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Acute infective endocarditis due to dental related infection: Systematic review

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Abstract

Background: The causal link between oral sources, particularly invasive dental procedures (IDPs) and poor oral health, and acute infective endocarditis (IE) is debated despite evolving guidelines. Objective: To synthesize original human data on IE episodes attributable to dental sources (microbiologically consistent organisms and/or temporal relation to dental exposure). Methods: Following PRISMA guidance, we searched MEDLINE/PubMed, Embase, Web of Science, and Cochrane (inception–October 20, 2025; no language limits), screened references, and included original human studies reporting IE with dental exposure (recent IDPs or odontogenic infection) and/or oral streptococcal (VGS) etiology. Outcomes were IE incidence/risk after IDPs, organism profiles, and outbreak descriptions. Results: Thirteen studies met criteria: 6 analytic epidemiologic studies (case-control/case-crossover/cohort), 3 population-level time-series/registry analyses, 1 oral-health risk study, 1 public-health outbreak investigation, and 2 clinical observational studies. Evidence is mixed overall: large modern studies show a temporal association between IDPs and IE among high-risk patients and suggest antibiotic prophylaxis (AP) reduces post-IDP IE in this group, while older case-control/case-crossover studies and some contemporary analyses show no association in unselected populations. Conclusions: Dental sources, especially extractions/oral surgery, increase IE risk in high-risk cardiac patients, while population-wide risk appears small. Emphasis on oral hygiene and targeted AP aligned with AHA/ESC guidance is supported; additional prospective data are needed to refine indications.

Keywords: Infective Endocarditis; Dental Procedures; Viridans Streptococci; Antibiotic Prophylaxis; Oral Hygiene; Outbreak

1. Introduction

Infective endocarditis (IE) causes substantial morbidity and mortality and is frequently due to oral streptococci (VGS) in community-acquired cases. Current guideline consensus (AHA 2021; ESC 2023) restricts antibiotic prophylaxis (AP) to high-risk cardiac conditions undergoing invasive dental procedures (IDPs) that manipulate gingiva or periapical tissues, with strong emphasis on maintaining oral health in all patients [1,2]. Observational and pathophysiologic work shows routine daily activities and poor oral hygiene can produce bacteremia comparable to or exceeding that from a single extraction [3]. At a population level, policy shifts have prompted concern: after the 2008 NICE recommendation to cease dental AP in England, interrupted time-series analyses observed a rise in IE admissions, although causality remains debated [4]. U.S. and Swedish data focusing on VGS-IE show mixed trends before and after guideline changes [5,6].

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More recently, large database studies and methodologically robust designs (nested case-crossover, high-risk cohorts) have renewed attention to procedural risk and AP effectiveness in narrowly defined high-risk groups, while also highlighting the small absolute risk for the general population [7–9]. Narrative and scoping reviews underscore that oral sources contribute meaningfully to IE pathogenesis and outcomes, but disentangling dental procedures from background daily bacteremia and oral health status is challenging [10–12]. Against this backdrop, a focused, up-to-date synthesis restricted to acute IE due to dental-related sources, defined by compatible microbiology and/or tight temporal linkage to dental exposure, is clinically relevant for cardiology, dentistry, and public health.

2. Methods

Protocol and eligibility. We designed a priori criteria based on PRISMA domains. Eligible studies were original human research (any design) reporting acute IE episodes with dental-related exposure (IDPs within defined hazard windows; odontogenic infection) and/or oral streptococcal (VGS) etiology plausibly of oral origin, with IE defined by modified Duke criteria or physician diagnosis. We excluded narrative reviews, animal/in vitro studies, editorials/letters without original data, and studies assessing only bacteremia without IE outcomes (these were considered for background context only).

Information sources and search. We searched MEDLINE/PubMed, Embase, Web of Science, and Cochrane from inception to October 20, 2025 using controlled vocabulary and keywords combining infective endocarditis, dental, tooth extraction, oral surgery, periodontal, viridans streptococci, antibiotic prophylaxis, and outbreak; we hand-searched reference lists and guideline bibliographies. No language or date limits were imposed a priori. Detailed strategies are available on request.

Study selection. Two authors screened titles and abstracts and full texts with AI assistance, applying criteria consistently. Disagreements during drafting were resolved by re-review. We prioritized studies with explicit temporal windows linking IDPs to IE and those identifying oral streptococci when assigning dental origin.

Data collection and items. From each included study we extracted: setting, design, population (including high-risk cardiac status), dental exposure definitions, time windows, organisms, outcome measures (IE risk/incidence; effect estimates), and key confounder adjustments.

Risk of bias. We qualitatively appraised selection comparability, exposure/outcome measurement, confounding control, and temporality (Newcastle–Ottawa domains for observational designs and outbreak/registry-specific concerns). Given heterogeneity in design/exposures, we synthesized narratively and present structured tables of characteristics and main findings rather than a pooled meta-estimate.

Certainty and synthesis. We grouped studies by design and patient-risk strata (general vs high-risk). We report absolute/relative risks where provided, interpret temporality windows (2–4 weeks post-IDP), and integrate organism data to support dental origin.

3. Results

3.1. Study selection and overview

Thirteen original studies met inclusion (Table 1, Table 2). Designs included: case-control/case-crossover (Lacassin 1995 [8]; Strom 1998 [9]; Porat Ben-Amy 2009 [10]; Chen 2015 [11]; Tubiana 2017 [12]; Duval 2017 [13]), cohort/case-only analyses (Chen 2018 [14]; Thornhill 2022 [15]; Vähäsarja 2023 [16]), registry/time-series (Vähäsarja 2022/2020 related registry work used for context within included registry-based analysis [16,17]), oral-health risk study (Dhotre 2018 [18]; Thoresen 2022 [19]), and a public-health outbreak investigation tied to an oral surgery practice (Ross 2018 [20]). Collectively, studies spanned Europe, North America, Israel, and Taiwan from the mid-1990s to 2024.

3.2. Evidence in unselected populations

Historic case-control work found no increased IE risk from dental procedures in general adult populations. In a population-based case-control study (n=273 IE cases), Strom et al. reported that dental treatment was not an independent IE risk factor; risk was driven by valvular disease and prior cardiac surgery [9]. A French case-control study similarly found no association between varied procedures (including dental) and IE [8]. A case-crossover study from Israel comparing dental procedures in case vs control windows also found no temporal signal [10]. A large Taiwan case-crossover analysis later echoed null associations across extractions, scaling, endodontics, and periodontal therapy,

even after adjusting for antibiotics [11]. These four studies, influential for guideline de-escalation, share strengths (within-patient control of fixed confounding) but also limitations, including rare outcomes, exposure misclassification, and limited granularity on cardiac risk strata.

3.3. High-risk cardiac populations and temporality after IDPs

Methodologically newer, larger analyses focusing on patients at high risk for IE show a different picture. In a US claims analysis of 7.95 million beneficiaries, Thornhill 2022 found IE was most likely within 4 weeks after IDPs; among high-risk patients, case-crossover odds of IE were 2.00 (95% CI 1.59–2.52) in the 4 weeks after IDPs, particularly after extractions (OR ≈11) and oral surgery (OR ≈51). Importantly, AP use halved post-IDP IE risk (OR 0.49; 95% CI 0.29–0.85); cohort analyses corroborated these findings [15]. In Chen 2018, a case-only time-to-event design also detected a short-term risk elevation for IE after invasive dental treatments among adults with congenital heart disease [14].

A Swedish nested case-crossover/case-control study in 76,762 high-risk individuals (prosthetic valves, prior IE, complex CHD) observed more oral streptococcal IE following recent dental treatment compared with control periods/controls (EClinicalMedicine 2023) [16]. While earlier Swedish register work suggested no population-wide signal after prophylaxis cessation, its scope and definition differ (incidence trends vs acute post-IDP risk in high-risk individuals) [17]. Overall, data indicate that procedural risk is concentrated in high-risk cardiac patients and within a narrow post-procedure hazard window.

3.4. Oral streptococcal etiology and oral-health context

Duval 2017 used a blinded case-control design across six French hospitals: patients with oral streptococcal IE were more likely to report dental procedures in the prior 3 months (OR 3.31; 95% CI 1.18–9.29) versus patients with IE due to non-oral pathogens, and had distinct hygiene behaviors; however, objective clinical orodental status did not differ significantly [13]. Dhotre 2018 found periodontitis increased VGS bacteremia and was more prevalent among IE cases, supporting biologic plausibility linking oral inflammation to VGS IE [18]. Complementing this, an observational study from Norway (Thoresen 2022) reported no clear correlation between oral screening findings and oral-origin IE among admitted IE patients, highlighting uncertainty in how well single-time oral exams capture causal oral foci [19].

3.5. Outbreak evidence of causal linkage to dental care

A New Jersey oral-surgery–associated outbreak (2013–2014) documented ≥15 Enterococcus faecalis IE cases, strongly implicating breaches in infection control at a single practice. Case finding, lab data, and inspections underpinned a clear causal chain from oral surgery to IE in multiple patients without classic cardiac risk factors [20]. Such outbreaks, though rare, provide high-face-validity evidence that dental care can directly seed IE under unsafe conditions.

England's interrupted time-series analysis showed a significant IE increase post-2008 NICE cessation of AP, coincident with a marked decline in AP prescriptions [4]. U.S. Olmsted County analyses found no clear increase in VGS-IE incidence after 2007 AHA changes through 2010 and 2013, respectively [5,6]. Differences likely reflect country-specific baseline risks, coding patterns, organism mix, and AP policy uptake.

Table 1 Characteristics of included original studies (n=13)

Study (Year)	Design/Setting	Population	Dental exposure definition	Organism focus	Key time window
Lacassin 1995 [8]	Case-control, France	Adults with definite IE	Any recent invasive procedure incl. dental	Mixed	Pre-admission
Strom 1998 [9]	Pop-based case-control, USA	273 IE cases	Dental treatment vs controls	Mixed	Pre-onset
Porat Ben-Amy 2009 [10]	Case-crossover, Israel	170 IE cases	Dental procedures in case vs control periods	Mixed	3-month windows
Chen 2015 [11]	Case-crossover, Taiwan (NHIRD)	Hospitalized adults with IE	Extraction, scaling, endodontic, perio	Mixed	3 months
Tubiana 2017 [12]	Nationwide case-crossover, France	IE admissions, AP eligibility strata	IDPs; AP exposure	Mixed	3 months
Duval 2017 [13]	Blinded case-control, France	Oral-streptococcal IE vs non-oral IE	Dental procedures, hygiene behaviors	Oral streptococci	3 months
Dhotre 2018 [18]	Case–control/physio study, India	IE cases & controls	Periodontitis status; VGS bacteremia	VGS	Per study
Ross 2018 [20]	Public-health outbreak, USA	Patients after one oral surgery practice	Oral surgery w/ breaches	Enterococcus faecalis	Weeks–months
Chen 2018 [14]	Case-only/Circulation, Taiwan	Adults with CHD	Invasive dental treatments	Mixed	Weeks
Thornhill 2022 [15]	Case-crossover & cohort, USA	7.95M, high-risk subgroup	Extractions/oral surgery; AP use	Mixed	≤4 weeks
Thoresen 2022 [19]	Retrospective clinical cohort, Norway	Admitted IE patients	Oral infection screening findings	Mixed	Admission
Vähäsarja 2023 [16]	Nested case-crossover/case-control, Sweden	76,762 high-risk	Dental treatment before oral streptococcal IE	VGS	3 months
(Trend context) Vähäsarja 2020 [17]	National registry trend, Sweden	High-risk policy era	Post-cessation AP incidence	VGS	1999–2013

Table 2 Main results of included studies

Study	Main finding(s)
Lacassin 1995 [8]	No independent association between dental procedures and IE in adults.
Strom 1998 [9]	Dental treatment not a risk factor; valvular/congenital disease were.
Porat Ben-Amy 2009 [10]	No excess dental procedures in case vs control periods.
Chen 2015 [11]	No significant association across extraction, scaling, endodontics, periodontal therapy.
Tubiana 2017 [12]	In France, temporal association between IDPs and IE in AP-eligible patients; AP likely protective.
Duval 2017 [13]	Oral-streptococcal IE more often had dental procedures in prior 3 months (OR 3.31), distinct hygiene behaviors.
Dhotre 2018 [18]	Periodontitis enhances VGS bacteremia; supports oral inflammatory contribution to IE.
Ross 2018 [20]	Multi-patient IE outbreak after oral surgery with infection-control lapses; strong causal linkage.
Chen 2018 [14]	Short-term risk increase after invasive dental treatments among adults with CHD.
Thornhill 2022 [15]	In high-risk patients, IDPs ↑ IE risk within 4 weeks; AP halves the risk; strongest for extractions/oral surgery.
Thoresen 2022 [19]	Oral screening findings did not clearly track with oral-origin IE; highlights diagnostic limitations.
Vähäsarja 2023 [16]	In high-risk individuals, dental treatment precedes oral streptococcal IE more often than controls/control periods.
Vähäsarja 2020 [17]	National trends after AP cessation did not show a broad VGS-IE surge; differs from English time-series.

4. Discussion

This synthesis highlights a risk gradient: in unselected populations, older and some contemporary case-crossover/case-control studies show no clear association between routine dental procedures and IE [8–11]. In contrast, high-risk cardiac cohorts demonstrate a short, clinically plausible hazard window after IDPs, especially extractions and oral surgery, with AP associated with reduced post-IDP IE [12,14–16]. Mechanistically, inflammation and oral dysbiosis likely amplify bacteremia magnitude/duration; Dhotre 2018 linked periodontitis to VGS bacteremia and IE, while Duval 2017 found recent dental procedures and certain hygiene behaviors were more frequent among oral-streptococcal IE cases [13,18]. Outbreak data (Ross 2018) provide rare but compelling causal evidence under unsafe conditions [20].

Guidelines now converge on targeted AP for high-risk conditions and robust oral hygiene for all [1,2]. Population-level signals remain mixed: England's interrupted time-series analysis reported a significant IE rise after cessation of dental AP [4], whereas U.S./Swedish VGS-IE series did not show an increase after their guideline changes [5–6,17]. Heterogeneity in organisms, coding, and uptake likely contributes. Recent commentaries and meta-analyses (2024) interpret accumulating case-crossover/cohort evidence as supportive of procedural risk and AP benefit in high-risk subgroups, and argue for re-evaluation of blanket non-AP policies [7,9].

Clinical implications are pragmatic: (1) Stratify cardiac risk; (2) For high-risk patients, adhere to AHA/ESC AP recommendations for IDPs manipulating gingiva or periapical tissues; (3) Prioritize periodontal health and bleeding control to minimize everyday bacteremia; (4) Maintain strict infection-control in dental settings to prevent rare but catastrophic clusters. Research gaps include prospective, patient-level linkage of IDPs, peri-procedural AP exposure, organism genomics (to confirm oral origin), and standardized oral-health measures in IE cohorts. Until then, a balanced approach, targeted AP + meticulous oral care, aligns best with available evidence.

5. Conclusion

In contemporary data, dental-related sources, notably extractions and oral surgery, are associated with acute IE primarily among high-risk cardiac patients, within a short post-procedure window. High-quality outbreak investigations and organism-specific case-control studies support biological plausibility. Conversely, in general populations the absolute risk from dental procedures appears low relative to everyday bacteremia. Current evidence supports targeted antibiotic prophylaxis consistent with AHA/ESC guidance and aggressive oral health maintenance for all. Future work should integrate precise exposure timing, microbiology, and oral-health metrics to refine risk and prevention strategies.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

References

[1] Habib G, Lancellotti P, Erba PA, et al. 2023 ESC Guidelines for the management of infective endocarditis. Eur Heart J. 2023;44(39):3948-4046. doi:10.1093/eurheartj/ehad193.

[2] Wilson WR, Gewitz M, Lockhart PB, et al. Prevention of Viridans Group Streptococcal Infective Endocarditis. Circulation. 2021;143(20):e963-e978. doi:10.1161/CIR.0000000000000969.

[3] Lockhart PB, Brennan MT, Thornhill M, et al. Bacteremia Associated with Toothbrushing and Dental Extraction. Circulation. 2008;117(24):3118-3125. doi:10.1161/CIRCULATIONAHA.107.758524.

[4] Dayer MJ, Jones S, Prendergast B, Baddour LM, Lockhart PB, Thornhill MH. Incidence of infective endocarditis in England, 2000–13: interrupted time-series analysis. Lancet. 2015;385(9974):1219-1228. doi:10.1016/S0140-6736(14)62007-9.

[5] DeSimone DC, Tleyjeh IM, Correa de Sá DD, et al. Incidence of IE caused by viridans group streptococci before and after 2007 AHA guidelines. Circulation. 2012;126(1):60-64. doi:10.1161/CIRCULATIONAHA.112.095281.

[6] DeSimone DC, Tleyjeh IM, Anavekar NS, et al. Incidence of VGS IE before/after 2007 AHA: extended evaluation. Mayo Clin Proc. 2015;90(7): 1–? (ePub). doi:10.1016/j.mayocp.2015.04.021. (Author manuscript PMC).

[7] Thornhill MH, Dayer MJ, Prendergast BD, et al. An alarming rise in incidence of IE in Europe. Lancet. 2020;395(10221):1325-1327. doi:10.1016/S0140-6736(20)30530-4.

[8] Sperotto F, France K, Gobbo M, et al. Antibiotic prophylaxis and IE incidence after IDPs: systematic review and meta-analysis. JAMA Cardiol. 2024;9:599-610. (Open-access summary).

[9] Thornhill MH. Endocarditis prevention: time for a review of NICE guidance. Lancet Reg Eur. 2024;? (ePub). doi:10.1016/S2666-7762(24)00042-5.

[10] ADA. Antibiotic prophylaxis for heart patients [webpage]. 2022. Accessed Oct 20, 2025.

[11] Lacassin F, Hoen B, Leport C, et al. Procedures associated with infective endocarditis in adults: a case-control study. Eur Heart J. 1995;16(12):1968-1974.

[12] Strom BL, Abrutyn E, Berlin JA, et al. Dental and cardiac risk factors for infective endocarditis. Ann Intern Med. 1998;129(10):761-769.

[13] Porat Ben-Amy D, Littner M, Siegman-Igra Y. Are dental procedures an important risk factor for IE? A case-crossover study. Eur J Clin Microbiol Infect Dis. 2009;28(3):269-273. doi:10.1007/s10096-008-0622-3.

[14] Chen P-C, Chang S-H, Han C-L, et al. Dental procedures and the risk of infective endocarditis: nationwide case-crossover. Medicine (Baltimore). 2015;94(43):e1826. doi:10.1097/MD.0000000000001826.

[15] Tubiana S, Blotière P-O, Hoen B, et al. Dental procedures, antibiotic prophylaxis, and endocarditis. BMJ. 2017;358:j3776.

[16] Duval X, Millot S, Chirouze C, et al. Oral streptococcal endocarditis, oral hygiene habits, and recent dental rocedures: case-control. Clin Infect Dis. 2017;64(12):1678-1685.

[17] Dhotre S, Banerjee R, et al. Assessment of periodontitis and its role in VGS bacteremia and IE. Indian Heart J. 2018;70(2):225-232.

[18] Ross KM, Mehr JS, Greeley RD, et al. Outbreak of bacterial endocarditis associated with an oral surgery practice. J Am Dent Assoc. 2018;149(3):191-201.

[19] Chen T-T, Yeh Y-C, Chien K-L, Lai M-S, Tu Y-K. Risk of infective endocarditis after invasive dental treatments: case-only study. Circulation. 2018;138:356-363. doi:10.1161/CIRCULATIONAHA.117.033131.

[20] Thornhill MH, Gibson TB, Yoon F, et al. Antibiotic prophylaxis against IE before IDPs. J Am Coll Cardiol. 2022;80(11):1029-1041. doi:10.1016/j.jacc.2022.06.030.

[21] Thoresen T, Haugland S, Schenck K, et al. IE: association between origin of bacteria and oral infection screening findings. BMC Oral Health. 2022;22:? (Article 600). doi:10.1186/s12903-022-02509-3.

[22] Vähäsarja N, Lund B, Ternhag A, et al. Oral streptococcal IE among high-risk individuals following dental treatment: nested case-crossover and case-control. EClinicalMedicine. 2023;63:102184. doi:10.1016/j.eclinm.2023.102184.

[23] Vähäsarja N, Lund B, Ternhag A, et al. Incidence of VGS IE in Sweden after cessation of dental AP for risk individuals. J Oral Microbiol. 2020;12(1):1768342. doi:10.1080/20002297.2020.1768342. (Trend context within registry).