Gingival Crevicular Fluid Myeloperoxidase in Periodontitis and Pancreatic Cancer

Níveis de Mieloperoxidase no Fluido Crevicular Gengival na Periodontite e no Câncer de Pâncreas

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RESUMO

Objetivo: Determinar a existência de associação entre os níveis de mieloperoxidase no fluido crevicular gengival (MFCG), periodontite e câncer de pâncreas.

Método: Neste estudo duplo cego randomizado, 66 indivíduos de classe média foram divididos nos quatro grupos a seguir e tiveram os níveis de MFCG analisados: câncer de pâncreas com periodontite, câncer de pâncreas sem periodontite, periodontite sem câncer de pâncreas, e normal (33:33 mulheres:homens; faixa etária: 30-65 aos). A periodontite foi definida pela presença de pelo menos 7 dentes com profundidade de sondagem (PS) maior que 5 mm e perda óssea radiográfica perceptível maior que 30% nos sítios dentários, determinada por meio de uma série de radiografias intra-orais de boca completa. Medidas clínicas da severidade da doença periodontal, tais como sangramento à sondagem (SS), PS e perda do nível de inserção clínica (NIC), foram determinadas usando uma sonda periodontal convencional.

Resultados: Os níveis médios de MFCG foram significantemente maiores nos pacientes com câncer de pâncreas e periodontite, em comparação aos outros pacientes. Os níveis médios de MFCG em pacientes sem câncer de pâncreas e pacientes com câncer de pâncreas e sem periodontite foram 0,68 ± 0,32 U/mL and 0,84±0,32 U/mL, respectivamente. Houve correlação positiva entre os níveis de MFCG e as percentagens de SS, NIC e PS. Pacientes com câncer de pâncreas e periodontite apresentaram maiores percentagens de SS, PS e NIC em comparação aos pacientes sem câncer de pâncreas (p<0,05).

Conclusão: A análise dos níveis de MFCG pode ser útil na detecção do risco de câncer de pâncreas em pacientes com periodontite.

ABSTRACT

Objective: To determine the association between levels of GCF myeloper oxidase (GM), periodontitis and pancreatic cancer.

Method: This was a double blind randomized study. Sixty six subjects middle class divided into four group such as pancreatic cancer with periodontitis, pancreatic cancer without periodontitis, non pancreatic cancer with periodontitis and normal (33:33 M:F; range 30-65 years) were selected for the study were recruited for the study and GM levels were analyzed. Periodontitis in patients was defined of the presence of at least seven teeth with probing depth > 5mm and demonstrable radiographic bone loss >30 percent of tooth sites by a full mouth intraoal radiographic series. Clinical measures of the severity of periodontal disease, such as bleeding on probing, probing depth (PD) and loss of clinical attachment level (CL) were determined using a conventional periodontal probe.

Results: GM levels were significantly higher in pancreatic cancer with periodontitis as compared to others. The mean GM level in non pancreatic cancer and pancreatic cancer without periodontitis were 0.68±0.32U/ml and 0.84±0.32 U/ml, respectively. A positive correlation was noted between GCF meyloperoxidase and percentage of BP, CL and PD. Pancreatic cancer with periodontits, pancreatic exhibited greater BP, PD and CL as compared to non- pancreatic cancer (p<0.05).

Conclusion: Thus, measurement of GM may prove to be useful in detection risk of pancreatic cancer in periodontitis patients.

INTRODUCTION

Periodontal disease, a common chronic oral inflammatory disease, is characterized by destruction of soft tissue and bone. The crucial casual relation might be established by prospective treatment studies, which elucidate the connection between treatment of poor dental health and systemic inflammatory markers[1,2].

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 thus 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[3-7]. It has been reported a positive association between self-reported periodontal disease and risk of pancreatic cancer in a cohort of 51 529 predominantly white US men aged 40- 75 years in the Health Professionals Follow- Up Study (HPFS)[8].

It has been reported that myeloperoxidase levels increase in pancreatic cancer[9]. Biomarkers of periodontal activity may be obtained from potential proteolytic and hydrolytic enzymes of inflammatory cell origin[10].

It has been also observed that increased myeloperoxidase which is present in azurophilic granules of polymorphonuclear neutrophils, activity in periodontitis as compared to controls[11]. Myeloperoxidase considered a promising marker of periodontal inflammation[12,13].

Hence, the present study was planned to determine the relationship between GCF myeloperoxidase (GM) levels in periodontitis and pancreatic cancer.

MATERIAL AND METHODS

This was a double blind randomized study. Sixty six subjects middle class (14:12 M:F; range 30-65 years) were selected for the study. Patients were excluded from the study if they had alcholic or chronic smoker. Thrity five diagnostic patients middle class non smoker and non alcoholic (20:15; range 33-62 years) of pancreatic cancer were selected and oral examination were done without any other systemic diseases.

The middle class periodontal cases comprised 20 subjects (10:10 F:M; range 24-60 years). The control middle class (healthy), non periodontal cases comprised 11 subjects (6:5 F:M; range 28-63 years), none of whom exhibited clinical signs over 5 mm or any clinical attachment loss. In none of the participants was

cardiovascular disease or any other ongoing general disease or infections diagnosed.

Periodontitis in patients was defined of the presence of at least seven teeth with probing depth > 5mm and demonstrable radiographic bone loss >30 percent of tooth sites by a full mouth intraoal radiographic series. All participants had chronic periodontitis who had not received any surgical therapy previously. All subjects were systemically healthy, with no medical conditions that would affect their participation in the study.

The exclusion criteria was a course of anti-inflammatory or antimicrodial therapy within the previous 3 months, a history of regular use of mouth washes, use of any vitamin supplementation or mucosal lesions, chemotherapy, rediation therapy, or medications that cause xerostmia. Informed consent was obtained from the subjects.

Clinical measures of the severity of periodontal disease, such as bleeding on probing, probing depth (PD) and loss of clinical attachment level (CL) were determined using a conventional periodontal probe (Hu-Friedy Chicago, IL). At six sites around each tooth (Mesio-buccal, midbuccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual, excluding third molars. The probe was directed parallel to the long axis of the tooth. Clinical loss of attachment measurement were made from the cemento-enamel junction to bottom of the sulcus. GCF sampling and processing were done as has been described in detail elsewhere[12]. MPO activity (GM) is analysed as previous studies[13].

Relationships between GM; probing depths and bleeding on probing, were analysed using a performed during SPSS (version 11.0, Chicago, USA).

RESULTS AND DISCUSSION

he mean GM level in non pancreatic cancer and pancreatic cancer without periodontitis were 0.68±0.32U/ml and 0.84±0.32 U/ml respectively (Table 1, p<0.05). To our knowledge, this is the first study to evaluate GCF meyloperoxidase level in periodontitis in pancreatic and non pancreatic cancer.

In the present study, high level as GCF meyloperoxidase were observed in pancreatic cancer patients and level were still higher in pancreatic cancer as compared to non- pancreatic cancer (Table 1, p<0.05).

A positive correlation was noted between GCF meyloperoxidase and percentage of BP, CL and PD (Table 1). In the present study, pancreatic cancer with periodontits, pancreatic exhibited greater BP, PD and CL as compared to non- pancreatic cancer (Table 1, p<0.05).

Table 1. Level of GCF myeloperoxidase (in U/ml), probing depth and bleeding on probing in pancreatic cancer with periodontitis (A), pancreatic cancer without periodontits (B), no pancreatic cancer with periodontitis (C), Normal healthy ie no pancreatic cancer and periodontitis (D) mean±S.D.

Parameters	A	B	C	D
GCF myeloperoxidase (in U/ml)	1.18 ±0.29	1.02±0.24	0.84±0.32	0.68±0.32
Probing depth (in mm)	5.63±0.13	1.62±0.23	3.56±0.33	72±0.12
Bleeding on probing(in%)	6.53±0.6	27.6±0.30	5.01±0.1	9.4± 0.30
Clinical loss of attachment	4.82±0.28	.23±0.14	2.8±0.22	.72±0.13

Although MPO is involved in the pathogenesis of inflammatory periodontal diseases and pancreatic cancer, it is also found in clinically healthy sites in lower levels than the periodentally diseases sites[9,13,14].

To the best of our knowledge, there is no published study determining the effects of pancreatic on GM levels. Therefore, we could not compare the results of present study with other results. Several mechanisms could potentially explain the observations from this study. Inflammation appears to play an important role in pancreatic cancer pathogenesis[8], although the inflammatory mediators that lead to the development of pancreatic cancer remain poorly defined.

An association between periodontal disease and systemic inflammation has been observed using biomarkers[15]. We hypothesize that periodontal disease may promote pancreatic carcinogenesis through inflammation through oxidative enzyme such as myeloperoxidase. While some authors belived that periodontal disease could influence pancreatic carcinogenesis through increased generation of carcinogens, namely nitrosamines[16].

Nitrosamines and gastric acidity have been hypothesized to have an important role in pancreatic cancer; numerous studies support this hypothesis[17]. We were not included more oxidative stress marker and more samples size as well as the same marker in saliva and serum to clarified the correlation between two diseases. GCF myeloperoxidase can be easily measured and may prove to be useful in identifying patients at risk of pancreatic cancer.

Further study is required on large scale while considering the risk factors and effect of periodontitis treatment on myeloperoxidase level in saliva and serum.

CONCLUSION

GM may prove to be useful in detection risk of pancreatic cancer in periodontitis patients this study also supports the inflammatory mechanism of pancreatic cancer.

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