Systematic Review

The association between poor oral hygiene and infective endocarditis risk in rheumatic heart disease patients: a systematic review

Farah Amiria[1*]
Nur Signa Aini Gumilas[2]

[1]Master Program, Faculty of Medicine, Universitas Jenderal Soedirman, Purwokerto, Indonesia
[2]Department of Biomedic, Faculty of Medicine, Universitas Jenderal Soedirman, Purwokerto, Indonesia

* Correspondence:
farahamiria@gmail.com

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ABSTRACT

Introduction: Infective endocarditis (IE) is a heart condition that may result from subacute bacterial infections. One cause of IE is oral bacterial infections, particularly *Streptococcus* species, which enter the blood vessels through injured gingiva during tooth brushing or dental procedures. Rheumatic heart disease (RHD) is a major predisposing factor in developing countries, with viridans streptococci implicated in its etiology. This background highlights the importance of maintaining oral hygiene, especially in RHD patients, to reduce the risk of IE. This systematic review aims to investigate the association between poor oral hygiene and the risk of IE in patients with RHD. **Method:** A systematic review was conducted in accordance with PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines. Articles published between 2014 and 2024 were sourced from PubMed, Scopus, and Google Scholar using specific keyword combinations related to IE, RHD, and oral health. Inclusion and exclusion criteria were applied based on the PECO framework. **Results:** A total of four eligible studies were identified. The findings suggest that RHD patients with poor oral hygiene are more vulnerable to developing IE due to an increased risk of bacteremia originating from oral infections. **Conclusion:** Poor oral hygiene is associated with an increased risk of infective endocarditis in patients with rheumatic heart disease. Bacterial adherence to damaged heart valves can lead to biofilm formation and vegetations, increasing the likelihood of infection. Preventive dental care and appropriate prophylactic measures should be prioritized in this high-risk population to reduce disease burden.

KEYWORDS

Infective endocarditis, rheumatic heart disease, poor oral hygiene, bacteremia.

INTRODUCTION

Infective endocarditis (IE) is an infection that occurs in the heart, specifically in the endocardial layer. This infection can be caused by bacteria originating from the mouth. IE is caused when bacteria from other body sites enter the heart through the bloodstream, attach to the inner lining of the heart, and damage the heart valves, potentially leading to blood vessel embolism, which can block the blood flow to the heart.[1,2] Despite improved therapeutic management, IE remains a disease with a significant 30-day mortality rate.[2,3]

The morbidity and mortality rates from IE are a considerable global concern. One cause of IE is bacterial infection originating from the oral cavity, specifically *Streptococcus* species.[2,4] These bacteria enter the bloodstream through injured gums during tooth brushing or dental procedures. Once in the bloodstream, these IE-causing bacteria attach to heart valves that have pre-existing issues (such as rheumatic heart disease (RHD), congenital defects, or prosthetic heart valves),

leading to the formation of vegetations. An increase in the number of bacteria attached to this area can enlarge the vegetation and disrupt valvular function. Rheumatic heart disease (RHD) is a primary predisposing factor in developing countries, with viridans streptococci implicated in its etiology.[1,2]

Streptococcus viridans bacteria are key contributors to the occurrence of bacteremia. This heterogeneous group of alpha-hemolytic Streptococci is part of the normal flora in the oral cavity. *Streptococcus* bacteria in the oral cavity are often found in cases of dental caries and pericoronitis.[5,6] These bacteria can enter the bloodstream through minor oral injuries caused by tooth brushing and dental procedures.[7] Once in the bloodstream, these bacteria can travel to the endocardial tissue, attach to it, and cause damage.[8] Damage can also occur in heart valves that possess pre-existing vulnerabilities, which may lead to other comorbidities.[9] Age and systemic diseases (diabetes mellitus and cardiovascular disease) can contribute to poor periodontal conditions. Additionally, the chronic use of drugs such as steroids, anticoagulants, and immunosuppressants can lead to alterations in periodontal tissue.[10]

Periodontal disease is a predisposing factor for cardiovascular disease because it is a low-grade chronic infectious disease and is thought to be a risk factor for atherogenesis and thromboembolism.[11] Oral infections are well-known causes for cardiovascular disease, especially bacteremia originating from the oral cavity, which can be a source of infection leading to heart valve damage and subacute bacterial endocarditis.[12] Oral health is closely related to the overall hygiene status of the oral cavity. Poor oral hygiene, smoking, systemic disease, stress, and obesity can all affect periodontal health.[13] There is a close relationship between the incidence of bacteremia after brushing teeth and indicators of oral hygiene and gingival disease (plaque and calculus).[14] Poor oral hygiene is also associated with a high risk of cardiovascular disease and low-grade inflammation.[15]

Oral diseases such as caries, gingivitis, and periodontitis often result from inadequate oral hygiene (OH). A case report by Puspita documented a patient with IE caused by *Streptococcus viridans* originating from cavities and diseased teeth.[1] In addition, other case reports have found cases of endocarditis caused by manifestations of periodontitis. This forms the background for conducting a scientific study to investigate the importance of maintaining oral hygiene, especially in RHD patients, to reduce the risk of IE.

Despite the established role of oral pathogens, particularly Streptococcus viridans, in the pathogenesis of infective endocarditis (IE), a significant gap remains in the literature regarding the specific risk posed by poor oral hygiene among patients with rheumatic heart disease (RHD). While several studies have examined the general relationship between oral health and cardiovascular infections, few have focused on RHD patients as a distinct high-risk group, particularly in low- and middle-income countries where RHD remains prevalent. This systematic review seeks to address this gap by critically evaluating and synthesizing existing evidence on the association between poor oral hygiene and the risk of IE in individuals with RHD. The novelty of this study lies in its targeted focus on the RHD population, emphasizing the overlooked importance of oral hygiene as a modifiable risk factor for preventing life-threatening infections. Specifically, this review aims to assess the impact of oral health conditions such as periodontitis, caries, and gingivitis on the incidence of IE in RHD patients, identify the dominant oral pathogens involved, and provide evidence-based recommendations for preventive dental care strategies tailored to this vulnerable population. This review aims to answer the question of whether poor oral hygiene increases the risk of infective endocarditis among patients with RHD.

METHODS

A systematic review was conducted following the PRISMA 2020 guidelines to ensure transparency and reproducibility. Literature searches were performed across three major electronic databases: PubMed, Scopus, and Google Scholar. The search strategy utilized Boolean operators, combining with the following keywords: "infective endocarditis," "rheumatic heart disease," "oral hygiene," "periodontitis," and "dental caries."

This methodology comprised four stages: identification, screening, feasibility, and results received. Literature for this systematic review was obtained by searching online electronic databases, including Google Scholar, PubMed, and Scopus. The PECO (Population, Exposure, Comparison, and Outcomes) framework was used to formulate eligibility criteria (Table 1). The inclusion criteria used were scientific journals published from January 2014 to December 2024 (the last 10 years), written English, and identified using the Boolean operator OR/AND with the main keywords: ('Endocarditis' OR 'Rheumatic Heart Disease') AND 'Oral Hygiene' OR ('Endocarditis' AND 'Periodontitis') OR ('Dental Caries' AND 'Rheumatic Heart Disease' AND 'Endocarditis'). In addition, the articles had to be accessible in full text. Meanwhile, the exclusion criteria used were studies conducted using animal models, studies involving cardiac surgery, review articles, letters to the editor, duplicate publications, case-control study articles, and case reports.

The methodological quality of the included studies was assessed independently by two reviewers, FA (Farah Amiria, Dental Medicine and Biomedical Sciences) and NSAG (Nur Signa Aini Gumilas, Biotechnology and Medical Sciences), using the Newcastle-Ottawa Scale (NOS) for observational studies.[16] Disagreements in scoring were resolved through discussion, and if necessary, a third reviewer was consulted to reach consensus. Each study was rated as having low, moderate, or high risk of bias based on domains of selection, comparability, and outcome assessment.

Data extraction was performed independently by two authors using a standardized data extraction form. Extracted data included study characteristics (author, year, country, design, sample size), population characteristics, oral health variables, and outcomes related to infective endocarditis. Inconsistencies between reviewers were discussed and resolved through consensus, following the recommendations of the Cochrane Handbook.[17]

Table 1. **PECO framework (*population, exposure, comparison, outcomes*)**

Item	Information
Population	Rheumatic Heart Disease
Exposure	Poor Oral Hygiene (OH)
Comparison	Good Oral Hygiene (OH)
Outcomes	Endocarditis

The results of the database search are presented using the PRISMA flow diagram (Diagram 1). All duplicate articles were removed. Next, the remaining articles were screened based on the inclusion criteria and exclusion criteria by evaluating the article type, title, abstract, and full content. Articles that did not meet the inclusion criteria or met any of the exclusion criteria were excluded from the review.

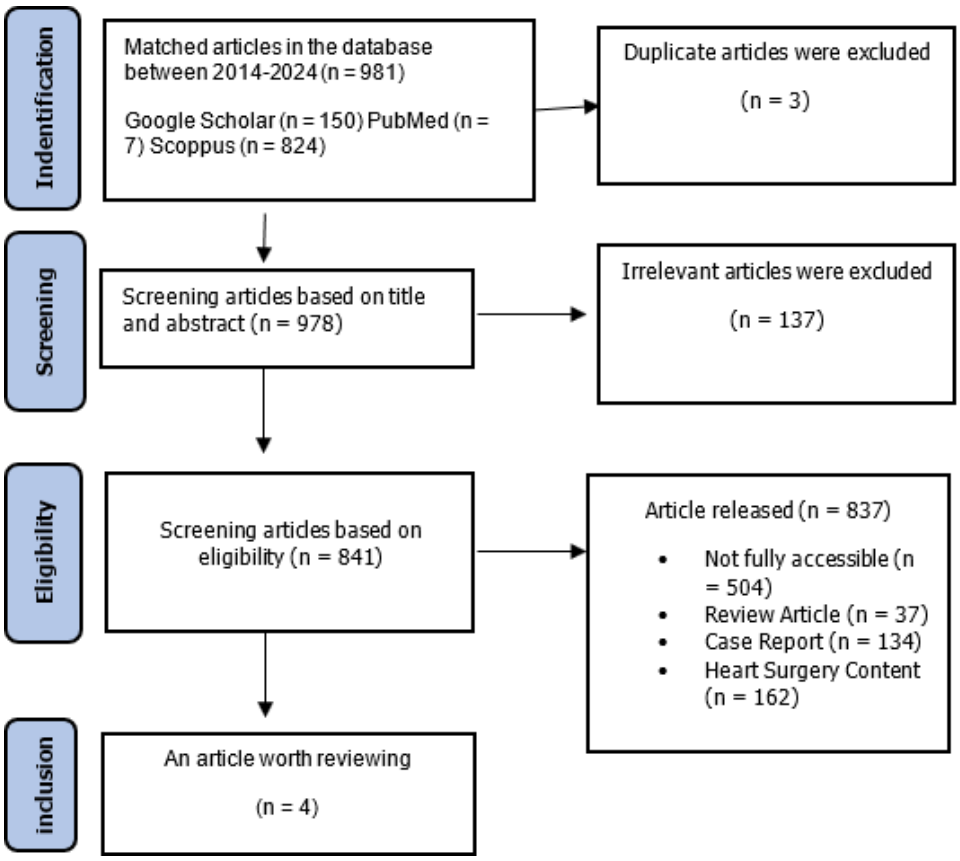

Figure 1. PRISMA flow chart

RESULTS

Based on the systematic literature search across three databases, 981 articles were initially identified. After duplicate removal, 978 articles underwent screening by article type, title, abstract, and content relevance to the topic, resulting in 841 articles. These articles were then assessed for eligibility based on full-text accessibility and conformity with inclusion criteria, yielding 4 eligible articles (Table 2).

Tabel 2. Literature search results

No	Research Title	Author and Year	Method	Sample	Observed Research Variables	Result
1	Mendelian Randomizatio n Reveals No Causal Association Between Periodontitis and Infective Endocarditis.	Liu Rui-lin et al., 2024	Mendelian Randomizati on (MR)	Acute Periodontiti s: n = 6; Chronic Periodontiti s: n = 12; Aggressive Periodontiti s: n = 8	Causal relationship between periodontitis and infective endocarditis	There is no direct causal relationship between periodontitis and infective endocarditis.
2	Oral Health Experiences of Turkish Children with Acute Rheumatic Fever or	Onsuren AS et al., 2022	Cross-sectional	Control Group: n = 43 (Male: 22, Female: 21) Treatment	Correlation between RHD patients and the oral hygiene index, specifically dental caries	There is a positive correlation between dental caries and acute rheumatic fever. The relationship

	Rheumatic Heart Disease.			group: n = 43 (Male: 24, Female: 21)		between sugar intake and acute rheumatic fever is significant, particularly due to the involvement of *Streptococcus mutans,* the causative agent of dental caries. The research also shows that penicillin use in RHD patients may accelerate caries in primary teeth.
3	The Assessment of Antimicrobial Resistance in Gram-Negative and Gram-Positive Infective Endocarditis: A Multicentric Retrospective Analysis.	Budea CM et al., 2023	Retrospective Cohort Study	IE mono-infected gram-negative group: n = 29; IE mono-Infected, gram-positive group: n = 142	Relationship between the bacteria causing IE and the severity of disease, considering comorbidities and predisposing factors	IE caused by gram-negative bacteria resulted in a higher mortality rate than gram-positive IE in this study. However, these findings must be tempered by the small sample size of gram-negative IE cases as well as potential bias factors, as patients with gram-negative infections had a higher proportion of comorbidities and predisposing factors.
4	The Clinical Features and Prognosis of Infective Endocarditis in the Elderly from 2007 to 2016 in a Tertiary Hospital in China.	Wu Zhenzhu et al., 2019	Retrospective Cohort Study	Patient with IE: n = 405; < 50 years: n = 205; 50-64 years: n = 141; > 65 years: n = 59. Patients aged > 65 were divided into survival and mortality groups.	Course and prognosis of IE in patients of different age ranges, based on bacterial causes	IE patients of older age show more comorbidities, poorer oral hygiene, and a more severe prognosis than younger patients. *Streptococcus* bacteria are the most frequently found microorganisms in this group.

Tabel 3. Quality assessment using the Newcastle–Ottawa Scale (NOS)

No.	Author (Year)	Selection (0–4)	Comparability (0–2)	Exposure (0–3)	Total Score (0–9)	Risk of Bias
1	Liu Rui-lin et al. (2024)	2	0	1	3	High
2	Onsuren AS et al. (2022)	3	1	2	6	Moderate
3	Budea CM et al. (2023)	4	1	2	7	Low
4	Wu Zhenzhu et al. (2019)	4	2	3	9	Low

The methodological quality of the included studies was assessed using the Newcastle–Ottawa Scale (NOS), as shown in Table 3. Each study was evaluated based on three domains: Selection (maximum 4 stars), Comparability (maximum 2 stars), and Outcome/Exposure (maximum 3 stars). The total score determined the overall risk of bias, which was classified as Low (7–9), Moderate (4–6), or High (0–3).

DISCUSSION

The results of the systematic review reveal variations in the data, as summarized in Table 2. While several studies reported a significant association between poor oral hygiene and an increased risk of infective endocarditis (IE) among patients with rheumatic heart disease (RHD), other studies found no clear relationship. This discrepancy may reflect differences in study design, population characteristics, diagnostic criteria for oral conditions, and methods of detecting bacteremia.

Notably, studies conducted in low- and middle-income countries tended to report stronger associations, possibly due to the higher baseline prevalence of both poor oral hygiene and RHD. In contrast, studies from high-income settings often involved patients receiving regular dental care and prophylaxis, which may have mitigated the observed risk.[12]

Across studies, Streptococcus species consistently emerged as the predominant bacteria implicated in the development of IE. Although part of the normal oral flora, these organisms can become pathogenic in environments characterized by poor oral hygiene and low pH (acidic conditions), leading to dental caries. Periodontal disease was also frequently identified as a potential source of bacteremia, reinforcing the role of oral infections as important contributors to IE pathogenesis in susceptible individuals.[12]

Initial supragingival bacterial colonization involves *Streptococcus sanguinis*, *Streptococcus oralis*, *Streptococcus mutans*, *Actinomyces naeslundii*, and *Actinomyces odontolyticus*. This is followed by secondary bacterial colonization, such as by *Fusobacterium nucleatum*.[5,6] The presence of bacteria originating from poor oral hygiene (OH) can result in oral infections, which may enter the bloodstream and contribute to the development of systemic disease.[18]

Poor OH condition is often characterized by oral infections such as caries and periodontal disease, including gingivitis, periodontitis, and abscesses. Periodontitis may influence the host's susceptibility to systemic disease in three ways: (1) through shared risk factors, (2) by subgingival biofilm acting as a reservoir for gram-negative bacteria, and (3) by the periodontium serving as a reservoir for inflammatory mediators.[19] This supports the notion that periodontal disease can be an individual risk factor for cardiovascular disease, considering the wide range of gram-negative species involved, the levels of pro-inflammatory cytokines detected, the immune infiltrate and severe inflammation present, the

high association with peripheral fibrinogen, and the white blood cell count. Periodontal disease can trigger pathways that lead to heart disease through the indirect effects of oral bacteria (Figure 1).

Cardiovascular disease is primarily caused by bacteremia originating from the oral cavity as a source of infection. Such infections can damage heart valves and result in subacute bacterial IE. This was confirmed by a study conducted in China in 2024, which found no direct causal relationship between periodontitis and IE. The study showed no statistically significant differences in IE cases among patients with acute, chronic, and aggressive periodontitis. The development of IE appears to result from a combination of infectious factors and individual endocardial susceptibility. While the presence of bacteremia is a major factor, it alone is not sufficient to cause IE.[20] Therefore, the study of predisposing factors for IE is essential.

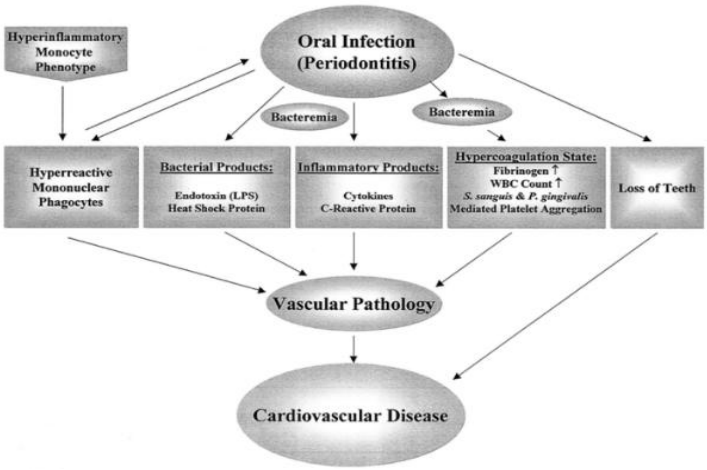

Figure 2. Mechanism of the link between oral infections (periodontitis) and cardiovascular disease.[8]

The main predisposing factor for infective endocarditis (IE) in developing countries is rheumatic heart disease (RHD). RHD itself is a heart valve disease resulting from damage to these valves. If complications arise and bacteremia leads to infection, RHD can be a predisposing factor for IE. According to research by Onsuren et al (2022), a positive correlation exists between RHD and dental caries. This study links the oral hygiene (OH) index with Acute Rheumatic Fever (ARF) and RHD. Oral hygiene data were collected through intraoral examination using the DMF-T (Decay, Missing, Filling, - Teeth) score. In addition to the DMFT score, the gingival index and plaque index were also examined to assess patients' OH. The DMFT results in this study indicate that the null hypothesis cannot be accepted; the dmft index in the primary teeth of the research group showed significantly higher results compared to the control group ($p<0.001$).

RHD patients with dental caries had more teeth extracted than filled teeth. This highlights the importance of controlling infection risk scheduled for extraction, which can be anticipated through the use of prophylactic antibiotics.[21] RHD patients with bacterial infections in the oral cavity are at increased risk of developing IE through complex pathophysiological mechanisms. Therefore, RHD patients should undergo regular dental health check-ups and maintain proper OH to minimize the risk of bacteremia originating from the oral cavity.

RHD is a complication of a throat infection caused by *Streptococcus* bacteria. The immune response to this infection can lead inflammation of the heart, especially the heart valves, resulting in damage and structural changes. Chronic inflammation and scar tissue formation on heart valves can create areas highly susceptible to infection. Damaged or abnormal valves can disrupt blood flow and increase turbulence, creating favorable conditions for microorganisms growth.

Microorganisms, especially bacteria, can adhere to the surface of damaged valves and form structures called vegetations. This vegetation is composed of platelets, fibrin, and microbes.[18]

This process is often initiated by trauma or damage to the valve surface. The most common microorganisms involved in IE are bacteria, although fungal infections may also occur. When bacteria infect pre-formed vegetation, they can cause further inflammation, worsening valve damage and increasing the risk of embolism. An immune reaction to the infection can also cause further damage to heart tissue. The resulting inflammation can lead to systemic symptoms such as fever, fatigue, and joint pain, which are commonly observed in patients with IE. Complications of IE can include further damage to the valves, heart failure, and embolism to other organs, which can lead to tissue damage or secondary infections.[6]

The inflammatory reaction caused by dental disease increases plaque formation, which stimulates the thickening of blood vessel walls. Research shows that people with dental disease have twice the risk of developing coronary heart disease. Bacterial endocarditis found in dental plaque is one of the factors causing endocarditis. Bacteria from tooth cavities (*Streptococcus* bacteria) and damaged gums can enter the bloodstream through bleeding gums. These bacteria easily attack heart valves and weakened heart muscles.[22]

In addition to the inflammatory mechanism, *Streptococcus* infection can trigger a strong immune reaction. This immune reaction forms antibodies that can attack heart tissue, resulting in heart inflammation. The ensuing heart inflammation leads to damage to endothelial cells and the formation of scar tissue. This damages the valve, which can change its structure and create a vulnerable area. When bacteria attach to this area, biofilm formation occurs, which then develops into vegetation. This process further triggers an immune reaction, continuing the inflammatory process and worsening tissue damage. The final complication has the potential to cause heart failure and embolism (Diagram 2). It is important for dentists to exercise caution when performing dental procedures on patients with a history of RHD; it is best to administer prophylactic antibiotics beforehand.

High-risk patients require prophylactic antibiotics before the procedure. The prophylactic antibiotic choices include amoxicillin 2 g (30-60 minutes before the procedure) or clindamycin 600 mg (30-60 minutes before the procedure). The treatment of IE requires selecting the appropriate antibiotic, depending on the type of infecting pathogen and the patient's clinical condition. Infections caused by *Streptococcus* bacteria can be treated with penicillin G antibiotics. The recommended dose is 12-18 million units/day IV for 4-6 weeks. Ceftriaxone-class antibiotics may be used as an alternative, particularly for infections caused by *Streptococcus viridans*, at a dose of 2 g every 24 hours for 4-6 weeks.

This review encountered several limitations. Access to full-text articles was restricted in some cases, limiting the depth of data extraction and quality assessment. Additionally, only a few studies met the strict inclusion criteria, impacting the generalizability of the findings. Differences in NOS scores also indicated methodological heterogeneity across studies.

Future studies should explore larger populations, employ uniform diagnostic criteria for oral hygiene (OH) and infective endocarditis (IE), and consider longitudinal designs that track patients over time. The role of modifiable behavioral factors and adherence to preventive guidelines should also be evaluated. In conclusion, the available evidence highlights the clinical importance of maintaining good OH, especially in high-risk populations such as RHD patients. Integrating dental care into the routine management of RHD could be a valuable preventive strategy against infective endocarditis.

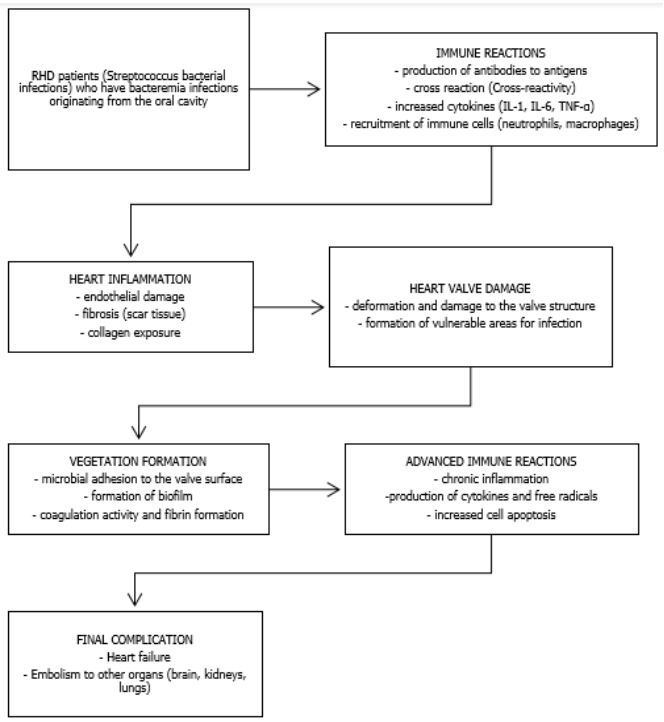

Figure 3. Pathophysiology of infective endocarditis (IE) in RHD patients with poor oral hygiene (OH)[2]

The limitations of this study include challenges in accessing full-text articles and a limited number of studies that met the inclusion criteria, which could affect the generalizability of the findings. Moreover, the small number of included studies increases the potential for publication bias, as studies reporting significant associations are more likely to be published and accessible than those with null findings.

Additionally, the heterogeneity in study designs, populations, and methodological quality introduces variability that may influence the observed associations and limit the ability to draw firm conclusions. This heterogeneity also raises the risk of selection and measurement biases within individual studies, which may, in turn, affect the overall synthesis.

A further limitation of this review lies in the absence of a meta-analysis, which precluded quantitative assessment of heterogeneity (e.g., I^2 statistics) and formal evaluation of publication bias (e.g., funnel plot or Egger's test). Consequently, the findings should be interpreted with caution, acknowledging these potential sources of bias.

CONCLUSION

This review highlights that poor oral hygiene significantly increases the risk of infective endocarditis (IE) among patients with rheumatic heart disease (RHD), mainly through bacteremia and bacterial colonization of damaged heart valves. These findings support the integration of oral health into cardiovascular disease prevention protocols, emphasizing regular dental care and the use of prophylactic antibiotics before invasive dental procedures in high-risk patients. Interdisciplinary collaboration between cardiologists and dental professionals is essential to reduce IE incidence through preventive strategies. Future research should assess the effectiveness of specific oral hygiene interventions and prophylaxis protocols using standardized methods and larger study populations.

Author Contributions: For research articles with several authors, a short paragraph specifying their individual contributions must be provided. The following statements should be used: "Conceptualization, F.A. and N.S.A.G.; methodology, F.A.; validation, N.S.A.G.; writing original draft preparation, F.A.; writing review and editing, F.A.; visualization, F.A.; supervision, N.S.A.G.; all authors have read and agreed to the published version of the manuscript."
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