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The oral health status of patients with peripheral vascular disorders: A systematic review

Sayed Abdulmotaleb Almoosawy[1], Mhairi McGowan[2], Karolin Hijazi[2], Rona Patey[1], Paul Bachoo[3] and George Cherukara[2]

Abstract

Objectives: Periodontal disease and tooth loss were found to be associated with several peripheral vascular disorders. Nonetheless, an evaluation of the literature on the broader domains of oral health in individuals with peripheral vascular disorders is lacking. This systematic review aims to collate the current evidence on the oral health status of individuals with peripheral vascular disorders.

Methods: Five electronic databases were searched for studies assessing oral health parameters in individuals with peripheral vascular disorders. Outcome measures considered were periodontal health, dentition status, caries indices, oral prostheses, oral pathologies and oral hygiene behaviours. The Newcastle-Ottawa scale was used to appraise the quality of the studies.

Results: From 3025 records identified, 24 studies involving 1232 participants with peripheral vascular disorders were included in this review. In nine studies, periodontitis was significantly more prevalent in peripheral vascular disorders compared to non-peripheral vascular disorders participants. A further six studies reported individuals with peripheral vascular disorders also had significantly fewer teeth and increased rates of edentulism. Only one study reported a higher incidence of dental caries in peripheral vascular disorders participants. Other aspects of oral health such as oral prosthesis, oral pathology and oral hygiene behaviours were seldom assessed.

Conclusions: The scarcity of studies reporting on broader domains limited our ability to arrive at a conclusion regarding the oral health status of individuals with peripheral vascular disorders. Future studies ought to assess these domains in individuals with peripheral vascular disorders and controls to gain a more complete understanding of oral health and its potential association with peripheral vascular disorders.

Keywords

Vascular medicine, peripheral vascular disease, oral health

Introduction

Emerging evidence illustrates an association between certain oral health conditions, such as periodontal disease and tooth loss, and several peripheral vascular disorders (PVD).[1–6] The International Statistical Classification of Diseases and Related Health Problems (ICD) defines PVD as an umbrella term for any disorder affecting blood flow in arteries or veins outside the heart.[7] A recent meta-analysis[8] reported a significantly higher risk of periodontitis and tooth loss amongst individuals with peripheral arterial disease (PAD), a type of PVD, than healthy controls. Their

[1]School of Medicine, Medical Sciences and Nutrition, University of Aberdeen, Aberdeen, Scotland, United Kingdom
[2]Institute of Dentistry, University of Aberdeen, Aberdeen, Scotland, United Kingdom
[3]Department of Vascular Surgery, Aberdeen Royal Infirmary, Aberdeen, Scotland, United Kingdom

Corresponding author:
George Cherukara, Institute of Dentistry, University of Aberdeen, Aberdeen, Scotland AB25 2ZR, United Kingdom.
Email: gcherukara@abdn.ac.uk

review only considered periodontitis and no other essential aspects of oral health as per the World Health Organisation's (WHO) manual for standardised oral health assessment. These include dentition status, dental erosions, oral mucosal lesions, dental caries, dentition and prosthesis status and oral hygiene behaviours.[9] More specifically, dental caries and poor oral hygiene had been investigated as markers of early initiation of atherosclerosis and were found to be associated with increased carotid intima–media wall thickness.[10,11] Poor oral hygiene also increases the risk of periodontitis[12] and can lead to increased abundance of potentially pathogenic bacteria colonising the teeth.[13] These bacteria may exacerbate atherogenesis via the oral infection–inflammation pathway.[2]

Several measures have been taken by vascular surgeons regarding their patients' dental health, including dental antibiotic prophylaxis to prevent vascular graft infections following dental sepsis.[14,15] However, the oral health status of individuals with PVD remains incompletely assessed clinically. This is due to a lack of consensus regarding the need for oral health assessment in this group and whether this would warrant an improvement in outcomes of PVD. The aim of this study was to review the literature relating to the oral health status of individuals diagnosed with PVD. Further, where studies are available with controls (individuals without PVD), a comparison of the oral health status between these two groups was undertaken.

Methods

Search strategy

We used the Preferred Reporting Items for Systematic Review and Meta-analysis guidelines (see supplemental material for the checklist) for this systematic review. The Population Intervention/Exposure Comparator Outcome criteria were used to form the review question – What are the oral health findings (O) in individuals with PVD (P) whom have undergone oral health assessment (I) compared to individuals without PVD (C). Electronic database and hand searches for articles in April 2020 were conducted in the following databases: MEDLINE, SCOPUS, EMBASE, Web of Science and Cochrane Central Register of Controlled Trials. The search strategy was formed using the Medical Subject Headings and relevant free-text terms and was applied to each database (Supplementary Table 1).

Inclusion and exclusion criteria

Inclusion criteria:

(i) Adults aged $\geq$18 years
(ii) Individuals with any vascular disease of arteries and veins outside the heart as grouped under the PVD diagnosis-related groups by the ICD[16]
(iii) Assessed any of the following oral health measures: dentition status, remaining/missing teeth, prevalence of dental disease including periodontal indices, dental caries indices, oral infections, oral pathology, presence/absence of oral prosthesis and oral hygiene behaviours
(iv) Published in English

Exclusion criteria:

(i) Systematic or literature reviews
(ii) Case reports
(iii) Studies with duplicate/overlapping cohorts.

Data extraction and quality assessment

Study characteristics and data on participants' oral health were extracted from each included study and compiled on data extraction tables (Table 1 and Supplementary Tables 2 and 3). Titles and abstracts of all studies were independently screened by two reviewers (SAA and MM). Full texts of the selected studies were critically reviewed based on the inclusion and exclusion criteria. Although there were no disagreements, an arbitrator (GC) was available for mediation. For quality assessment, both reviewers independently used the Newcastle–Ottawa Scale (NOS) for case–control studies[17] and a modified form for cross-sectional studies (Supplementary Table 4).

Results

Search results

The search identified 3025 studies. RefWorks (ProQuest Refworks, 2020) was used to process the search results and to de-duplicate 80 studies. After application of inclusion and exclusion criteria, 58 studies were selected for full text screening, following which 24 studies comprising 1232 PVD participants were included in this systematic review (Figure 1).

Characteristics of studies

The characteristics of studies included in this systematic review are summarised in Table 1. These studies were published between 1994 and 2018 and were from 14 countries. Twenty-three studies were on arterial disorders, and one was on a venous disorder. Thirteen studies compared the oral health status between PVD participants and healthy controls,[3,18–29] whilst three compared the oral health of PVD participants to controls with cardiac diseases.[30–32] Six studies[14,33–37] did

Table 1. Characteristics of included studies.

Author(s)	Country	Study design	*N*, Mean age (±SD), M/F		Examiner calibration Statistical power calculation	Clinical parameters of PVD/PVD diagnosis
			PVD group	Non-PVD group		
Stansby et al.[14]	England	CSS	70, 68, 32/18	N/A	N/A	ABI $\leq$ 0.90/PAD or AA
Hamasha et al.[36]	USA	CSS	19, 80.7, unspecified	N/A	N/A	Medical record of atherosclerotic vascular disease
Häyrinen-Immonen et al.[33]	Finland	Prospective clinical study	50, 65, 33/17	N/A	N/A	AAA
Bloemenkamp et al.[26]	Holland	CSS	212, 48.2 ± 7.0, 0/212	475, 45.5 ± 3.1, 0/475	N/A	Clinical symptoms (intermittent claudication, non-healing ulcers, gangrene); angiographic findings indicating $\geq$50% of stenosis in peripheral arteries/PAD
Cairo et al.[39]	Italy	Controlled clinical and laboratory trial	19, 71.37 ± 6.14, 14/5	21, 73.33 ± 6.11, 15/6	N/A	Carotid stenosis
Kurihara et al.[23]	Japan	CCS	32, 73, 27/5	Unspecified	N/A	Angiographic, ultrasonic and CT evaluation of AAA
Iwai et al.[24]	Japan	CCS	14, 60, 14/0	7, unspecified, 5/2	N/A	Shionoya's criteria, angiographic findings, Allen's test/Buerger's disease
Chen et al.[22]	Japan	CCS	19, 56.6 ± 11, 19/0	19, 56.6 ± 11, 19/0	N/A	Shionoya's criteria, angiographic findings, Allen's test/Buerger's disease
Chen et al.[18]	Japan	CCS	25, 67.6 ± 10, 21/4	32, 63.10 ± 10, 28/4	N/A	Clinical symptoms, angiographic findings and ABI/PAD
Friedlander et al.[28]	USA	CCS	36, 64.4 ± 10.0, 97.2%/2.8%	36, 64.9 ± 10. 10, 97.2%/2.8%	N/A	Carotid stenosis confirmed by Doppler ultrasonography
Toyofuku et al.[25]	Japan	CCS	53, 69.0 ± 9.1, 49/4	21, 70.6 ± 8.9, 20/1	N/A	Fontaine classification, angiographic, ultrasonic and CT imaging/arteriosclerosis obliterans
Zaremba et al.[38]	Poland	CCS	20, 67, 13/7	unspecified	N/A	Internal carotid artery stenosis
Sánchez et al.[3]	Spain	CCS	97, 60.63 ± 15.07, 43/54	100, 61.86 ± 8.49, 54/46	N/A	Doppler ultrasonography and CT angiography/VTED
Soto-Barreras et al.[27]	Mexico	CCS	30, 63.23 ± 9.06, 27%/73%	30, 61.86 ± 8.49, 30%/70%	Yes/N/A	ABI $\leq$ 0.90. <mild-to-moderate: 0.40 to 0.90 Severe: <0.4/PAD
Figuero et al.[34]	Sweden	CSS	42, 68.95 ± 8.65, 31/11	N/A	N/A	Carotid stenosis, PAD and AAA
Fernandes et al.[35]	Brazil	CSS	13, 68.5 ± 10.1, 6/7	N/A	Yes/N/A	AA and carotid stenosis
Suzuki et al.[31]	Japan	CCS	12, 70.6 ± 3.5, 9/3	25, 71.4 ± 2.1, 60%/40%	N/A	AAA
Ding et al.[21]	China	CCS	89, 57.8 ± 7.6, 79/10		N/A	

(continued)

Table 1. Continued.

Author(s)	Country	Study design	*N*, Mean age (±SD), M/F		Examiner calibration Statistical power calculation	Clinical parameters of PVD/PVD diagnosis
			PVD group	Non-PVD group		
				59, 56.8 ± 6.3, 44/15		AA diagnosed by CT showing aortic diameter >50% than normal
Suzuki et al.[32]	Japan	CCS	25, 71.4 ± 2.1, 60%/40%	142, 68.8 ± 0.4, 78.2%/21.8%	N/A	AAA
Igari et al.[37]	Japan	CSS	58, 48, 55/3	N/A	N/A	Buerger's disease diagnosed by Shionoya's criteria
Çalapkorur et al.[29]	Turkey	CSS	40, 60.45 ± 9.94, 32/8	20, 60.4 ± 9, 18/2	N/A/yes	ABI ≤ 0.90 and Rose questionnaire/PAD
Aoyama et al.[30]	Japan	CSS	34, 65.6 ± 11.8, 23/11	956, 64.4 ± 13, 693/263	N/A	Clinical symptoms, ABI and angiographic findings/PAD
Nicolaiciuc et al.[19]	Romania	CCS	*N* = 35 IMT > 1 mm: 18, atheroma plaque: 17, IMT > 1 mm: 62.9 (±10.2), atheroma plaque: 62.5(±10/8), IMT > 1 mm: 9/9, atheroma plaque: 12/5	15, 40,8 (±10,9), 4/11	N/A	Evaluation done with USS to measure IMT and record presence of atherosclerotic plaques/carotid atherosclerosis
Ding et al.[20]	China	CCS	169, 56.2, 30/39	156, 54.8 ± 5.0, 129/27	N/A	AA diagnosed by CT when aortic diameter >50% normal diameter

AA: aortic aneurysm; AAA: abdominal aortic aneurysm; ABI: ankle brachial index; CCS: case–control study; CSS: cross-sectional study; CT: computed tomography; F: Female; IMT: intima-media thickness; M: male; PAD: peripheral artery disease; PVD: peripheral vascular disease; SD: standard deviation; USS: ultrasound scan; VTED: venous thromboembolic disease.

not have controls. Another study included controls with cardiovascular disease; however, they did not measure oral health in the control group.[38] One study recruited edentulous PVD participants as controls; however, no oral health assessment was conducted in this group, therefore, a comparison was not done between cases and controls.[39]

Examiner calibration and statistical power calculation

Of the 24 studies, three studies reported examiner calibration through intraclass correlation coefficient,[27] re-evaluation of random referred patients[29] and another through previously calibrated examiners using Kappa values (0.80–0.97).[35] Only one study provided details of statistical power calculation to determine sample size.[29]

Case definition of PVD

The case definition of PVD varied amongst the studies (see Table 1). Seven studies reported a PVD diagnosis of case groups but with no description of the parameters used to diagnose PVD.[31–35,38,39] One study used previous medical records of vascular disease.[36] Four studies used clinical findings of PVD as diagnostic parameters such as ankle-brachial index, clinical symptoms and Rose questionnaire.[14,26,27,29] Stansby et al.[14] reported the parameters used for diagnosing PAD but had only reported a diagnosis of aortic aneurysm (AA). Six studies used imaging parameters only such as angiography, computed tomography and Doppler ultrasonography.[3,19–21,23,28] Six studies used a combination of clinical findings and imaging parameters for diagnosis of PVD.[18,22,24,25,30,37]

Oral health measures

Periodontal health

Periodontal health was assessed in 22 studies with results displayed in Supplementary Table 2. The case definition of periodontitis varied across each study (see Supplementary Table 2). Prevalence of periodontitis and moderate to severe periodontitis was reported to be significantly higher in participants with arteriosclerosis obliterans,[25] Buerger's disease,[22] PAD,[18,26,27] carotid atherosclerosis,[19] AA[20,21] and venous thromboembolic disease (VTED)[3] compared to non-PVD participants. Two studies found no differences between groups.[28,29] In studies with no controls, a high percentage of the PVD participants had periodontitis[23,24,37] except for one study.[33] Gingivitis was more prevalent in non-PVD than in PVD participants in four studies.[20,21,26,29]

Probing pocket depth (PPD) is a measurement of the distance from the gingival margin to the pocket base surrounding a tooth. This measurement is one of a range of clinical criteria used to diagnose and assess severity of periodontal diseases.[40] Aoyama et al.[30] and Çalapkorur et al.[29] found no difference between PVD and non-PVD participants in the mean PPD. Çalapkorur et al.[29] reported a significantly higher number of sites with PPD over 5 mm amongst PVD participants. Likewise, a significantly higher number of sites with PPD over 4 mm were seen in AA,[20,21,31,32] Buerger's disease,[22] carotid atherosclerosis[19] and PAD participants[18,27] compared to non-PVD participants. It is important to note that Çalapkorur et al.[29] defined periodontitis as having at least five teeth with at least one site of PPD equal to or greater than 5 mm. This differed from the other studies which defined periodontitis as the presence of more than one site with PPD equal to or greater than 4 mm in each quadrant.

Clinical attachment loss (CAL) indicates the extent of periodontal tissue support loss around a tooth. A significantly higher percentage of sites with CAL greater than 4 mm was found amongst AA,[20,21] Buerger's disease,[22] PAD[18,27] and VTED[3] participants compared to non-PVD participants. There was no significant difference in CAL greater than 4 mm between PVD and non-PVD participants in two studies.[29,30]

Zaremba et al.[38] found 4 of 20 participants demonstrating presence of periodontal bacteria in their atherosclerotic plaques after carotid endarterectomy. These participants had significantly higher bleeding indices and PPD greater 4 mm compared to participants with no periodontal bacteria in the atherosclerotic plaque. Mean CAL was not significantly higher in the group with periodontal bacteria present in atherosclerotic plaques than those without.

Dentition status

Thirteen studies assessed the dentition status of PVD participants with results displayed in Supplementary Table 2.[3,14,18,20,23,28,30–36] Nine studies compared remaining or missing teeth in PVD to that of non-PVD participants.[3,18,20,28,30–32,34,36] All but three[28,31,34] reported significantly less retained teeth,[3,18,30,32] more missing teeth[20,30,36] or higher edentulism rates[30,36] in PVD compared to non-PVD participants. Three studies reported lower percentages of PVD participants who were edentulous.[14,23,33] Fernandes et al.[35] found that 96.1% of teeth were missing amongst 13 PVD participants.

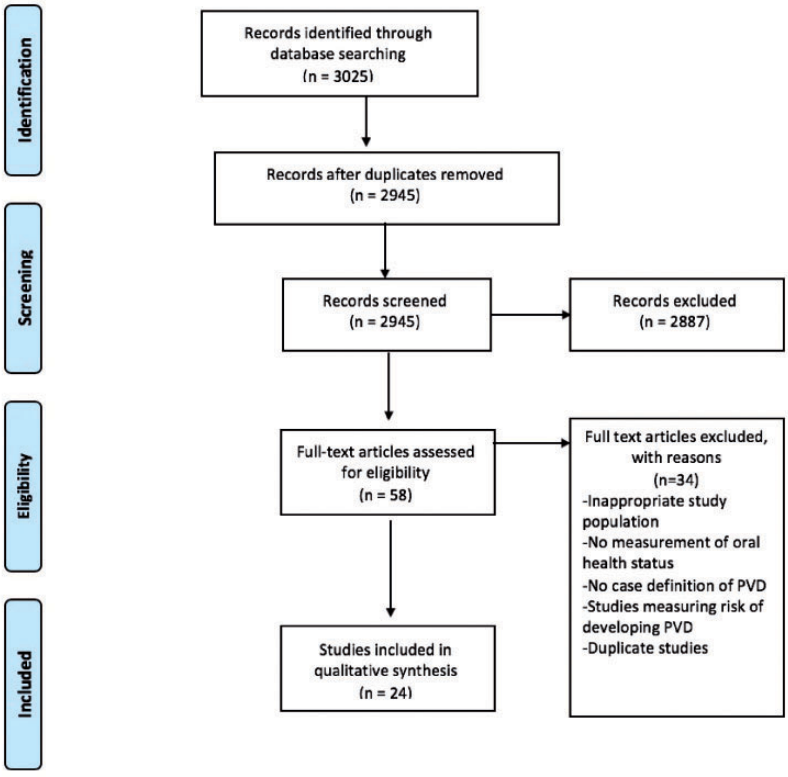

Figure 1. Preferred reporting items for systematic reviews and meta-analyses flow diagram of literature search and paper selection process.

Dental caries

Five studies investigated caries in PVD participants with findings displayed in Supplementary Table 3.[27–29,33,35] The DMFT is the sum of the number of **D**ecayed, **M**issing and **F**illed **T**eeth due to dental caries in the permanent teeth. It is a widely used index to measure dental caries and dental treatment needs amongst populations.[9] Two studies reported the mean DMFT in PVD participants but had no controls.[33,35] Calapkorur et al.[29] reported no significant difference in the DMFT of PVD compared to non-PVD participants. Likewise, Friedlander et al.[28] found no significant difference in the mean number of carious retained roots or coronal/pulpal caries between PVD and non-PVD participants. Only one study reported a significantly higher DMFT index in PVD than non-PVD participants.[27]

Other oral diseases

Two studies commented on oral diseases other than caries and periodontal disease (see Supplementary Table 3).[28,33] Friedlander et al.[28] found no difference in the mean number of teeth with periapical lesions between PVD versus non-PVD participants. Immonen et al.[33] reported 80% of the PVD participants had an oral infection, with only 11% of them having 'good oral health'. Candida infection was present in 17% of dentate and 47% of edentulous PVD participants. Fifty-four percent of dentate participants had intraosseous foci compared to 20% of edentulous participants. Periapical lesions and intraosseous foci are areas of localised chronic infection of dental origin that may influence chronic systemic diseases.[41]

Dental prosthesis

Three studies assessed the presence of dental prosthesis in PVD participants (see Supplementary Table 3). Stansby et al.[14] reported 46% PVD participants had partial dentures. Another study reported that amongst a total of 56 dentures in 37 PVD participants, 45% of dentures were poor and had to be replaced.[33] A third study reported the mean number of implants but had no controls.[34]

Oral hygiene behaviours

Only one study investigated oral hygiene behaviours. They used a self-reported assessment of oral hygiene behaviours amongst AA participants.[20] Compared to the non-AA group, more AA participants had inaccurate brushing methods, less brushing time and

frequency, no flossing and less routine dental examinations (Supplementary Table 3).

Discussion

This review included broader oral health domains in the WHO's manual to assess oral health in individuals. Participants with PVD were found to have compromised oral health across various measures such as poorer periodontal health, more missing teeth and a higher prevalence of edentulism in comparison to non-PVD participants. However, the findings on dental caries and presence of periapical lesions in PVD and non-PVD participants were conflicting. Although not reported in the studies, reasons for this may include differences in dental health-seeking behaviour in PVD participants, and in the diagnostic protocols in different countries. Other aspects such as oral prosthesis, oral hygiene behaviours and oral pathology were seldom assessed.

This review has also considered other subtypes of PVD such as PAD, carotid atherosclerosis, AA, Buerger's disease and VTED. The assessment of oral health amongst individuals with a venous disorder was performed in only one of the included studies.[3] Association studies demonstrated a possible relationship with valvular incompetence in individuals with varicose veins,[42] the latter being a known risk factor for VTED.[43] This could be mediated by the role of certain bacteria in periodontal disease, which were found to be risk factors for vascular endothelial damage and pro-coagulation.[44,45] Despite Sánchez and colleagues'[3] significant oral health findings in their cohort with a venous disorder, further studies are required to confirm the validity of these findings and to compare the oral health status between individuals with venous and arterial disorders. Two studies in Yang and colleagues'[8] meta-analysis were excluded from our review, as they assessed the risk of developing PVD in participants with prior periodontitis.[1,46]

Tooth loss in PVD participants

Several studies reported a significantly higher prevalence of tooth loss amongst PVD as opposed to non-PVD participants.[3,18,20,30,32,36] The putative mechanisms underpinning this finding could be varied. Firstly, tooth loss was shown to have a possible association with atherosclerosis, even with adjusting for shared risk factors including age, smoking, sex, diabetes mellitus and hypertension.[47] Secondly, an important mediator of incident tooth loss in PVD participants is antecedent periodontal disease. Periodontal infection via the oral infection–inflammation pathway may promote systemic inflammation and exacerbate atherogenesis.[2] Tooth loss in turn may lead to compromised masticatory ability, causing an altered diet which may predispose to PVD.[48] Finally, the attitude and approach to dental disease management amongst participants and their dental care providers in different countries may have resulted in increased tooth loss.

Dental prosthesis, other oral diseases and oral hygiene behaviours

The studies reporting on the presence of dental prostheses in PVD participants were non-controlled and descriptive in nature.[14,33,36] Therefore, no inference could be made on this oral health domain in PVD and non-PVD participants. The current literature is also insufficient to establish any difference between PVD and non-PVD participants regarding oral diseases such as Candida infections, periapical lesions and intraosseous foci. For a more complete assessment of these domains, further analytical studies are required.

Poor oral hygiene behaviours have been associated with an increased risk of cardiovascular events and elevated concentrations of inflammatory molecules such as C reactive protein.[49] Conversely, improvements in oral hygiene have been shown to reduce the risk of cardiovascular events.[50] Although an important determinant of oral health, oral hygiene behaviour was assessed in only one study.[20] That study only involved participants diagnosed with AA, who showed poor oral hygiene behaviours. Further analytical studies assessing oral hygiene behaviours in other PVD subsets are required.

Overall appraisal of included studies and recommendations for future research

Cross-sectional and case–control studies were the predominant study designs in this review. Twelve[14,19,21,23–25,33–35,37–39] studies did not adjust for shared risk factors that underpin both tooth loss and certain types of PVD such as age, diabetes mellitus, hypertension and smoking. Lack of adjustments in observational studies is a known limitation.

Key methodological limitations such as the lack of control groups,[14,33–35,38] the use of controls with various cardiac[30–32] or vascular[24] co-morbidities, and unmatched controls have undermined the comparability of results between PVD and non-PVD participants. Furthermore, high heterogeneity of outcome measures and case definitions precluded a quantitative analysis. The case definitions of PVD and the clinical parameters employed for diagnosis were varied between the studies included. Several studies stated the diagnosis of PVD and its subtypes.[31–35,38,39] They did not, however, describe the parameters used to make the diagnosis.

Some studies used imaging modalities to diagnose PVD.[3,20,21,23,28] These studies would have further benefited from categorising different PVD severities according to imaging findings, as this may allow correlation with associated oral health findings. This was evident in one study in which greater severities of periodontitis were seen with increased intima–media thicknesses on imaging.[19] Other studies utilised a combination of clinical and imaging tools to diagnose PVD which would also allow correlation of PVD severity with the oral health findings.[18,22,24,25,30,37] However, none of these studies had performed such correlation, which could have established whether or not increasing PVD severities are associated with poorer oral health findings.

Similarly, differences in case definitions of periodontitis and value ranges for categorising different severities of periodontitis were observed. In addition, variations in the periodontal indices used to assess clinical parameters of periodontal health were apparent. Such variations may cause inaccuracies in the prevalence of periodontitis amongst PVD participants. In general, studies would benefit from using a standardised case definition of periodontitis as recommended by Eke et al.[51] which is aimed for use in population-based research. Standardisation of clinical assessment parameters is also essential in future studies.[52]

Moreover, Bloemenkamp et al.[26] assessed periodontal disease using patient self-reporting. This method was shown to have acceptable validity for large-scale epidemiologic studies surveying periodontal disease.[53] Nevertheless, another review indicated that employing a combination of self-reporting alongside other clinical indicators, such as CAL and PPD, of periodontal disease may be most beneficial;[54] this approach was utilised in two studies.[20,21] Therefore, future studies utilising self-reporting methods amongst PVD participants may benefit by combining them with other parameters of assessing periodontal disease. One study performed periodontal examination one to two months postoperatively on PVD participants who had undergone vascular surgery.[34] There was no mention of whether these participants had received dental care during that period, when their oral health may have worsened.

The strengths of this review relate to the assessing a spectrum of essential oral health domains, other than periodontitis, in participants with various subtypes of PVD as per WHO guidance. The principle limitation was the inability to undertake a quantitative analysis due to high heterogeneity of the included studies. Additionally, only 4 of the 24 studies were considered of high quality (scores greater than six) according to the NOS scale. Further, only 3 of the 16 studies that included controls were graded as high quality. Therefore, the scarcity of high-quality studies compromises the ability to establish a clear description of the oral health status amongst PVD participants. Nor does it permit a meaningful comparison to be made between PVD and non-PVD participants. Several recommendations for future research on this topic are listed in Table 2 based on the format recommended by Brown et al.[55] These recommendations include but are not limited to:

- Future studies to assess the magnitude of the relationship between PVD and oral health parameters other than periodontal disease and tooth loss
- Using standardised case definitions for PVD and periodontitis

Table 2. Research recommendations (based on format from Brown et al., 2006).[55]

Core elements	Recommendation for future research
(E) Evidence (current)	Systematic review identified predominantly cross-sectional and case–control studies.
	Future studies should focus on assessing the magnitude of the relationship between poor oral health findings and PVD.
(P) Population	Adults with PVD aged $\geq$18 years old.
	Standardised clinical parameters for diagnosis of any subset of PVD.
(I) Intervention/exposure	Standardised periodontal probing and mouth examination protocol.
	Assessment of oral health parameters as per the WHO's manual for standardised oral health assessment using Oral Health Assessment and Review Dental Clinical Guidance of the Scottish Dental Clinical Effectiveness Programme.[56]
	Assessment of dental caries indices, oral pathologies, oral hygiene behaviours.
(C) Comparison	Adults aged $\geq$18 years old without PVD.
(O) Outcomes	Periodontal disease prevalence according to Eke et al.[51] definition of periodontitis.
	Dentition status and presence of dental prosthesis in individuals with PVD.
	Prevalence of dental caries and oral pathologies.
	Oral hygiene behaviours in individuals with PVD.

PVD: peripheral vascular disorders.

- Using standardised parameters for PVD diagnosis and oral health assessment according to WHO guidance and customised from Oral Health Assessment and Review Dental Clinical Guidance of the Scottish Dental Clinical Effectiveness Programme.[56]
- Assessing the effect of oral health treatment on PVD.

Conclusion

Due to the paucity of the high-quality studies addressing oral health domains other than periodontal disease and tooth loss, a definitive conclusion regarding oral health status/conditions in individuals with PVD could not be made. Therefore, it is not yet possible to make an evidence-based recommendation regarding the value of routine oral health assessment in individuals with PVD. However, on considering the evidence regarding the link between oral and systemic health, it would be good practice to advise on oral health assessment and maintenance in individuals with PVD.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical statement

No application to the Research Ethics Services was required for this study.

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ORCID iD

Sayed Abdulmotaleb Almoosawy https://orcid.org/0000-0002-9236-0470

References

1. Mendez MV, Scott T, LaMorte W, et al. An association between periodontal disease and peripheral vascular disease. *Am J Surg* 1998; 76: 153–157.
2. Hung HC, Willett W, Merchant A, et al. Oral health and peripheral arterial disease. *Circulation* 2003; 107: 1152–1157.
3. Sánchez-Siles, Rosa-Salazar V, Camacho-Alonso F, et al. Association between periodontal disease and venous thromboembolic disease. *Quintessence* 2013; 44: 567–573.
4. Salhi L, Rompen E, Sakalihasan N, et al. Can periodontitis influence the progression of abdominal aortic aneurysm? A systematic review. *Angiology* 2019; 70: 479–491.
5. Cowan LT, Lakshminarayan K, Lutsey PL, et al. Periodontal disease and incident venous thromboembolism: the Atherosclerosis Risk in Communities study. *J Clin Periodontol* 2019; 46: 12–19.
6. Schillinger T, Kluger W, Exner M, et al. Dental and periodontal status and risk for progression of carotid atherosclerosis: the inflammation and carotid artery risk for atherosclerosis study dental substudy. *Stroke* 2006; 37: 2271–2276.
7. ICD 2020. ICD-10-CM Diagnosis Code I73.9: peripheral vascular disease, unspecified, www.icd10data.com/ICD10CM/Codes/I00-I99/I70-I79/I73-/I73.9 (2020, accessed 8 April 2020).
8. Yang S, Zhao LS, Cai C, et al. Association between periodontitis and peripheral artery disease: a systematic review and meta-analysis. *BMC Cardiovasc Disord* 2018; 18: 141.
9. Peterson PE, Baez RJ and World Health Organisation. *Oral health surveys: basic methods*. Geneva, Switzerland: World Health Organisation, 2013.
10. Uyar IS, Akpinar MB, Sahin V, et al. Carotid and popliteal artery intima-media thickness in patients with poor oral hygiene and the association with acute-phase reactants. *Cardiovasc J Afr* 2013; 24: 308–312.
11. Uyar IS, Sahin V, Akpinar MB, et al. Does oral hygiene trigger carotid artery intima-media thickness? *Heart Surg Forum* 2013; 16: 232–236.
12. Lertpimonchai A, Rattanasiri S, Arj-Ong Vallibhakara S, et al. The association between oral hygiene and periodontitis: a systematic review and meta-analysis. *Int Dent J* 2017; 67: 332–343.
13. Loesche WJ. Association of the oral flora with important medical diseases. *Curr Opin Periodontol* 1997; 4: 21–28.
14. Stansby G, Byrne MTL and Hamilton G. Dental infection in vascular surgical patients. *Br J Surg* 1994; 81: 1119–1120.
15. Wilson WR, Bower TC, Creager MA, et al. Vascular graft infections, mycotic aneurysms, and endovascular infections: a Scientific Statement from the American Heart Association. *Circulation* 2016; 134: e412–e460.
16. ICD 2020. DRG List Page, www.icd10data.com/ICD10CM/DRG/299 (2020, accessed 9 April 2020).
17. Wells G, Shea B, O'Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality if non-randomized studies in meta-analyses, www.ohri.ca/programs/clinical_epidemiology/oxford.asp (2012, accessed 23 September 2020).
18. Chen YW, Umeda M, Nagasawa T, et al. Periodontitis may increase the risk of peripheral arterial disease. *Eur J Vasc Endovasc Surg* 2008; 35: 153–158.
19. Nicolaiciuc O, Sufaru IG, Sioustis I, et al. Study on the association between the severity of chronic periodontitis and carotid atherosclerosis. *Rom J Oral Rehabil* 2018; 10: 88–94.

20. Ding F, Wu D, Han X, et al. Oral hygiene and periodontal conditions in the Chinese patients with aortic aneurysm. *BMC Oral Health* 2018; 18: 136.
21. Ding F, Lyu YL, Han X, et al. Detection of periodontal pathogens in the patients with aortic aneurysm. *Chin Med J (Engl)* 2014; 127: 4114–4118.
22. Chen YW, Iwai T, Umeda M, et al. Elevated IgG titers to periodontal pathogens related to Buerger disease. *Int J Cardiol* 2007; 122: 79–81.
23. Kurihara N, Inoue Y, Iwai T, et al. Detection and localization of periodontopathic bacteria in abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 2004; 28: 553–558.
24. Iwai T, Inoue Y, Umeda M, et al. Oral bacteria in the occluded arteries of patients with Buerger disease. *J Vasc Surg* 2005; 42: 107–115.
25. Toyofuku T, Inoue Y, Kurihara N, et al. Differential detection rate of periodontopathic bacteria in atherosclerosis. *Surg Today* 2011; 41: 1395–1400.
26. Bloemenkamp DGM, Van Den Bosch MAAJ, Mali WPTM, et al. Novel risk factors for peripheral arterial disease in young women. *Am J Med* 2002; 113: 462–467.
27. Soto-Barreras U, Olvera-Rubio JO, Loyola-Rodriguez JP, et al. Peripheral arterial disease associated with caries and periodontal disease. *J Periodontol* 2013; 84: 486–494.
28. Friedlander AH, Sung EC, Chung EM, et al. Radiographic quantification of chronic dental infection and its relationship to the atherosclerotic process in the carotid arteries. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2010; 109: 615–621.
29. Çalapkorur MU, Alkan BA, Tasdemir Z, et al. Association of peripheral arterial disease with periodontal disease: analysis of inflammatory cytokines and an acute phase protein in gingival crevicular fluid and serum. *J Periodont Res* 2017; 52: 532–539.
30. Aoyama N, Suzuki J, Ichi Kobayashi N, et al. Periodontitis deteriorates peripheral arterial disease in Japanese population via enhanced systemic inflammation. *Heart Vessel* 2017; 32: 1314–1319.
31. Suzuki JI, Aoyama N, Aoki M, et al. High incidence of periodontitis in Japanese patients with abdominal aortic aneurysm. *Int Heart J* 2014; 55: 268–270.
32. Suzuki J, Ichi Aoyama N, Aoki M, et al. Incidence of periodontitis in Japanese patients with cardiovascular diseases: a comparison between abdominal aortic aneurysm and arrhythmia. *Heart Vessel* 2015; 30: 498–502.
33. Häyrinen-Immonen R, Ikonen TS, Lepäntalo M, et al. Oral health of patients scheduled for elective abdominal aortic correction with prosthesis. *Eur J Vasc Endovasc Surg* 2000; 19: 294–298.
34. Figuero E, Lindahl C, Marín MJ, et al. Quantification of periodontal pathogens in vascular, blood, and subgingival samples from patients with peripheral arterial disease or abdominal aortic aneurysms. *J Periodontol* 2014; 85: 1182–1193.
35. Fernandes CP, Oliveira FAF, Silva PGDB, et al. Molecular analysis of oral bacteria in dental biofilm and atherosclerotic plaques of patients with vascular disease. *Int J Cardiol* 2014; 174: 710–712.
36. Hamasha AAH. Medical conditions associated with missing teeth and edentulism in the institutionalized elderly. *Spec Care Dentist* 1998; 18: 123–127.
37. Igari K, Inoue Y and Iwai T. The epidemiologic and clinical findings of patients with Buerger disease. *Ann Vasc Surg* 2016; 30: 263–269.
38. Zaremba M, Górska R and Leszczyński J. The role of periopathogens in pathogenenesis of atherosclerotic disease. *Acta Angiol* 2012; 18: 99–109.
39. Cairo F, Gaeta C, Dorigo W, et al. Periodontal pathogens in atheromatous plaques. A controlled clinical and laboratory trial. *J Periodont Res* 2004; 39: 442–446.
40. Needleman I. The good practitioner's guide to periodontology. *Br Soc Periodontol* 2016; 10–14.
41. Jansma J and Vissink A. Dental foci. Role, treatment and prophylaxis in patients at risk. *Ned Tijdschr Tandheelkd* 1998; 105: 52–56.
42. Kurihara N, Inoue Y, Iwai T, et al. Oral bacteria are a possible risk factor for valvular incompetence in primary varicose veins. *Eur J Vasc Endovasc Surg* 2007; 34: 102–106.
43. Hippisley-Cox J and Coupland C. Development and validation of risk prediction algorithm (QThrombosis) to estimate future risk of venous thromboembolism: prospective cohort study. *Br Med J* 2011; 343: 1–12.
44. Roth GA, Aumayr K, Giacona MB, et al. Porphyromonas gingivalis infection and prothrombotic effects in human aortic smooth muscle cells. *Thromb Res* 2009; 123: 780–784.
45. Roth GA, Moser B, Huang SJ, et al. Infection with a periodontal pathogen induces procoagulant effects in human aortic endothelial cells. *J Thromb Haemost* 2006; 4: 2256–2261.
46. Ahn YB, Shin MS, Han DH, et al. Periodontitis is associated with the risk of subclinical atherosclerosis and peripheral arterial disease in Korean adults. *Atherosclerosis* 2016; 251: 311–318.
47. Asai K, Yamori M, Yamazaki T, et al. Tooth loss and atherosclerosis: the Nagahama study. *J Dent Res* 2015; 94: 52S–58S.
48. Al-Shammari KF, Al-Khabbaz AK, Al-Ansari JM, et al. Risk indicators for tooth loss due to periodontal disease. *J Periodontol* 2005; 76: 1910–1918.
49. De Oliveira C, Watt R and Hamer M. Toothbrushing, inflammation, and risk of cardiovascular disease: results from Scottish Health Survey. *Br Med J* 2010; 340: c2451.
50. Park SY, Kim SH, Kang SH, et al. Improved oral hygiene care attenuates the cardiovascular risk of oral health disease: a population-based study from Korea. *Eur Heart J* 2019; 40: 1138–1145.
51. Eke PI, Page RC, Wei L, et al. Update of the case definitions for population-based surveillance of periodontitis. *J Periodontol* 2012; 83: 1449–1454.
52. Gupta N, Rath SK and Lohra P. Comparative evaluation of accuracy of periodontal probing depth and

attachment levels using a Florida probe versus traditional probes. *Med J Armed Forces India* 2015; 71: 352–358.
53. Abbood HM, Hinz J, Cherukara G, et al. Validity of self-reported periodontal disease: a systematic review and meta-analysis. *J Periodontol* 2016; 87: 1474–1483.
54. Blicher B, Joshipura K and Eke P. Validation of self-reported periodontal disease: a systematic review. *J Dent Res* 2005; 84: 881–890.
55. Brown P, Brunnhuber K, Chalkidou K, et al. How to formulate research recommendations. *Br Med J* 2006; 333: 804–806.
56. SDCEP. Oral health assessment and review. *Scottish Dental Clinical Effectiveness Programme*, www.sdcep.org.uk/wp-content/uploads/2015/04/SDCEP-OHAR-Version-1.0.pdf (2012, accessed 3 August 2020).