

Periodontitis leading to pancreatic cancer: A proposed mechanism

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Abstract

Periodontal disease, a common chronic oral inflammatory disease is characterized by destruction of soft tissue and bone of tooth. Atherosclerosis starts early in life, since disease progression is usually slow, clinical symptoms or hospitalization are rare before 40 years of age.

Epidemiological associations between periodontitis and cardiovascular disease have been reported by several studies^{1,2}. Periodontitis and atherosclerosis have complex etiologies, genetic and gender predispositions and may share pathogenic mechanisms as well as common risk factors. It is becoming increasingly clear that infections and chronic inflammatory conditions such as periodontitis may influence the atherosclerotic process. The crucial casual relation might be established by prospective treatment studies, which elucidate the connection between treatment of poor health and systemic inflammatory marker^{3,4}. Haemostatic and theological variable are associated with both prevalent and incident cardiovascular disease, and may be mechanisms through which risk factors such as smoking, hyperlipidemia and infections may promote vascular events. Low grade chronic infections are increasingly being recognized as potential instigators of systemic diseases. Periodontal disease manifests as a prevalent chronic infection impinging throughout the entire adult life in a significant proportion of the population and is probably a significant risk factor for cardiovascular disease in the population as a whole and particularly in certain groups^{1,2,3,4,5}. The oral cavity provides a gateway between the external environment and the gastrointestinal tract, and it facilitates both food ingestion and digestion. Oral hygiene and tooth loss can potentially affect gastrointestinal flora and nutritional status, and they have implications for the development of chronic diseases. Poor dental health, tooth loss, or both have been associated with increased risk for gastrointestinal malignancies, including oral esophageal and gastric cancers^{6,7,8,9,10}.

Several mechanisms have been proposed for relationship of periodontitis leading to pancreatic disease^{11,12}. We believed that Inflammation appears to play an important role in pancreatic cancer pathogenesis, although the inflammatory mediators that lead to the development of pancreatic cancer (important inflammatory marker C-reactive protein). An association between periodontal disease and systemic inflammation has been observed using biomarkers. We hypothesize that periodontal disease may lead pancreatic carcinogenesis through inflammation.

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