LOCALIZED AGGRESSIVE PERIODONTITIS AMONG TWINS - A CASE REPORT

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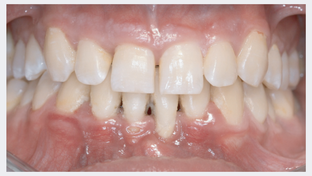
INTRODUCTION

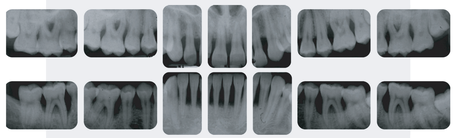
It has been suggested that immune response influences the development of localized aggressive periodontitis in young adults by guiding the bacteria to a greater pathogenicity. In this context, the genome of the patient could play a role.

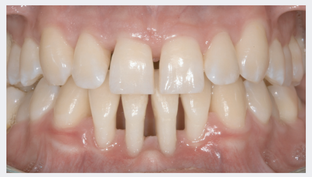
CLINICAL CASE

BRICE



INITIAL CLINICAL VIEW AND RADIOGRAPHS





CLINICAL VIEW AT ONE YEAR
AFTER ETIOLOGIC TREATMENT

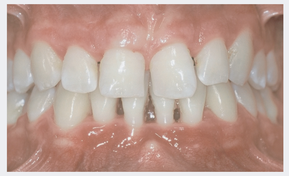
Brice and Florent are 20 years old twins brothers, in good general health.
Both have a localized aggressive periodontitis characterized by a moderate to advanced alveolar bone destruction around incisors and first molars.

Clinical parameters, including periodontal pocket depth and clinical attachment level measured before treatment, are similar in both brothers. The microbiological tests show high levels of Aggregatibacter actinomycetemcomitans in the deepest pockets.

Etiological treatment is composed of oral hygiene teaching and two sessions of scaling and root planning. It is carried out under antibiotic treatment (amoxicillin and metronidazole).

At one year postoperative, no periodontal pocket depth exceeds 4 mm. The microbiological sampling around incisors and first molars does not reveal any putative periodontal pathogens.

FLORENT



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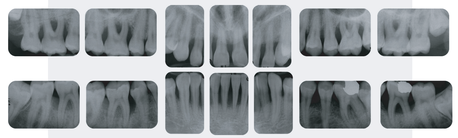
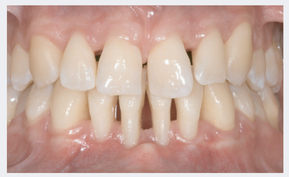




CLINICAL VIEW AT ONE YEAR
AFTER ETIOLOGIC TREATMENT

ETIOLOGY

HOST

GENETICS

Aggregation of the disease in several members of a family is considered as a diagnostic criteria of localized aggressive periodontitis.
The transmission of an autosomal gene coding for a defect of polymorphonuclear fonction has been proposed.

DEFENSE

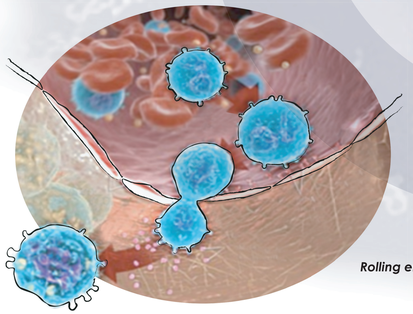
Patients suffering from localized aggressive periodontitis may have/present a defect of the PMN chemotaxis and a decrease of their antibacterial function.

Polymorphism of FC Receptors (FC Gamma RII) present on the PMN is responsible for phagocytosis decrease.

Ratio of T helper/T supressor is low compared to the levels measured in a healthy gingivae or peripheral blood.

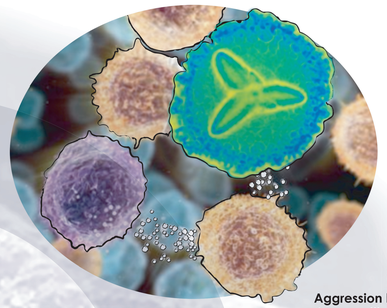
Serum levels of IgG2 are higher in this periodontal patients compared to healthy patients.

Local inflammatory response is characterized by high levels of IL-1, TNF-alpha and PGE2 in crevicular fluid and tissues. PGE2 levels are particularly higher than PGE2 levels among healthy individuals or suffering of chronic periodontitis.



Rolling endothelium

LOCALIZED AGGRESSIVE PERIODONTITIS



Aggression by *Aggregatibacter actinomycetemcomitans*

BACTERIA

PATHOGENS

Bacterial species mostly found in localized aggressive periodontitis are *Aggregatibacter actinomycetemcomitans*.
Virulence factors for *Aggregatibacter actinomycetemcomitans*:
- double membrane whose outer one is rich in endotoxin
- ability to invade tissues and cells
- secretion of factors that inhibit polymorphonuclears chemotaxis
- production of leucotoxins that :
- inhibit host defense
- destroy polymorphonuclears, macrophages, granulocytes and monocytes

LPS:
- induce a cytotoxic reaction towards macrophages
- stimulates bone lysis in the secretion of mediators of inflammation such as PG, IL1-Beta ,et TNF alpha
- promotes the adherence of the bacteria to the mucosa

VIRULENCE FACTORS FOR *AGGREGATIBACTER ACTINOMYCETEMCOMITANS*

It was observed that parents could have the same bacterial strain as children, stressing the possibility of bacterial contamination or transmission parent to child.

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CONCLUSION

Periodontitis are the result of an interaction between bacteria and host response.
The role given to the bacteria continues to dominate our understanding of periodontal disease, while the influence of genetic factors remains unclear. However the clinical case above suggests the major influence of genetics in localized aggressive periodontitis.