SEVERE PERIODONTAL IMPAIRMENT IN SYSTEMIC CONDITIONS: A CASE REPORT

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ABSTRACT
Aggressive forms of periodontitis are defined by rapid localized or generalized loss of the supportive periodontal structures, occurring in familial groups in otherwise medically healthy subjects. Aggressive periodontitis consists in disease different phenotypes of plurifactorial etiology that appear as a result of complexe interactions between specific genes of the host and environment. The interactions between the disease process and the modifying factors (stress, smoking habit) are considered to influence the specific manifestations of the disease. We present the case of a patient with localized aggressive periodontitis, with a late interception, in a context of stress, chronic smoking and inadequate oral hygiene and diet as a background. Smoking, stress and a poor oral hygiene are definitively risk factors that enhance the phenotypic manifestations of the periodontal disease with an aggressive character. Therefore, such aspects must be considered in the diagnostic, prognostic and therapy of the periodontal disease.

Key words: aggressive periodontitis, stress, cigarette smoking

INTRODUCTION
Aggressive periodontitis consists in disease different phenotypes of plurifactorial etiology that appear as a result of complexe interactions between specific genes of the host and environment. The hereditary character of the susceptibility to aggressive periodontitis is insufficient for the disease to appear and to evolve: the exposure to potential pathogens with specific virulence factors represents an essential step. The incapacity of the host to deal with the bacterial aggression and to avoid inflammatory tissue damage results in the initiation of the disease process. Interactions between the disease process and environmental (e.g. stress and cigarette smoking) factors are thought to contribute to determining the specific clinical manifestation of the disease.

The mechanisms by which psychosocial stress may affect the periodontal status are complex. It has been suggested that one of the plausible pathways may involve behavioral changes leading to smoking and poor oral hygiene [1]. Numerous studies have assessed the relationship between stress and periodontal disease. Linden et al. [2] evaluated the association between occupational stress and the progression of periodontitis and reported that longitudinal attachment loss was significantly predicted by
increasing age, lower socioeconomic status, lower job satisfaction and type A personality (characterized by aggressive, impatient and irritable behavior). In a recent study, Breivik et al. [3] demonstrated that experimentally induced depression in rats accelerated tissue breakdown in a ligature periodontitis model and that pharmacologic treatment of depression attenuated this breakdown.

Cigarette smoke is a very complex mixture of substances with over 4000 known constituents. These include carbon monoxide, hydrogen cyanide, reactive oxidizing radicals, a high number of carcinogens, and the main psychoactive and addictive molecule – nicotine [4]. Nicotine is absorbed rapidly in the lung. The administration of nicotine causes a rise in the blood pressure, an increase in heart rate, an increase in respiratory rate, and decreased skin temperature due to peripheral vasoconstriction. However, at other body sites, such as skeletal muscle, nicotine produces vasodilatation.

An analysis of the data from NHANES III study concluded that smokers have a risk four times higher than non-smokers [5]. The informations suggest a dose-effect relation between the number of cigarettes smoked per day and the susceptibility to periodontitis. The study estimated that more than 40% of the periodontal disease in adult cases are enhanced by current smoking habit. Clinically relevant, smoking interferes with the healing after root planing and curettage [6,7,8], postsurgical healing [9, 10, 11, 12] and healing after guided osseous regeneration procedures [13]. The mechanisms for the adverse effects induced by cigarette smoking are stated but the molecular pathways remain to be discovered [14,15]. Smoking represents, without a doubt, a risk factor for the majority of the inflammatory periodontal diseases.

CASE REPORT

A patient (T.N.), 41 years old, with a base occupation of medical assistant, presents for dental treatment, accusing a high dental mobility (tooth 2.2) and moderate mobility (sextant V), multiple recessions and carious lesions (2.8, 3.7).

The diagnosis was based on the anamnestic informations (systemic diseases questionnaire), local clinical examination with imagistic assessment (photographs, intraoral camera) and paraclinical evaluation (imagistic: panoramic radiograph and laboratory analysis chart).

From the discussions with the patients we obtained the following data: the patient is a heavy smoker (2 packs/day, for more than 20 years); he worked abroad for a long time (mostly in improper conditions), a period of time characterized by environmental and psychosocial stress, inadequate diet and inconstant oral hygiene measures. Momentarily, he is still under working stress.

After the intraoral clinical examination (Fig.1,2) we observed the following aspects:

**Maxilla:**
- Cervical carious lesions 1.4, 1.5 and sextant II; 2.8-class II carious lesion
- Diastema, trema
- Multiple recessions, II and III Miller class
- Slight bleeding (degree 1-2)

**Mandible:**
- Class II Kennedy edentulous bridge with 2 modifications (narrowed edentulous space in IVth quadrant)
- Cervical carious lesions 3.7, 4.4, 4.5
- Class II and III multiple recessions
- High probing depths (Fig.3)
- Moderate bleeding (degree 2-3).

The radiologic examination revealed deep infrabony pockets, suggesting a localized form of aggressive periodontitis (previously known as juvenile periodontitis), with vertical osseous defects (Fig.4).
Figure 1. Intraoral examination – initial aspect maxilla

Figure 2. Intraoral examination – initial aspect mandible

Figure 3. Maxillary and mandibular periodontograms
On the first dental visit we conducted the following measures:
- a rigorous clinical examination
- supragingival scaling – mixed technique (manually and mechanically - ultrasounds)
- the periodontograms
- we collected venous blood to assess the neutrophils function
- collection from 2.2, 4.1, 4.4-4.5 situses with micro-ident kit
- biochemical blood testing (haemoleucogram, lipidic profile, glycemia, glycosylated hemoglobin).

The laboratory examination revealed dyslipidemia (that can be included in smoking context) and a high glycemia (fig.5).

qPCR test revealed in direct culture Fusobacterium nucleatum and Porphyromonas gingivalis.

On the second dental visit we conducted the following measures:
- Subgingival scaling
- Root planing
- Curettage without surgical access
- Systemic antibiotherapy: Metronidazol 250mg at 8 hours, 7 days, Ciprinol 500mg at 12 hours, 10 days
- Oral hygiene measures correction
- Recommendation for Parodontax toothpaste, dental brush of soft or
ultrasoft type and additional oral hygiene products (interdental brush, oral rinses with mouthwash: non-alcoholic solution of chlorhexine digluconate – Parodontax for two weeks)

- Dental immobilization on sextant V with splint and composite
- Selective polishing.

After one month we observed the decreasing of the probing depths and of the dental mobility. We conducted the treatment for the carious lesions on teeth 1.4, 1.5, sextant ii, 4.4 and 4.5 with compomers; tooth 3.7 received a temporary obturation with glassionomers.

Because the dental mobility was still present of the tooth 2.2, we decided for a dental immobilization on the maxillary teeth too.

Due to the difficult mobility of the patient (he still works abroad), the following evaluation will be conducted after 3 months. For that session we proposed, with the patient agreement, to treat the edentulous space in the iiird quadrant, to extract tooth 2.8 and to begin an interdisciplinary therapy for smoking cessation, therefore reducing a major risk factor for the periodontal disease.

DISCUSSIONS
Aggressive forms of periodontitis are defined by rapid localized or generalized loss of the supportive periodontal structures, occurring in familial groups in otherwise medically healthy subjects [16]. Aggressive forms can affect the primary or permanent dentition. Typically, susceptible patients are less than 30 years old at disease onset [17]. The similar phenotypes of aggressive periodontal disease are probably the clinical expression of multiple disease forms with discrete etiologies [18].
The reported prevalence of early-onset aggressive periodontitis varies from study to study. The comparability of the data is affected by the somewhat ambiguous disease definitions and the various diagnostic techniques used. A review concluded that aggressive forms of periodontitis have a low prevalence in most regions of the world, occurring in 0.1–1.0% of the population [19].

Although earlier reports by Saxen [20] showed a female majority among subjects with early-onset aggressive periodontitis, a more recent study conducted in the USA did not confirm this observation [21]. Furthermore, based on the results of a genetic segregation analysis performed in 100 families, the aggressive disease has an autosomal dominant inheritance pattern [22]. This contrasts with the autosomal recessive inheritance pattern identified in northern Europe [23], suggesting different pathways to disease for each of the two populations.

Many of the smoke components could modify the host response in periodontitis. Cigarette smoking represents the second most important risk factor for the periodontal disease, after a poor oral hygiene. Depending on the period of exposure to cigarette smoking, daily number of cigarettes and periodontal status, one of the main treatment measures consists in smoking cessation counseling. In the case of a heavy smoker, professional programs in which he can be included might represent a necessity.

CONCLUSIONS

Smoking and psychosocial stress must be addressed in the diagnostic, prognostic and periodontal treatment. Cigarette smoking represents a predictive factor in the activity and evolution of the periodontal disease and smoking cessation is an important part of the therapy plan.

REFERENCES