“Evidence for risk factors related to a specific form is still weak”

An interview with Dr Cristiano Tomasi, Sweden, on aggressive vs. chronic periodontitis

Evidence for risk factors related to a specific form is still weak. With the identification of risk factors associated with both forms of periodontal disease remains difficult.

Daniel Zimmermann: Both chronic and aggressive periodontitis are complex infections. What is the basic microbiology underlying this disease?

Dr Cristiano Tomasi: Probably the most important microbiological feature is the establishment of a sub-gingival biofilm. The evidence suggests that periodontal disease is not related to a specific microorganism but rather to a complex environment of many different species that live in symbiosis. In a susceptible subject, the biofilm challenge will prompt a host response that will lead to the destruction of periodontal support.

It is estimated that between 10 and 15 per cent of adults in developed countries suffer from chronic periodontitis. Are there any figures available for the aggressive form?

This question is not easy to answer. In fact, even for chronic periodontitis, prevalence differs significantly, depending on disease definition and the population studied. Furthermore, most epidemiological studies have only addressed the prevalence of periodontitis, with no distinction between the aggressive and chronic forms.

The range in prevalence when mild cases are included may reach 40 per cent in a population. The prevalence of the aggressive form, according to one study, was four per cent for localised forms and two per cent for generalised forms in a population ranging between the ages of 18 and 50. Other studies have suggested prevalence of severe cases in a young population of up to eight per cent.

Generally speaking, we still lack epidemiological data from studies that directly address this question.

One of the main differences between both forms appears to be the age group in which they commonly occur. Age remains an important parameter for distinguishing the two forms. While severe cases at age 20 are commonly recognised as aggressive, those at 60 are mainly diagnosed as chronic. The diagnosis of
both forms, however, is clinical and basically follows the same steps.

A problem is that in many cases it is not actually possible to identify the age at which the periodontal disease started, so it is not easy to draw conclusions on clinical features related to age of onset.

**What are the main challenges in differentiating between both forms?**

I really think that the most important thing is to diagnose and intercept periodontitis as early as possible. A screening probing can reveal initial periodontal destruction and signs of inflammation quite easily, allowing for an early and effective intervention.

Marking the fine distinction between aggressive and chronic forms could be another step, but the implications of these studies would be more interesting for researchers than for clinicians. If we are successful in our treatment, is it really important what we call the disease? And if we are not successful, do we blame the name of the disease?

One clinical consideration may be that the systemic use of antibiotics as an adjunctive treatment is supported by studies on aggressive cases, but I think that with regard to the problem of microbial resistance induced by excessive use of antimicrobials, this approach should never be the choice for initial treatment, but be considered after re-evaluation to accompany mechanical retreatment of the remaining diseased sites. This view, however, is not shared by some periodontologists, who view the first treatment attempt as the important one.

**Both forms of periodontitis share risk factors. What are the most common?**

Periodontal disease is clearly the result of an unbalanced host response to the microbial challenge. It is therefore obvious that the genetic set-up of the host and the microbial composition of the biofilm are recognised as risk factors for the development of the disease.

Environmental factors like smoking and stress have also been correlated with the progression of the disease and its most severe forms.

It is a more difficult task to determine risk factors that are clearly associated with one of the two forms of the disease. A few studies have shown specific bacteria to be associated with aggressive forms, but others have also reported aggressive forms without the presence of those bacteria. The same thing happened with specific genetic polymorphisms. New insights are expected to come from epigenetic studies, in which the activation of specific genes is related to local environmental factors.