

Review of: "Periodontal disease in association with Myocardial Infarction with Non-Obstructive Coronary Arteries and Microvascular Coronary Artery Disease"

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Potential competing interests: No potential competing interests to declare.

This is an interesting paper aimed at doctors many of whom will have little knowledge about the importance of the links between periodontal and heart disease and also diabetes mellitus. I would have liked to see a mention of the major study from Scotland confirming these links. The supplementary tables should be included in the main text and the discussion expanded.

The authors have concentrated on the theory that it is bacteria entering the circulation causing endothelial damage. The other concept is that it is circulating inflammatory markers themselves that are the initiators, and these include not only cytokines but C-reactive protein and tissue necrosis factor among others.

Mention should be made that periodontal disease is not a classic bacterial infection because it does not meet Koch's postulates and no single organism has ever been implicated. It is believed to be a hypersensitivity reaction to toxic products from Gram negative bacteria in mature dental plaque.

I agree that it was a good plan to use a single dental examiner to reduce intra-examiner variability but would have expected that there were checks taken. One method is the re-examination of a random selection of previous subjects and comparison of results. If none had been done this could be an error in their methodology.

The illustrations of the way periodontal disease developed were helpful, but there should have been more written about the scores recorded and what they mean in relation to the clinical findings using the WHO descriptions for each number, and how the final assessment was made. This includes showing that the scores were taken over six areas of the mouth, sextants, and that the highest score in each area was first used, and then presumably the highest score from those six to determine the periodontal status.

The authors should look at the way they have used the periodontal score to assess risk. I disagree with their classification because it is different from previously published others regarded as an international standard for future research (see Yoshioka, 2020, Turner, 2022, Turner & Bouloux, 2023).

The preferred classification is 0-1, low risk, 2-3 medium risk, and 4 or 4* high risk because score 2, calculus, is for both supra- and sub-gingival types, the latter of which unless professionally removed will continue to retain biofilm, and develop further towards the apex of the tooth over time as the inflammatory process continues and exacerbate the disease process.

It follows that if the above classification of dental risks was used there could be significant changes in their results.

Yoshioka M et al. → <https://doi.org/10.1155/2020/4042129>

Turner CH → <https://doi.org/s41415-022-5029-5>

Turner CH, Bouloux P-M → <https://doi.org/10.15277/bjd.2023.403>