ASSOCIATIONS BETWEEN PERIODONTAL DISEASE AND CORONARY ARTERY DISEASE IN HIV-POSITIVE INDIVIDUALS

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Background: HIV-positive individuals are at increased risk for coronary artery disease (CAD). Periodontal disease, and IgG antibody levels for periodontal pathogens, have been associated with CAD in the general population. People living with HIV (PLHIV) have an increased prevalence of periodontal disease, but it is not yet known whether antibody levels for periodontal pathogens are associated with CAD in this population.

Methods: Twenty-four HIV-positive individuals with a history of CAD (cases) were age- and sex-matched 1:2 with 46 HIV-positive individuals without CAD (controls). Antibody levels to periodontal pathogens; Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, and Fusobacterium nucleatum, as well as markers of inflammation; sCD14, CXCL10, and hsCRP, were compared between cases and controls using enzyme-linked immunosorbent assays on stored samples from the 12 months prior to diagnosis of CAD in the cases.

Result: P. gingivalis IgG levels were significantly higher in cases (median 1.48 g/mL [IQR 1.06-2.05]) than controls (0.70 g/mL [IQR 0.35-1.24], *p*<0.001), which remained significant following adjustment for traditional cardiovascular risk factors and HIV viral load (p<0.001). There was no association with A. actinomycetemcomitans IgG antibody levels (cases, median 3.86 g/mL [IQR 3.19-4.72]; controls, 3.34 g/mL [IQR 2.59-4.07], p=0.050) nor between F. nucleatum antibody levels and CAD. sCD14 levels were higher in cases compared with controls (median 3.45 g/ml [IQR 3.03-4.11] vs 2.65 g/ml [IQR 2.32-2.99] p<0.001), while CXCL10 and hsCRP levels were not different between groups.

Conclusions: Plasma levels of IgG antibodies to P. gingivalis were significantly higher in HIV-positive individuals with CAD compared with controls, suggesting that periodontal bacteria and poor oral health may be contributing to CAD in PLHIV. Further research is needed to determine if oral health interventions have the capacity to reduce CAD in PLHIV.

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