and which may in certain considerations even be life-threatening for the patient; in this article, we will try to explain through a clinical case, the diagnosis and therapeutic approach to be followed in a case combining aggressive periodontitis with a specific infection

Keywords: Gingival enlargement; Gingival tuberculosis; Oro-facial granulomatous lesion; Tuberculosis; Aggressive periodontitis; Treatment outcome

Introduction

Aggressive periodontitis is a destructive disease involving multiple teeth with a distinctive pattern of periodontal tissue loss; a high rate of disease progression; an early age of onset and the absence of systemic diseases. Besides infection with specific microorganisms, a host predisposition seems to play a key role in the pathogenesis of aggressive periodontitis, as evidenced by the familial aggregation of the disease [1,2]. A contemporary case definition of aggressive periodontitis is presented by Albandar and Colleagues [3], it includes: Initially, the periodontal lesions show a distinctive pattern, depicted radiographically as vertical bone loss at the proximal surfaces of posterior teeth, and the bone loss usually occurs bilaterally. In advanced cases of aggressive periodontitis the periodontal lesions may be depicted radiographically as a horizontal loss of bone. Early age of onset, Involvement of multiple teeth with a distinctive pattern of clinical attachment loss and radiographic bone loss Aggressive periodontitis may be localized or generalized, in localized aggressive periodontitis, tissue loss usually starts at the permanent first molars and incisors, and with increasing patient age the disease may progress to involve the adjacent teeth. The generalized form of aggressive periodontitis involves most or all of the permanent teeth. A relatively high rate of disease progression and the absence of systemic diseases that compromise the host's response to infection. Although in some patients the disease may start before puberty, in most patients the age of onset is during, or somewhat after, the circum-pubertal period. A typical patient shows disease onset at an early age (i.e., before 25 years of age), although identification of the affected patient usually occurs after disease commencement. Two kinds of gingival responses are seen in patients with generalized aggressive periodontitis. First response is severe acutely inflamed tissue which is ulcerated and red in color with spontaneous bleeding indicating destructive stage and the other one with pink gingiva free of inflammation, with some degree of stippling and deep periodontal pockets are present representing quiescence stage as seeing in the definition, Aggressive periodontitis is a destructive disease characterized by tissue loss, therefore the presence of gingival hyperplastic enlargement in not a normal may be the it is the result of other associated factors (hormonal, drug, genetic orother). Apart from the pain linked to radicular hypersensitivity which can accompany root denudations, acute gingival pain is not described in the semiology of aggressive periodontitis. The aggressive periodontitis treatment requires a good disorganization of the biofilm accompanied by an antibiotic cure aiming to complete the mechanical action undertaken during the non-surgical treatment [4,5]. Despite the literature that estimates a less favourable prognosis for aggressive periodontitis compared to other forms, several other studies have shown a stable periodontal parameters for a long-term in some moderate or severe cases of periodontitis which have been correctly followed by non-surgical treatment and then by surgical treatment and strict personal and professional maintenance [6-8].

Aimetti et al., have showed that non-surgical treatment can be a highly effective treatment for patients and sites affected by aggressive periodontitis. Their results dealt with short-term outcomes [9]. The reparative potential associated with severe aggressive periodontitis may exceed that observed in moderate aggressive and chronic periodontitis and suggests a conservative therapeutic approach [10,11].

Gingival enlargement is one of the frequent features of gingival diseases. However due to their varied presentations, the diagnosis of these entities becomes challenging for the clinician. A perfect diagnosis is critically important, since the management of these lesions and prevention of their recurrence is completely dependent on it. Based on etiopathogenesis, enlargements could be inflammatory, drug influenced, those associated with systemic conditions or diseases, neoplastic or false enlargements. The gingival enlargement according to its location is classified in 3 classes: localized, regional, and generalized enlargement [12].

The generalized gingival enlargement can be classified according to its clinical aspect on inflammatory gingival enlargement and Fibrotic enlargement. The fibrotic gingiva usually presents normal color and firm consistency, with abundant stippling. In The inflammatory gingival enlargement (GE), Gingiva is soft, friable and deep red in colour with increased tendency to bleed. This can be treated by removal of local factors with scaling & root planning after which the gingiva shrinks and becomes firm. The persisting soft gingival enlargement even after the conventional therapy is best treated by gingivectomy while the persisting firm GE is best treated by flap surgery in these two cases, the clinical

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sign such as spontaneous pain associated with gingival enlargement is very rare except for cases who experience overtrauma on hyperplastic lesions or in the case of necrotic ulcertaive gingivitis. The fibrotic gingival enlargement can be classified according to its etiologyon : Drug induced gingival enlargement, Genetic disorders associated with gingival enlargement, Conditioned gingival enlargement : Hormonal, Vitamin C deficiency, Plasma cell gingivitis, Gingival enlargement associated with systemic disease : Leukemia, Wegener's Granulomatosis, Crohn's disease, Sarcoidosis, Tuberculous gingival enlargement, Unusual presentations, Idiopathic gingival enlargement, the most communally recurred gingival enlargement is the Idiopathic gingival fibromatosis. The fibromatosis may potentially cover the exposed tooth surfaces, thereby hampering the functioning of the stomatognathic system. The gingival tissues are usually pink and non-hemorrhagic and have a firm, fibrotic consistency. Considering the recurring character of this entity, the timing of gingivectomy and gingivoplasty for gingival fibromatosis patients is controversial. According to several authors, the ideal time is when all permanent dentition has erupted, because the risk of recurrence is higher before this [13], In the other conditions, or there is no intake of hyperplasiting medication or hormonal cause, and in the case where the gingival increase will take an unusual aspect, the dentist will have to push his investigations further in search of a possible cause such as the specific infection or the tumor or immunological cause.

The gingival enlargement can be also a result of specific infections as primary localization or a second reinfection, one of the rare examples described in literature is the gingival enlargement associated with extra pulmonary tuberculosis EPTB. Tuberculosis (TB) is an asymptomatic airborne tropical infectious disease caused by *Mycobacterium tuberculosis* (*M. tuberculosis*). Tuberculosis can infect any parts of the body but generally affects the lungs. The development of tuberculosis outside the lungs is termed as extrapulmonary tuberculosis (EPTB) and occurs mainly in young adults and immune suppressed persons [14].

Till date, diversified types of EPTB have been reported such as Osteoarticular TB (OAT), Ocular TB (OTB), Ear and nasal TB, Larynx and oral cavity TB, Neurological TB, Lymph node TB (LNTB), Abdominal tuberculosis, Cutaneous tuberculosis, Hepatic and renal tuberculosis, and Genitourinary tuberculosis (GUTB) [15]

The oral cavity is an uncommon site of involvement by tuberculosis. Infection in the oral cavity is usually acquired through infected sputum coughed out by a patient with open pulmonary tuberculosis or by haematogenous spread. Tongue is the most common site of involvement and accounts for nearly half the cases. The lesions are usually found over the tip, borders, dorsum, and base of the tongue [16,17]. Other sites of involvement include floor of mouth, soft palate, anterior pillars, and uvula. Primary infection of the salivary glands is also known, but, it is rare. Parotid gland is most commonly involved.

In 2012, Andrade et al. proposed classification of orofacial tuberculosis based on the site involved [18]. Oral localization is often appearing in the form of painful ulceration of generally giant size with an erythematous outline and a yellowish background with a superficial induration, satellite cervical lymphadenopathy is constant. The differential diagnosis should be made with epidermoid carcinoma of the oral mucosa [18,19]. Usually in extrapulmonary tuberculosis, there are two steps to making a positive diagnosis, first we have to go through orientation exams which allow us to push the investigations in the direction of tuberculosis, and then if the first exams are positives, we have to go to the confirmation stage.

Orientation exams

The tuberculin intradermal injection test: It is a skin exam that explores the delayed hypersensitivity reaction (type 4), induced by mycobacterial antigens (*Mycobacterium tuberculosis* Complex, BCG and certain atypical mycobacteria). This hypersensitivity appears between two weeks and two months after the primary infection [20]. It consists of the intradermal injection of 0.1 ml of the 10-unit tuberculin solution on the anterior side of the forearm. The skin reaction should be read at 72 hours. The induration limits are determined by palpation and measured in millimeters without taking into account any associated erythematous reaction [21]. So, the reaction can be: <5 mm: negative reaction. 5-10 mm (weakly positive reaction): This threshold is sufficient to evoke the diagnosis of primary tuberculosis infection, provided that the infection is recent and narrow or that the patient is infected with HIV. 10 mm (positive reaction): From 10 to 15 mm: The primary tuberculosis infection is retained when the classic risk factors are present. Greater than or equal to 15 mm: This is the necessary threshold, in the absence of any specific storytelling or predisposing factor.

Interferon Gamma Releasing Assay (IGRA): The tests are based on the *in vitro* measurement of the release of gamma interferon (IFN-gamma) by T-lymphocytes sensitized towards certain peptides specific for the *Mycobacterium tuberculosis* complex but absent in *M. bovis* BCG (ESAT-6 and CFP-10) and in most non-tuberculous mycobacteria. They have a comparable sensitivity to that of the tuberculin test in immunocompetent subjects but greater specificity, in particular in subjects vaccinated with BCG. Blood tests avoid the major defect of the tuberculin test, which is the existence of false positives due to prior vaccination with BCG and contact with environmental mycobacteria.

IGRA is much more specific and predictive of latent tuberculosis infection. However, because of the risk of a false negative, this test should not be used in the diagnosis of exclusion from active tuberculosis [22]. Although (IGRA) tests do not differentiate tuberculosis disease from latent tuberculosis, the presence of anti-tuberculosis memory cells in a sterile non-cellular site could sign an active infection other tests such as PCR and blood cell count are not specific for this type of disease [23].

Confirmation exams

Bacteriology: By direct examination to reveal the tuberculosis bacilli on microscopic examination in the cases of obvious locations, or by bacterial culture in the case of a tubercular lesion with negative microscopy especially extra-pulmonary tuberculosis where the diagnosis is difficult to reach by direct examination.

Faced with a positive culture, the differentiation of the tuberculosis complex from other atypical mycobacteria is essential; their management calls for different therapeutic protocols. Classically The identification of mycobacteria of the tuberculosis complex is based on the one hand on the study of the appearance (characteristic cauliflower colonies) and on the other hand on the biochemical identification (research for the production of nicotinic acid or niacin, catalase activity at 22°C and after heating at 68°C for 20 minutes, and sensitivity to para-aminosalicylic acid (PAS). Currently, this characterization can be done by much faster antigenic or molecular tests.

Definite diagnosis however requires the detection of *Mycobacterium tuberculosis*. Stains to detect acid-fast bacilli such as Ziehl-Nelsen and auramine stains allow a quick diagnosis. Nevertheless quantities of between 5000 and 10,000 bacilli/ml are needed in the sample for them to be detected by these stains. This is why the diagnostic yield of smear in EPTB is higher for samples obtained through biopsy (sensitivity>70-80%) than for biological fluids (5-20%). We must always take into account that a variable percentage (30-50%) of EPTB may be smear-negative [24].

Histology: Definite diagnosis of EPTB requires samples from fluids and/or tissues through fine needle aspiration biopsy (FNAB), for smear, culture and PCR testing, even requiring an open biopsy of the affected tissue in case of negative FNAB. histopathological studies of the biopsies show the typical necrotizing granuloma containing macrophages, lymphocytes and Langhans giant cells. Caseous necrosis can be sometimes found in the central part of the granuloma. Its presence has a high specificity and it could justify the decision to initiate anti-tuberculous therapy. However, the presence of granulomatous lesions without necrosis suggests the diagnosis but it requires the exclusion of other infectious and non-infectious diseases. Acidfast bacilli are only found in 10% of the samples and culture is sometimes impossible since the samples have been preserved in formaldehyde. Polymerase chain reaction of samples fixed in formaldehyde would have a greatly variable sensitivity of between 30 and 60%. It is therefore important to preserve biopsy samples in distilled water [25].

The practitioner will have to use all the means of useful science going from the simple to the complex in order to be able to identify the origin of this gingival enlargement in order to offer to his patient the appropriate treatment. In addition to its role in treating periodontal disease, the periodontist will also be responsible for detecting systemic lesions that have oral signs. In this article we report a case of generalized and painful gingival enlargement with loss of attachment and bone destruction similar to that observed in the case of aggressive periodontitis. The lack of improvement after all standard therapies and especially the painful character of the gingival enlargement with underlying severe bone destruction drove us to a progressive diagnostic approach which, at the end, allowed the diagnosis of granulomatous gingival tuberculosis with necrosis and bone sequestration simulating an aggressive periodontitis.

Case Presentation

A 26-year-old girl consults for a generalized gingival enlargement, with bleeding at the slightest touch and pain when chewing. The history of the disease goes back one year ago, the patient was followed by several dental surgeons who tried to treat the patient by means of local periodontal debridement with several attempts of gingivectomys on the lower canine incisor sector associated with prolonged antibiotic therapy. On clinical examination we note in Figures 1A and 1B, the absence of lymphadenopathy or cervical fistulisation. Very poor oral hygiene with a Loe and Silness plaque index of 2.75. Generalized marginal and papillary gingival enlargement. Swollen, turgid, erythematous, shiny, bleeding interdental papillae at the slightest touch. Gingival recession on 31, 41 occurring following gingivectomys already performed. Purulent periodontal pockets. Muhlemen level 3 mobility at level 31, 41.

On radiographic examination we note a moderate to deep generalized bone defect on all teeth with terminal lysis between 31, 41. (Figures 2A and 2B).

Diagnosis and initial treatment plan

The medical history excludes the possibility of a drug gingival enlargement, as well as the requested blood test does not show a significant alteration of the blood formula. Given the clinical and radiological signs observed, a diagnosis of generalized aggressive periodontitis was made, for which a conventional treatment plan including sessions of periodontal debridement (scaling, root planning) according to the principle of full mouth disinfection was adopted (Table 1).

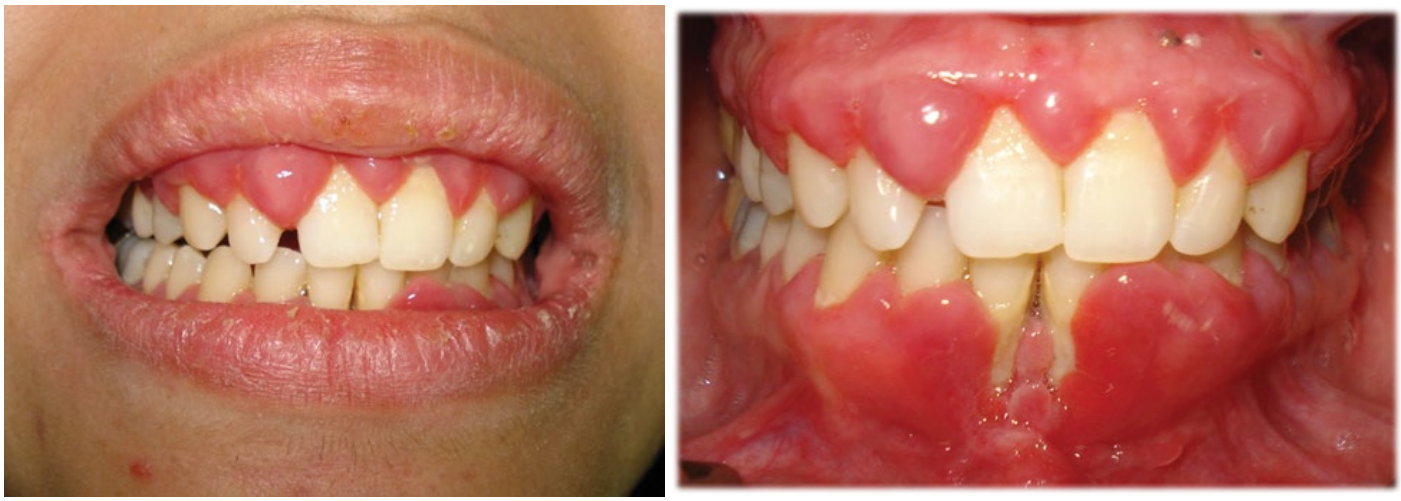

Figure 1. (A-B): Clinical examination, general gingival enlargement with inflammatory appearance and painful ulceration sensation.

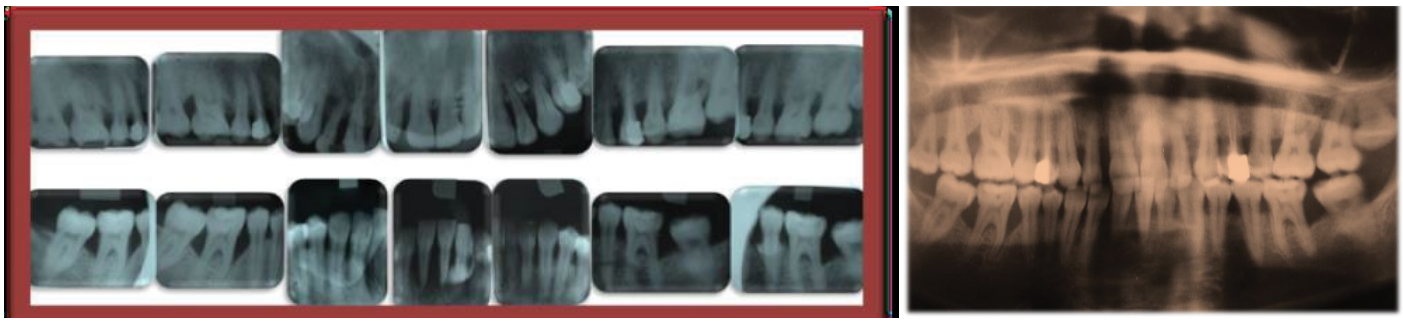

Figure 2. (A-B): X rays examination, a general angular alveolar bone loss confirming a diagnosis of aggressive periodontitis.

Table 1. The following table shows the results and instructions for each medical discipline.

Discipline	Results	Instructions
Hematology	Normal BC (blood count), RS (sedimentation rate) , CRP (C reactive protein)	Insignificant result
Endocrinology	normal endocrinological assessment including sex hormones, growth hormone	Preliminary assessment not significant
Gynecology	regular menstrual cycle Normal Pelvic ultrasound	There is no cause and effect relationship
Dermatology	No signs of dermatological diseases or autoimmune diseases	Prescription of corticosteroid therapy and request another biopsy
Infectiology	Normal RS Negative HIV test Normal liver transaminases	Needs a second histological exam

Re-evaluation

8 weeks after the initial treatment, we did not notice any significant improvement.

Curative phase

Faced with this limited result, periodontal debridement sessions with sub-gingival irrigation with solutions based on 0.2% chlorhexidine and iodinated polyvidone were renewed. To improve patient comfort and facilitate patient hygiene, a gingivectomy of the mandibular areas was performed (Figure 3). One month after the debridement and gingivectomy sessions, there was a recurrence of gingival enlargement in the areas treated. A first biopsy performed by removing a fragment of the vestibular papillary gum between 12 and 11 shows a chronic granulomatous gingivitis without giant cells or caseous necrosis. A second gingivectomy extended to the mandibular and maxillary sectors was performed in combination with a prescription for corticosteroid therapy for 10 days with progressive cessation. During and a few days after corticosteroid therapy, a marked improvement was observed (Figure 4). Periodontal preparation made it possible to have harmonious gingival contours facilitating teeth brushing the one hand and allowing reassurance for the patient.

Two months later, the gingival enlargement recurred in a few places at the oral cavity, resuming spontaneous gingival bleeding (Figure 5). Faced with this unexpected result, and to complete the diagnosis and to be able to make a differential diagnosis with other situations of gingival hypertrophy which can accompany some hematological or dermatological diseases, or even certain endocrine or genetic disorders; the patient was referred to fellow medical specialists in hematology, endocrinology, gynecology, dermatology and finally in infectiology [26].

Following the instructions of these multidisciplinary consultations and after analyzing the data from the literature [27], a second biopsy was performed on the same site as that taken during the first biopsy, but this time accompanied by the note: suspicion of oral tuberculosis. Histopathological analysis of the gingival fragment effectively shows the presence of non-necrotizing granulomatous tuberculoid inflammation. In collaboration with the infectious diseases department of the Casablanca University Hospital, a chest X-ray combined with the tuberculin intradermal injection test and a sputum analysis were carried out. Despite a negative response to these tests, and in cases of tuberculoid infection extrapulmonary sites, the results of the pathological examination are the first to be taken into account, and the diagnosis of gingival tuberculosis has been confirmed [28].

The medical prescription for tuberculosis can involve different combinations; the following table shows the most common combinations as well as the rules of prescriptions (Table 2).

Medical treatment for tuberculosis is compulsory and systematic, and is codified almost everywhere in the world. In Morocco, patients are treated according to the national tuberculosis control program 2011. [29] With the exception of neuro-meningeal and osteo-articular involvement, the recommended diet for extra-pulmonary tuberculosis is that of 6 months: 2RHZE/4RH (Table 3).

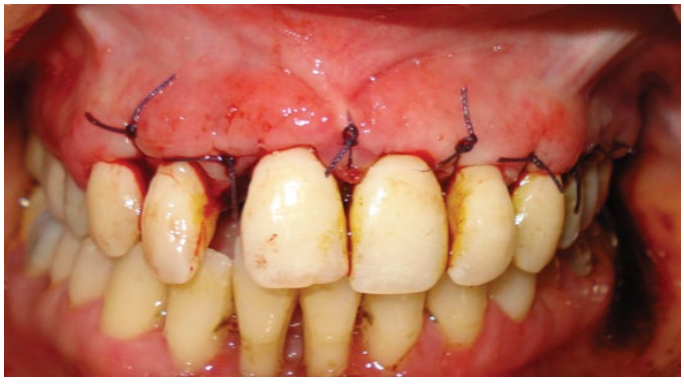

Figure 3. Sectorial gingivectomy 2 months after conventional periodontitis treatment.

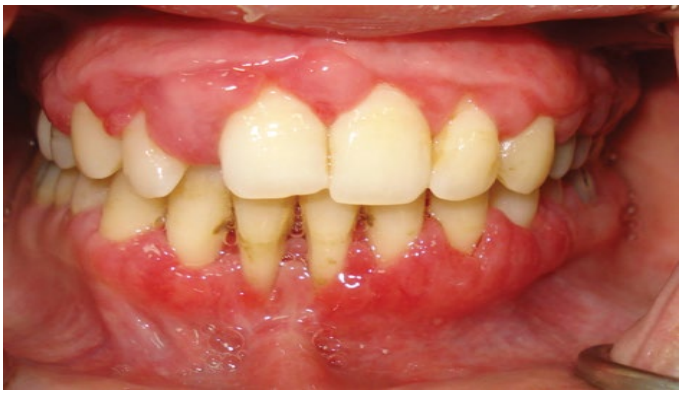

Figure 4. Clinical result 3 weeks after chemical (antibiotherapy, corticotherapy) and mechanical and surgical treatment, we note a clinical improvement.

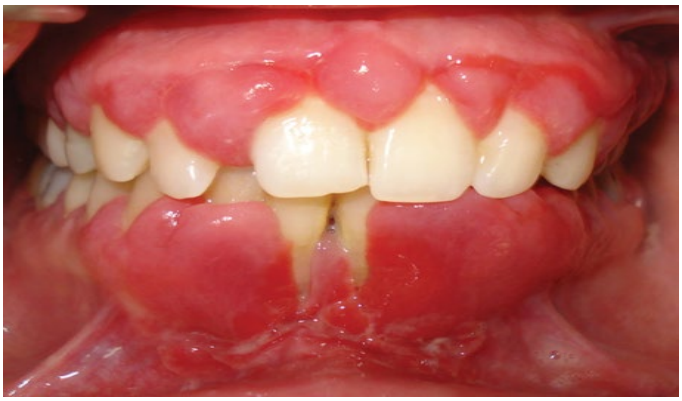

Figure 5. Less painful recurrent gingival enlargement a few weeks after initial treatment.

Table 2. Combination, composition and dose.

Combination	Composition	Dose
RH	Isoniazid + Rifampicin	• 300 table (150 mg of Isoniazid+- 300 mg of Rifampicin) • 150 tablet (100 mg of Isoniazid+ 150 mg of Rifampicin)
RHZ	Isoniazid+ Rifampicin+ Pyrazinamide	• Tablets (75 mg of Isoniazid + 150 mg of Rifampicin+ 400 mg Pyrazinamide)
RHZE	Isoniazid+ Rifampicin+ Pyrazinamide+ Ethambutol	• Tablets (75 mg d' Isoniazide + 150 mg de Rifampicine+ 400 mg de Pyrazinamide+ 275 mg d' Ethambutol)

Table 3. From a pharmacological point of view, the best known anti-tuberculosis drugs.

Drugs	Action	Characteristics
Isoniazid	• An active bactericidal activity on intra and extracellular mycobacteria • Prevents the appearance of resistance	• Major anti-tuberculosis drug • Quick oral absorption, • Liver metabolism • Good tissue diffusion
Rifampicin	Bactericide and sterilizer, active on all populations of BK including those contained in the caseum	• Semi-synthetic anti-tuberculosis drug • Good tissue diffusion • Hepatic degradation
Pyrazinamide	Bactericidal action mainly on bacilliintracellular	• Secondary resistance develops rapidly • Primary Resistance and crossover with other anti-tuberculosis drugs is rare.
Ethambutol	Bacteriostatic on mycobacteria	• Good a tissue diffusion • Renal elimination
Streptomycin	• A bactericide action • A high Activity on the extracellular bacillary population	• antibiotic of the family of aminoglycosides

For our patient, the initial phase involves the combination of 4 anti-tuberculosis drugs: Isoniazid, Rifampicin, Pyrazinamide and Ethambutol for 2 months, the continuation of treatment combines Isoniazide and Rifampicin for 4 months. The patient was followed periodically during antibacillary treatment every two weeks. Local treatment consisted on periodontal debridement and a gingivetomy with in maxillary and mandibular arch. Indeed, two weeks after debridement and gingivectomy, we noted a clear improvement in quality of gingival healing and in terms of patient comfort. (Figure 6) the results improved gradually over time, and we did not notice any recurrence over the 6 months of antibacillary treatment. The results remained stable until 6 years later (Figure 7). We then confirmed the positive diagnosis of gingival tuberculosis.

Discussion

In the case described in this article, the different pathological entities included in the differential diagnosis were an enlargement due to drugs, malignant tumors, traumatic ulcers and/or an infection (bacterial, fungal and viral). Since the patient was not taking any systemic drugs, gingival enlargement due to the drugs was excluded [30-32]. Various routine investigations have been advised to investigate the possibility of this atypical appearance of the gum. The results of the complete blood count, urinalysis and biochemical tests were within normal limits, excluding the possibility of an associated leukemic enlargement. HIV, HBs antigen tests and VDRL (Venereal Disease Research Laboratory) were also negative, eliminating any infectious etiology of the enlargement. In addition, complete supragingival debridement under universal precautions did not show any improvement in the condition of the gum on call. Thus, an incisional biopsy was performed on the upper labial gum relative to the maxillary right central incisor. Finally, the Histopathological analysis of the gingival fragment effectively shows the presence of non-necrotizing granulomatous tuberculoid inflammation.

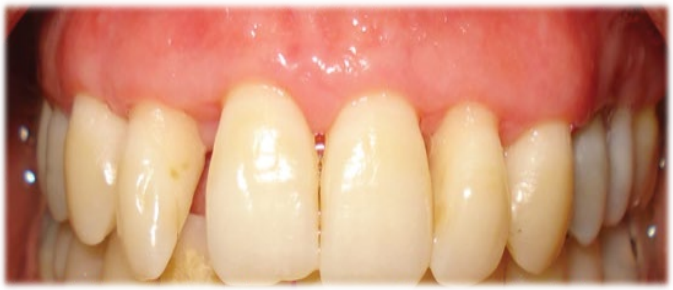

Figure 6. A stable result 6 years later, no recurrent gingival enlargement.

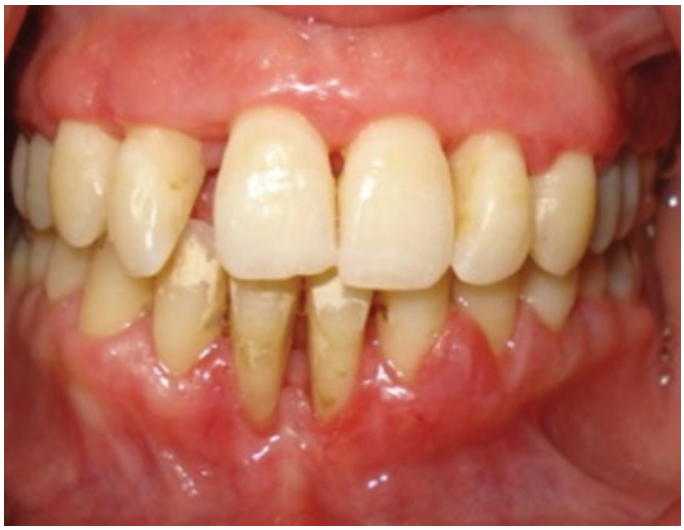

Figure 7. Clinical aspect of the gum two weeks after the start of anti-tuberculosis treatment, we clinically note a significant regression of gingival growth and a very good improvement in periodontal parameters.

According to the definition proposed by Jain and al, the tuberculous gingivitis may appear as nodular or papillary proliferation of gingival tissues which is diffuse and hyperaemic. There may be absence of any clinical attachment loss, alveolar bone loss or significant cervical lymphadenopathy. Such diffuse gingival enlargements fail to respond to initial usual therapy consisting of supragingival debridement. Sometimes tuberculous gingivitis can be seen simultaneously with marginal periodontitis and enlarged cervical lymph nodes or may present as periodontal loss of tooth support leading to loose teeth and gingival enlargement. A biopsy of the lesion is mandatory for arriving at the diagnosis of tuberculosis [33-35].

In our situation, it is a case of gingival growth associated with bone loss simulating to the bone loss encountered in the case of aggressive periodontitis. In this combined context, the diagnosis of aggressive periodontitis was obvious via clinical and radiological examination, it remains to be seen whether this periodontitis is primary and subsequently aggravated by periodontal tuberculosis or that it is secondary to tuberculous involvement [36,37].

The positive response of this form of lesion to local therapy associated with systemic anti-tuberculosis therapy and not to the local therapy alone, suggests a saprophytic reaction of the pardontopathogenic bacteria following tuberculosis infection which increases their pathogenic potential and their resistance to the conventional therapy.

In 1991 WHO published principles for the national tuberculosis control programs for the first time, including criteria for the diagnosis of smear-positive and negative smear pulmonary tuberculosis and extra-pulmonary tuberculosis.

These principles were then revised in 1997 and 2003. To respond to the fear that the principles revised in 2003 do not take sufficient account of the very great difficulty in the diagnosis and treatment of tuberculosis associated with HIV, WHO reviewed its recommendations regarding the diagnosis of tuberculosis in the context of HIV prevalence.

It is understood from these recommendations that anti-tuberculosis treatment should be started as soon as other common conditions likely to give a similar clinical symptom have been excluded.

In fact, most patients with extrapulmonary forms of tuberculosis have sufficiently characteristic symptoms that tuberculosis treatment can be started without waiting for bacteriological or histological confirmation of the diagnosis [14,15].

Although extra-pulmonary tuberculosis can be confirmed in the majority of patients by an invasive biopsy and/or by several cultures, it is not recommended to systematically perform these investigations because of their cost and the delay they can bring at diagnosis, which in turn may reduce the chances of a satisfactory response to treatment.

The anti-tuberculosis panel is specific and very limited. Currently they can be divided into first and second line molecules. First-line molecules are the drugs of choice used in the standard treatment, namely: isoniazid, rifampicin, ethambutol and pyrazinamide. Second-line molecules correspond to all other molecules intended to treat tuberculosis resistant to first-line anti-tuberculosis drugs. They are less effective, more toxic and more expensive.

In the case reported in this article, types of various anatomopathological forms in favor of a tuberculous lesion were considered as the main sign to start anti tuberculosis treatment [38-41]. In the absence of other general signs and to avoid the presence of localization, an X-ray of the thorax as well as sputum cultures was carried out (Figure 8).

The patient was declared to the Moroccan tuberculosis control services, and treatment was started. Note that treatment and monitoring of tuberculosis are provided free of charge in Morocco. Periodontal debridement as well as the gingivectomys performed all finds their interest in promoting the optimal conditions for the success of anti-tuberculosis treatment

For other extra pulmonary localizations of tuberculosis such as tuberculous meningitis, some authors believe that the administration of corticosteroids from the moment when the diagnosis of tuberculosis is made and during the first two months of anti-tuberculosis treatment makes it possible to significantly increase

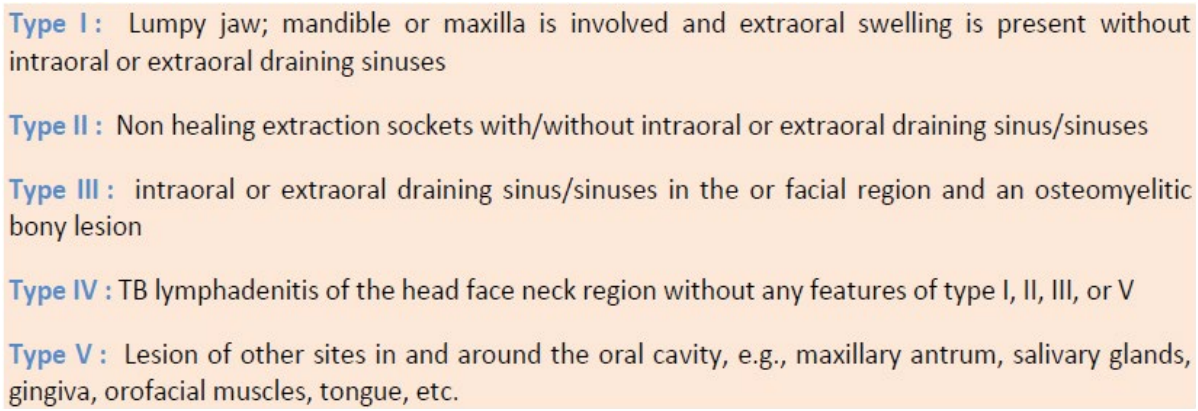

Figure 8. Types of various anatomopathological forms of a tuberculous lesion.

the survival rate of HIV negative patients. For other forms of extrapulmonary tuberculosis and for HIV-related tuberculous meningitis, the effects of steroids are not yet proven. Others believe it is advantageous to add steroids to the treatment of tuberculous pleural effusions [42]. By extrapolation and in the absence of consensus, we believe that the administration of corticosteroids will allow results to be obtained more quickly and more consistently over time in the case of gingival tuberculosis in seronegative patients.

Conclusion

The case presented in this article highlights that all the difficulty encountered in cases of gingival enlargement lies in establishing a correct diagnosis. Once the diagnosis is established, treatment becomes easier by choosing the most appropriate means. Periodic debridement, gingivectomys, corticosteroid therapy, and anti-tuberculosis antibiotic therapy are the means that each practitioner can use when the diagnosis of tuberculosis with gingival enlargement is correctly made. Logical reasoning makes it possible to choose the therapeutic approach and the means of treatment.

References

1. Vaibhavi, Joshipura, Umesh Yadalam, "Aggressive periodontitis: A review." Journal of the International Clinical Dental Research Organization 7 (2015).
2. Daniel, H. Fine, Amey G. Patil, Bruno G. Loos. Classification and diagnosis of aggressive periodontitis." J Periodontol 89 (2018): 103–119.
3. Jasim, M. Albandar. "Aggressive and acute periodontal diseases." J Periodontol 2000 65 (2014).
4. Larry A. Sweeting, Karen Davis, Charles M. Cobb. "Periodontal Treatment Protocol (PTP) for the General Dental Practice." The Journal of Dental Hygiene Special supplement.
5. Krebs KA, Clem DS. "American Academy of Periodontology. Guidelines for the management of patients with periodontal diseases." J Periodontol 77 (2006): 1607-1611
6. Kamma JJ, Baehni PC. "Five year maintenance follow-up of early onset periodontitis patients." J Clin Periodontol 30 (2003): 562-72.
7. Gary, C, Armitage, Mary P. Cullinan. "Comparison of the clinical features of chronic and aggressive periodontitis." Periodontology 2000 53 (2010): 12–27
8. Novak KF, Goodman SF, Takei HH. "Determination of prognosis. In: Newman MG, Takei H, Klokkevold PR, Carranza FA, eds. Clinical Periodontology." Philadelphia: Saunders/Elsevier 10 (2006): 614-625.
9. Aimetti M, Romano F, Guzzi N, Carnevale G."One-stage fullmouth disinfection as a therapeutic approach for generalized aggressive periodontitis." J Periodontol (2011): 82-845
10. Amal, Bouziane, Latifa Benrachadi ,Redouane Abouqal , Oumkeltoum Ennibi. "Outcomes of nonsurgical periodontal eissn 2093-2286 therapy in severe generalized aggressive periodontitis." J Periodontal Implant Sci 44 (2014): 201-206.
11. Wennstrom A, Wennstrom J, Lindhe J. "Healing following surgical and non-surgical treatment of juvenile periodontitis: a 5-year longitudinal study." J Clin Periodontol 869 (1986): 13-82.
12. Amit Arvind Agrawal. "Gingival enlargements: Differential diagnosis and review of literature." World J Clin Cases 16 (2015): 779-788.
13. Rashi Chaturvedi. "Idiopathic Gingival Fibromatosis Associated with Generalized Aggressive Periodontitis: A Case Report." JCDA 75 (2009).
14. Ramirez M. Lapausa, Menendez A. Saldana, Noguerado A. Asensio. "Extrapulmonary tuberculosis: an overview." Rev Esp Sanid Penit 17 (2015): 3-11.
15. Ameer Khusro, Chirom Aarti, "Extrapulmonary tuberculosis: An overview on infection beyond Lungs." World News of Natural Sciences 28 (2020): 131-141.
16. Memon GA, Khushk IA. "Primary tuberculosis of tongue." J Coll Physicians Surg Pak 13 (2003): 604–5.
17. Hashimoto, Y, Tanioka H. "Primary tuberculosis of the tongue: Report of a case." J Oral Maxillofac Surg. 47 (1989): 744–6
18. Andrade, NN, Mhatre TS. Orofacial. "Tuberculosis−A 16−year experience with 46 cases." J Oral Maxillofac Surg 70 (2012): 12−22.
19. Wu-tong Ju, MD,a Yong Fu, MD,a Ying Liu, and Mda Yi-ran Tan, et al." Clinical and pathologic analyses of tuberculosis in the oral cavity: report of 11 cases." Oral and Maxillofacial Pathology 125 (2018).
20. Richeldi L. "An update on the diagnosis of tuberculosis infection." Am J Respir Crit Care Med 174 (2006): 736 742.
21. Surajit, Nayak and Basanti Acharjya. "Mantoux test and its interpretation." Indian Dermatol Online J. 3(2012): 2–6.
22. Lalvani, A, Pathan AA. "Rapid detection of Mycobacterium tuberculosis infection by numeration of antigen-specific T cells." Am J Respir Crit Care Med 163(2001): 824-828.
23. Mazurek GH, Jereb J, Vernon A, and Iobue Pet al. "Updated guidelines for using interferon gamma release assays to detect mycobacterium tuberculosis infection. United States." MMWR Recomm Rep 25 (2010): 59: 1.
24. Wu, tong Ju, MD Yong Fu, MD Ying Liu, Mda Yi-ran Tan. "Clinical and pathologic analyses of tuberculosis in the oral cavity: report of 11 cases." Oral and Maxill Orofacial Pathology 125 (2018).
25. Savithri, V, Janardhanan M, Suresh R and Aravind T. "Orofacial Granulomatosis Presenting as Gingival Enlargement – Report of Three Cases." J Dent & Oral Disord 3 (2017).
26. Lanka, Mahesh, Ashi Chugh, Sagrika Shukla and Varun Raj Kumar. "Palatal Overgrowth Presenting as Oral Tuberculosis. A Case Report." ACTA Scientific Dental Sciences 3 (2019).
27. Ravindranath Vineetha. "Diffuse gingival enlargement: An unusual diagnostic clue for pulmonary tuberculosis." Clinical Picture 18 (2018): 1288.
28. Maria Subash, Aaron Muthuraj, Arun Parappa Maradi, Srihari Janakiram, Koshy Chithresan. "Tuberculous gingival enlargement: A rare clinical manifestation." Journal of Indian Society of Periodontology 21 (2017).
29. Ministère de la Santé Publique. "Royaume du Maroc. Le nouveau programme national de lutte antituberculeuse." 1990.

30. Lanka, Mahesh, Ashi Chugh, Sagrika Shukla and Varun Raj Kumar. "Palatal Overgrowth Presenting as Oral Tuberculosis. A Case Report." ACTA Scientific Dental Sciences 3 (2019).

31. Sanjeev, Jain, Bharti Vipin, and Pankaj Khurana. "Gingival tuberculosis". J Indian Soc Periodontol 13 (2009): 106–108.

32. Aaron Muthuraj MS, Maradi AP, Janakiram S, Chithresan K. "Tuberculous gingival enlargement: A rare clinical manifestation." J Indian Soc Periodontol 21 (2017): 156-9.

33. Bangalore Varadhan Karthikeyan, Avani Raju Pradeep, C.G. Dileep Sharma. "Primary Tuberculous Gingival Enlargement: A Rare Entity." Can Dent Assoc 72 (2006): 645-8.

34. Kafil Akhtar, MohdTalha, Sadaf Haiyat Saif Khan. "Gingival tuberculosis: A rare presentation." AAMS 2 (2018): 1.

35. Geeti Gupta, Khattak B. P, Vipin Agrawal. "Primary gingival tuberculosis: A rare clinical entity." Contemporary Clinical Dentistry 2 (2011).

36. Chandan Kumar, Prasad Vijayrao Dhadse, Ankita Arun Agarwal, Bhairavi Vikas Kale, Bindu Suresh Sigh. "Oral and Periodontal Status in Patients with Tuberculosis in Vidarbha Region of Central India." Journal of the International Clinical Dental Research Organization 10 (2018).

37. Kumar C, Dhadse PV, Agarwal AA, Kale BV, Sigh BS. "Oral and periodontal status in patients with tuberculosis in Vidarbha region of Central India." J Int Clin Dent Res Organ 18 (2018) 81-7.

38. Savithri V, Janardhanan M, Suresh R and Aravind T. "Orofacial granulomatosis presenting as gingival enlargement – Report of three cases." J Dent & Oral Disord 3 (2017).

39. Varun, R. Kumar. "Palatal overgrowth presenting as oral tuberculosis. A case report." Acta Scientific Dental Sciences 3.11 (2019): 44-48.

40. Suhail M. Jan, Fayiza Youqoob Khan, Muzafar A. Bhat, and Roobal Behal. "Primary tuberculous gingival enlargement - A rare clinical entity: Case report and brief review of the literature." J Indian Soc Periodontol 18 (2014): 632–636

41. J. S. Gill, S. Sandhu and S. Gill. "Primary tuberculosis masquerading as gingival enlargement." British Dental Journal 208 (2010): 8.

42. Tamilarasu, Kadhiravan and Surendran Deepanjali. "Role of corticosteroids in the treatment of tuberculosis: An evidence-based update." The Indian Journal of Chest Diseases & Allied Sciences 52 (2010).

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