Oral Infection, Periodontitis and Atherosclerotic Cardiovascular Disease

A focused evidence review of periodontitis, chronic apical/endodontic infection, atherosclerosis, stroke and myocardial infarction

Literature reviewed through 18 September 2026

Scope. This document is an academic literature review. It distinguishes epidemiologic association, biological mechanism, direct microbiological observations, and intervention evidence. It does not treat a popular video or book as primary evidence, and it does not infer individual diagnosis or treatment.

1. Executive summary

A substantial peer-reviewed literature supports an association between periodontitis and atherosclerotic cardiovascular disease (ASCVD), including coronary heart disease, myocardial infarction (MI), carotid atherosclerosis and stroke. Recent longitudinal meta-analysis estimates are generally modest at the population level but consistent, and several analyses report stronger associations with more severe periodontal disease. The 2026 American Heart Association (AHA) scientific statement treats periodontal disease–ASCVD as a serious research and prevention topic, while retaining the distinction between association and demonstrated causation. [1–3]

Two related but distinct oral disease literatures must be separated. **Marginal periodontitis** is chronic inflammatory destruction around the supporting tissues of teeth. **Apical periodontitis (AP)**, including chronic apical periodontitis (CAP), is inflammation around the tooth root apex, commonly associated with pulpal/endodontic infection and often asymptomatic. The latter is particularly relevant to claims about "silent" dental infections. Systematic reviews and meta-analyses now report an association between AP/CAP and cardiovascular disease and atherosclerosis, but certainty remains limited by heterogeneity and observational design. [12–15]

Mechanistic evidence is biologically plausible and multi-pathway: intermittent bacteremia, bacterial products such as lipopolysaccharide, systemic inflammatory signalling, endothelial activation/dysfunction, altered coagulation/platelet responses, and possible local microbial participation in plaque or thrombus biology. Periodontal treatment can improve endothelial function and inflammatory surrogate markers, but randomized evidence has not yet shown that periodontal therapy prevents hard cardiovascular endpoints such as MI or stroke. [21–23]

A striking 2013 study of 101 STEMI patients found bacterial DNA characteristic of endodontic infection in 78.2% of aspirated coronary thrombi and periodontal-pathogen DNA in 34.7%; in a 30-patient dental imaging subgroup, periapical abscesses were associated with oral viridans-streptococcal DNA-positive thrombi (OR 13.2, 95% CI 2.11–82.5). However, a 2020 study of 109 STEMI patients detected targeted bacterial DNA in only 10/109 thrombi (9.2%). The difference is important: direct microbial/thrombus evidence exists, but detection rates are method-sensitive and do not by themselves establish that oral infection caused an individual MI. [16,17]

2. Terminology and why it matters

The older literature often uses "chronic periodontitis." Modern periodontal classification no longer relies on that chronic/aggressive dichotomy, instead staging and grading periodontitis. For literature searches, therefore, useful terms include *periodontitis*, *periodontal disease*, *clinical attachment loss*, *probing depth*, *bleeding on probing*, and *radiographic bone loss*.

The phrase "subclinical periodontitis" is not a standard modern diagnostic category. For asymptomatic disease, the endodontic literature is especially relevant: AP may be chronic and asymptomatic,

and its worldwide prevalence is substantial. [11]

3. Marginal periodontitis and cardiovascular disease

3.1. Longitudinal cardiovascular risk

Larvin et al. reviewed 32 longitudinal cohort studies (30 in meta-analysis) and reported higher incident CVD risk in people with periodontal disease (RR 1.20, 95% CI 1.14–1.26). Severe periodontal disease showed RR 1.25 (1.15–1.35). Stroke risk was RR 1.24 (1.12–1.38) and coronary heart disease risk RR 1.14 (1.08–1.21). [3]

A 2025 stroke meta-analysis of 22 observational studies found OR 2.22 (1.48–3.34) in case-control studies and RR 1.49 (1.23–1.80) in cohorts. Prospective cohorts followed longer than 10 years showed RR 1.57 (1.35–1.84) with substantially lower heterogeneity than the overall cohort pool. The authors explicitly concluded that the data support association rather than proof of causation because residual confounding and heterogeneity remain. [4]

A 2026 meta-analysis focused on ischemic stroke and periodontitis severity reported a stronger association with increasing periodontal severity; moderate disease was associated with OR 1.63 (1.16–2.29) and severe disease with OR 4.19 (3.14–5.59) in the relevant subgroup analyses. These effect measures should not be naively compared across designs, but the severity-response signal is noteworthy. [5]

For acute myocardial infarction, a 2026 meta-analysis of 17 observational studies (146,001 participants; 3,199 AMI cases) gave pooled OR 1.84 (1.51–2.23), but heterogeneity was high (I^2=83.3%). Publication-bias correction by trim-and-fill reduced the estimate to OR 1.26 (1.03–1.55), illustrating why a single headline percentage exaggerates certainty. [6]

3.2. Carotid atherosclerosis and subclinical vascular disease

The ARIC study reported higher odds of carotid intima-media thickness (IMT) $\geq$ 1 mm with moderate periodontitis (unadjusted OR 1.40) and severe periodontitis (OR 2.09); after multivariable adjustment, severe periodontitis remained associated (OR 1.31, 95% CI 1.03–1.66). [8]

A 2022 meta-analysis of 8,558 participants in seven cross-sectional studies found increased CIMT with periodontitis (OR 1.42, 95% CI 1.16–1.75) and severe periodontitis (OR 1.70, 1.24–2.33). [9] An updated 2025 meta-analysis of carotid atherosclerosis reported pooled OR 1.97 (1.64–2.36), with publication-bias-corrected OR 1.30 (1.06–1.58). [7]

3.3. Stroke versus gingivitis

An earlier cohort meta-analysis reported stroke risk RR 1.63 (1.25–2.00) with periodontitis and RR 1.39 (1.13–1.65) with tooth loss, but did not find a significant increase for gingivitis. This supports keeping gingivitis separate from established destructive periodontitis in evidence synthesis. [10]

4. Chronic apical/endodontic infection and cardiovascular disease

The endodontic literature is directly relevant to the idea of an asymptomatic or “silent” oral infection. Berlin-Broner et al. (2017) reviewed 19 studies: 13 reported a positive AP–CVD association, but the overall evidence was rated moderate-to-low and causality could not be established. [12]

Noites et al. (2022) subsequently performed a systematic review and meta-analysis of adult AP and cardiovascular disease. Cross-sectional evidence suggested a positive association, while case-control/cohort pools were less stable; heterogeneity and limited longitudinal evidence constrained causal inference. [13]

A 2025 umbrella review synthesized ten systematic reviews, including five meta-analyses spanning study populations from 1,108 to 673,083 participants. Pooled estimates were approximately RR 1.32 (95% CI 1.00–1.62) and OR 1.83 (1.33–2.53), but between-study heterogeneity ranged from I^2=54% to 100%. [14]

A separate 2025 systematic review/meta-analysis specifically examined atherosclerosis and CAP. Five eligible studies yielded an overall OR 2.94 (1.83–4.74) for CAP prevalence among patients with atherosclerosis, but the evidence was rated low certainty and the design does not establish direction of causation. [15]

5. Direct microbiological evidence: plaque and coronary thrombus

5.1. Coronary thrombus studies

Pessi et al. (2013) examined thrombus aspirates and arterial blood from 101 STEMI patients undergoing primary PCI. The median total bacterial DNA signal in thrombi was 16-fold higher than in paired arterial blood. DNA typical of endodontic infection, principally oral viridans streptococci, was detected in 78.2% of thrombi; periodontal-pathogen DNA was detected in 34.7%. Electron microscopy identified bacteria-like structures in all nine thrombi examined by that method and whole bacteria in three. In the 30-patient subgroup receiving panoramic dental imaging, periapical abscesses were associated with oral viridans-streptococcal DNA-positive thrombi (OR 13.2, 95% CI 2.11–82.5, p=0.004). [16]

The peer-review-safe interpretation is narrower than "78.2% of heart attacks were caused by infected root canals." The study found microbial DNA and a dental-pathology association in a selected STEMI thrombus cohort; it did not establish a complete causal chain in each patient.

Piñon-Esteban et al. (2020) tested aspirated thrombus material from 109 STEMI patients using probes for 12 organisms. Target bacterial DNA was detected in 10/109 (9.2%) thrombi: viridans streptococci in six (5.5%), *Staphylococcus aureus* in two, *Porphyromonas gingivalis* in one (0.9%) and *Prevotella intermedia* in one (0.9%). No bacterial DNA was found in paired peripheral blood. [17] The large difference in detection rate relative to Pessi et al. indicates strong sensitivity to assay design, microbial targets, cohort and laboratory methodology.

5.2. Atherosclerotic plaque studies are mixed

Haraszthy et al. (2000) reported periodontal-pathogen DNA in human atheromatous plaques, including *P. gingivalis*. [18] Other controlled carotid studies failed to detect the target periodontal organisms in atheromas despite finding them in periodontal pockets. Cairo et al. (2004) found no target periodontal bacterial DNA in carotid samples, and Aimetti et al. (2007) likewise detected bacterial DNA generally in 31/33 endarterectomy specimens but none of the specified periodontal pathogens. [19,20]

Therefore, persistent colonisation of atheroma by classical periodontal pathogens is not consistently demonstrated. A systemic inflammatory/bacteremic mechanism does not require permanent plaque colonisation.

6. Mechanistic bridge: inflammation and endothelial dysfunction

The mechanistic model can be expressed as several interacting routes rather than a single pathway:

1. chronic local periodontal/endodontic inflammation and repeated epithelial barrier disruption;
2. intermittent bacteremia and exposure to microbial products;
3. systemic inflammatory signalling (including CRP/IL-6-associated pathways), oxidative stress and endothelial activation;

4. impaired endothelial-dependent vasodilation and vascular dysfunction;
5. platelet/coagulation effects and, in susceptible atherosclerotic lesions, possible contribution to plaque instability or thrombosis.

A randomized NEJM study of 120 patients with severe periodontitis showed an acute inflammatory/endothelial deterioration immediately after intensive therapy, followed by improved flow-mediated dilation at 60 and 180 days; the six-month between-group FMD difference was 2.0 percentage points (95% CI 1.2–2.8). [21]

A 2026 systematic review/meta-analysis of FMD found periodontal therapy associated with a pooled improvement of 3.3 percentage points (95% CI 1.7–4.9; I^2=77%) and reduced CRP, while emphasizing substantial heterogeneity and the small number of quantitative studies. [22]

These are vascular/inflammatory surrogate outcomes. They strengthen biological plausibility but are not equivalent to demonstrating fewer myocardial infarctions or strokes.

7. Does periodontal treatment prevent MI or stroke?

This is where the evidence is weakest. The 2022 Cochrane review found only two eligible randomized controlled trials, both at high risk of bias. Evidence for primary prevention was very low-certainty and inconclusive; for secondary prevention there was no reliable evidence allowing a conclusion. [23]

Thus the current evidence hierarchy is roughly:

Strongest/most consistent	Association of established periodontitis with ASCVD/stroke/CHD; biological plausibility; endothelial/inflammatory abnormalities.
Moderate but heterogeneous	Severity-response signals; association of AP/CAP with CVD/atherosclerosis; direct bacterial DNA observations in thrombi.
Not yet established	That periodontal or endodontic therapy reduces hard cardiovascular event rates; that oral infection causes most MIs; that bacterial plaque colonisation is necessary for the association.

8. Assessment of the Berg/Levy material

The user-supplied Eric Berg video (YouTube ID `AgD4hrdAYzk`) and Thomas E. Levy's 2017 book *Hidden Epidemic: Silent Oral Infections Cause Most Heart Attacks and Breast Cancers* are secondary sources and are not used here as evidentiary foundations. Levy's central subject – clinically silent oral/endodontic infection as a possible contributor to cardiovascular disease – is a legitimate peer-reviewed research topic. The stronger proposition in the book title, that silent oral infections cause *most* heart attacks, is not established by the present clinical evidence.

A particularly important source apparently underlying popular presentations is Pessi et al. (2013). Its 78.2% figure refers to detection of DNA typical of endodontic infection in coronary thrombi, not to a demonstrated attributable fraction of myocardial infarctions caused by infected teeth. [16]

9. Overall conclusion

The most defensible synthesis is:

> **Periodontitis is consistently associated with incident atherosclerotic cardiovascular disease, ischemic stroke and myocardial infarction in observational research, with evidence of severity-response and plausible biological mechanisms. Chronic apical/endodontic infection is also associated with cardiovascular**

> **disease and atherosclerosis in a growing but more heterogeneous literature. Direct studies have detected oral/endodontic bacterial DNA in some coronary thrombi and atheromatous material, although detection is inconsistent across laboratories and cohorts. Periodontal treatment improves several inflammatory and vascular surrogate endpoints, but randomized evidence has not yet established prevention of myocardial infarction or stroke.**

This formulation preserves the substantial positive evidence without converting association, mechanism or microbial detection into a claim of proven individual causation.

Full references

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