What is periodontitis? Aetiology and pathogenesis

Aetiology is defined as the cause or origin of a disease or condition. Pathogenesis refers to the biological and physiological processes that occur in the body as a disease develops and progresses.

Whilst the plaque biofilm (film on the tooth surfaces) is a prerequisite for periodontitis (irreversible gum disease), it alone is not enough to initiate the disease. The pathogenesis of periodontitis involves a complex interplay between a dysbiotic (unbalanced) plaque biofilm and the host (the individual's) immune-inflammatory response, leading to tissue destruction.

Periodontitis begins with gingivitis – inflammation, due to plaque biofilm accumulation, which is reversible. In some individuals, this progresses to periodontitis - irreversible destruction of tooth supporting structures (periodontium).

Initiation

Neutrophils (a key component of the body's immune system and the first responder to infection or injury) are present in the gingiva (gum), maintaining normal homeostasis in humans without clinical signs of inflammation. When the plaque biofilm is not disrupted at the gingival margin, it accumulates resulting in gingivitis (inflammation of the gums). Gingivitis involves the loss of collagen locally. However, it is reversible upon resolving the inflammation.

Progression

Periodontitis is initiated when the normal low-grade inflammation in the gingival tissues, during gingivitis, fails to manage the biofilm effectively. This plaque biofilm consists of a range of bacterial communities (microbiome), with certain species becoming more frequent as the disease progresses. The initial stage of the disease process involves a shift from a commensal (harmless) microbiome to a more pathogenic (has the ability to cause disease) one. This change, known as dysbiosis, is crucial in disease progression. Dysbiosis is not only an overgrowth of specific pathogens (bacteria) but also includes alterations in the composition of the microbiome, metabolic activities, and interactions.

The host's immune-inflammatory response plays a double role – it aims to eliminate the threat of the harmful bacteria but causes an over-reaction of the immune system which in turn can cause collateral tissue damage to the periodontium. This inflammation, characterised by increased inflammatory mediators and processes in the body, further promotes dysbiosis and creates a self-sustaining cycle of destruction.

The disease's progression is influenced by factors such as genetic predisposition, environmental factors (e.g. variations in the microbiome), systemic diseases and lifestyle (e.g. smoking). Therefore, effective management requires addressing both the plaque biofilm, the host's immune-inflammatory response and the factors that influence it.

References

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