Review

Generalized Aggressive Periodontitis as a Risk Factor for Dental Implant Failure: A Systematic Review and Meta-Analysis

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Background: Dental implant placement is a widely used treatment that provides functional and esthetic resolution for patients suffering from tooth loss. However, the incidence of peri-implant diseases has been rising recently. Periodontal diseases and peri-implant diseases share many similarities. Hence, it is important to find out whether patients with aggressive periodontal disease possess a higher risk of developing peri-implant diseases. The aim of this study is to study whether generalized aggressive periodontitis (GAgP) has similar survival rates (SRs) and marginal bone loss (MBL) when compared with patients with chronic periodontitis (CP) and/or healthy patients (HPs).

Methods: An electronic literature search was conducted by one reviewer (AM) in several databases from 2000 to 2013, including MEDLINE, EMBASE, Cochrane Central Register of Controlled Trials, and Cochrane Oral Health Group Trials Register databases, for articles written in English up to November 2013. Human clinical trials, either prospective or retrospective, that compared implant SR and MBL in patients with a history of GAgP versus those with CP or HPs were included.

Results: A total of six non-randomized prospective clinical trials met the inclusion criteria. The results showed SRs of 83.3% to 100% (GAgP), 96.4% to 100% (CP), and 96.9% to 100% (HP) over a mean period of 48.01 ± 71.99 months, with an overall risk ratio of 0.96 (95% confidence interval [CI] = 0.91 to 1.01, $P = 0.14$, GAgP versus HP) and 0.94 (95% CI = 0.87 to 1.01, $P = 0.09$, GAgP versus CP). However, when the "failure rate" as studied outcome was examined, meta-analysis presented an overall risk ratio of 4.00 for the comparison between patients with AgP and HPs and an overall risk ratio of 3.97 when compared with patients with CP. The MBL weighted mean difference for each subgroup was 0.15 mm (95% CI = 0.04 to 0.26, HP versus CP), –0.28 mm (95% CI = –0.36 to –0.19, HP versus GAgP), and –0.43 mm (95% CI = –0.53 to –0.33, CP versus GAgP) over a mean period of 30 ± 18 months.

Conclusions: Implant placement in patients with a history of GAgP might be considered a viable option to restore oral function with survival outcomes similar to those found in both patients with CP and HPs. However, the risk ratio for failure in patients with AgP is significantly higher when compared with HPs (4.0) and those with CP (3.97). *J Periodontol 2014;85:1398-1407.*

KEY WORDS

Dental implantation, endosseous; evidence-based dentistry; meta-analysis; periodontal diseases; periodontitis; review.

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Success in implant dentistry relies on the initial osseointegration and long-term stability.[1] Patient systemic factors and susceptibility to periodontal diseases, implant macrodesign and microdesign, and periodontal pathogenic bacteria, among others, have all been shown to play a role in achieving long-term implant stability.[2] Consequently, many clinical studies have aimed at analyzing the effect of a history of previous periodontal disease on implant treatment success.[3-10] Although a higher incidence of peri-implantitis and a lower implant survival rate (SR) were reported in patients susceptible to periodontitis,[11,12] there is still disagreement on the level of this relationship.[13,14] As occurs in natural dentition, once the disease is established and progressing, it determines implant prognosis. In this sense, it is important to mention that the preexisting ecologic conditions of the oral cavity influence biofilm formation on implants. It has been established that residual pockets act as niches of infection for dental implants[15] and that putative periodontal pathogens are present even 1 year after periodontally affected teeth are extracted.[16] Furthermore, it also has been established that putative periodontal pathogens increase with longer loading time and that this increase is more accentuated in patients with a history of periodontitis or peri-implant infections.[17]

Whereas chronic periodontitis (CP), associated with either local or systemic factors, is the most common form of the disease, aggressive periodontitis (AgP) is less frequent (<1% of the population).[18] However, the presence of potential risks such as AgP may also have an influence on implant success. Despite some common histopathologic characteristics shared between chronic and aggressive forms[19] and the different criteria and methods that were used to diagnose and define AgP, three major characteristics were used to define the aggressive disease: 1) clinically healthy with the exception of periodontitis; 2) rapid attachment loss (AL) and bone breakdown; and 3) familial aggregation.[20,21] It often occurs in younger patients (<30 years of age), specifically the localized form, but it may also affect older patients.[22] Other characteristics can also be used in the diagnosis of the disease: 1) amounts of microbial deposits inconsistent with the severity of periodontal tissue breakdown; 2) elevated proportions of *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis*; 3) phagocyte abnormalities; 4) hyper-responsive macrophage phenotype, including elevated levels of prostaglandin E_2 and interleukin (IL)-1β; and 5) self-arresting progression of AL and bone loss.[20-22] Polymorphisms in genes regulating the expression of IL-1, IL-6, IL-10, tumor necrosis factor, E-selectins, Fc-γ receptor, cluster of differentiation 14, toll-like receptors, caspase recruitment domain 15, vitamin D receptor, lactoferrin, caldesmon, heat shock protein 70, and Stac protein[23] and major histocompatibility complexes A9 and B15[24] were associated with AgP. As a consequence of these polymorphisms, the inflammatory profile is altered, including, but not limited to, polymorphonuclear neutrophil (PMN) transendothelial migration and signaling functions,[25] reduced chemotactic response, and depression in neutrophil phagocytosis and superoxide production.[26]

Assuming that each periodontal entity has a distinct progressive pattern and different bacteria associated with it, it is critical to note that the numerous factors related to implant failure and the absence of long-term studies in association with a history of generalized AgP (GAgP) do not permit the drawing of noticeable correlations with implant survival/success. Nonetheless, it is necessary to treat and control periodontal disease, regardless of its progression pattern and subtype, before implant therapy is initiated to improve implant long-term treatment success.[12]

Several studies[3-8] aimed at analyzing the influence of a history of aggressive periodontal disease on implant treatment outcome in terms of SR and marginal bone loss (MBL). Results from these longitudinal studies suggest that patients with GAgP experienced higher implant failure rates when compared with patients with CP and healthy patients (HP). However, there is still no consensus, which is achievable by a well-designed systematic review that clarifies the effect of previous history of GAgP on implant treatment outcome. Henceforth, the present study aims at assessing whether patients who suffered from GAgP have a higher implant failure rate and MBL in implant prostheses when compared with patients with CP and/or HPs.

MATERIALS AND METHODS

Information Sources and Development of Focused Question

An electronic literature search was conducted by one reviewer (AM) in several databases, including MEDLINE, EMBASE, Cochrane Central Register of Controlled Trials, and Cochrane Oral Health Group Trials Register databases, for articles written in English from January 2000 through November 2013. The following PICO (patient, intervention, comparison, and outcome) question was aimed to be answered: Do edentulous patients restored with implant-supported prostheses have a higher or similar implant SR and/or MBL among patients with a history of GAgP and/or HPs and/or patients with CP? The reporting of these meta-analyses adhered to the Preferred Reporting Items for Systematic Review and Meta-Analyses statement.[27]

Screening Process

Combinations of controlled terms (MeSH and EMTREE) and keywords were used whenever possible. The search terms used for the search in PubMed were as follows (in which [mh] represents the MeSH terms, [tiab] represents title and/or abstract, [pt] represents publication type, and [la] represents language): (“periodontitis”[mh] OR “aggressive periodontal disease”[tiab] OR (“dental implantation, endosseous”[mh] OR “dental implants”[mh]) AND (“aggressive periodontitis”[tiab] OR “aggressive periodontitis”[mh])) AND (generalized [tiab]) AND English [la] NOT (letter [pt] OR comment [pt] OR editorial [pt]) NOT (“animals”[mh] NOT “humans”[mh]). For the screening process in EMBASE, Cochrane Central Register of Controlled Trials, and Cochrane Oral Health Group Trials Register databases, the following terms were used: “aggressive periodontal disease,” “aggressive periodontitis” in combination with “dental implants” or “endosseous implants.” Additionally, a manual search of implant-related journals, including *Clinical Implant Dentistry and Related Research*, *Journal of Oral and Maxillofacial Implants*, *Clinical Oral Implants Research*, *Implant Dentistry*, *Journal of Dental Research*, *Journal of Clinical Periodontology*, *Journal of Periodontology*, and *The International Journal of Periodontics & Restorative Dentistry*, from November 2012 through November 2013, was also performed to ensure a thorough screening process. References in the excluded articles were also checked to find studies that fulfilled the inclusion criteria.

Eligibility Criteria

Articles were included in this meta-analysis if they met the following inclusion criteria: prospective or retrospective human clinical trials in which the outcomes of the implant-supported prosthesis in patients with a history of GAgP were studied and compared with the results obtained by a second and/or third group (HPs and/or patients with a history of CP). Furthermore, the articles included had to report the implant survival for ≥12 months after loading. Accordingly, several factors were extracted from the selected studies and analyzed (if possible): 1) the number of implants included; 2) implant design, length, and width; 3) location of the implants; 4) MBL; 5) success rate; 6) age; 7) patient’s systemic conditions; 8) smoking; 9) bone augmentation procedure before implant placement; and 10) microorganisms present during the last follow-up appointment. Conversely, case reports, systematic reviews, animal studies, studies with no control group, and those studies in which information was not clear enough or was inconsistent were excluded from this meta-analysis. In addition, it is of paramount importance to note that non-randomized clinical trials might be subjected to a higher risk of bias.[28] For that reason, the Newcastle-Ottawa scale (NOS) was used to assess, by two masked examiners (AM and MP-M), the quality of such studies for a proper understanding of non-randomized studies.[29]

Data Analyses

The primary outcome was SR, and MBL was the secondary outcome. The risk ratio of SR was estimated using a computer program.‡ The contribution of each article was weighed. Random-effects meta-analyses of the selected studies were applied to avoid any bias caused by methodologic differences among studies. Forest plots were produced to graphically represent the difference in outcomes of AgP and CP or AgP and HPs for all included studies using implant as the analysis unit. $P = 0.05$ was used as the level of significance. Heterogeneity was assessed with χ^2 test and I^2 test, which ranges from 0% to 100%, and lower values represent less heterogeneity. In addition, the funnel plot was used to assess for the presence of publication bias. In addition, a regression of year on logit event rate was performed to ascertain the effect of implant failure rate in AgP versus the year of follow-up. Authors did not explicitly state through the included articles the definition for “implant failure.” However, according to the accepted terminology in this matter, “failure” is considered when implant can no longer be in function because of mobility. Accordingly, to obtain “implant failure rates” and meta-analyses, they were deducted to 100, and the corresponding SR (percentage) was reported.

The weighted mean difference (WMD) and 95% confidence interval (CI) of MBL were calculated between three subgroups: 1) HP versus CP; 2) HP versus GAgP; and 3) CP versus GAgP. Bar charts were used to present the results of comparisons.

RESULTS

Study Selection

The database search resulted in a total of 120 articles, of which 68 potentially relevant articles were selected after an evaluation of their titles and abstracts. After the initial screening, 18 articles were selected for additional evaluation of the full-text version (for being directly related with the aim of the present study). Of these, only six articles fulfilled the inclusion criteria and were subsequently analyzed in this meta-analysis (Fig. 1). Details of all included studies[3,4,6,30-32] are summarized in Table 1. Reasons for exclusion were as follows: 1) case reports (n = 7); 2) no control group (n = 1); and 3)

‡ Review Manager v.5.0, The Nordic Cochrane Centre, The Cochrane Collaboration, Copenhagen, The Netherlands.

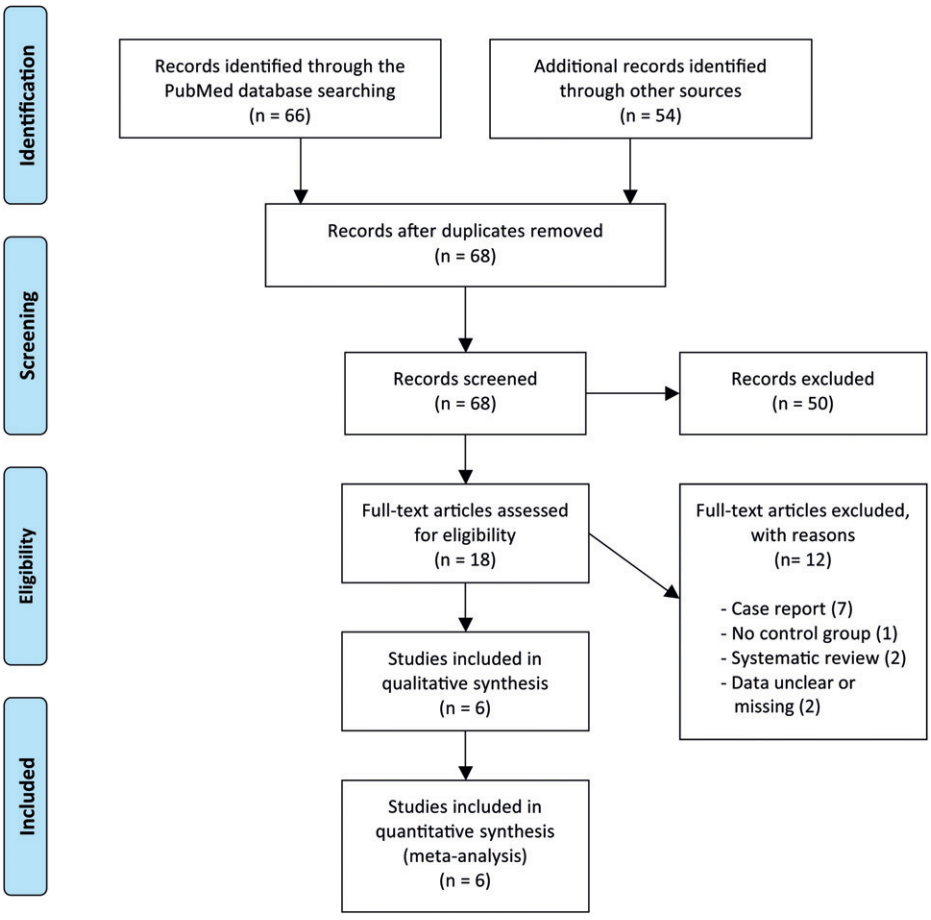

Figure 1.
Flowchart of the screening process used for this systematic review. Records excluded (n = 50) did not meet inclusion criteria.

systematic reviews (n = 2). References of the excluded articles have been included in supplementary Figure 1 (in online *Journal of Periodontology*). In addition, two more studies were excluded because of a lack of either clear data display or the data needed for the analysis.[10,33] Conversely, all the included studies[3,4,6,30-32] were comparative prospective controlled trials assessing implant treatment outcome in patients with GAgP compared with HPs and/or those with CP. From one of the included studies,[6] data at the 1-year follow-up evaluation was the only available data comparable with the other group (CP) and, therefore, the only data that could be extracted and analyzed.

Study Quality

All the articles included in the present systematic review are prospective human clinical trials evaluating implant survival in patients with a history of GAgP. The NOS was used to assess the quality of such studies for a proper understanding of non-randomized studies.[29] It is important to state that five[4,6,30-32] of the six included articles were developed by the same group (Department of Periodontology, Dental School of Medicine, Philipps University, Marburg, Germany). This may lead to some risk of bias attributable to data overlapping. In addition, some data were not clear enough to be extracted from these studies (i.e., standard deviation of MBL or success rate). Nonetheless, according to the NOS, a mean score of 6.33 ± 0.66 (33.3% [seven stars], 66.6% [six stars]) was obtained, showing the "medium-high" level of evidence of the included studies.

Effect of GAgP on SR

The SR of the included studies ranged from 83.3% to 100% for the GAgP group, 96.4% to 100% for the CP group, and 96.9% to 100% for the HP group. Meta-analysis for the comparison of SR among selected studies presented an overall risk ratio of 0.96 (95% CI = 0.91 to 1.01) for the comparison between AgP and HP with no significant difference ($P = 0.14$) (Fig. 2). For the comparison of SR between AgP and CP, an overall risk ratio of 0.94 (95% CI = 0.87 to 1.01) with $P = 0.09$ was found (Fig. 2). The comparisons presented a low (P value for χ^2 test = 0.14 and I^2 test = 14%) to moderate (P value for χ^2 test = 0.09 and I^2 test = 39%) heterogeneity among the pooled data, for the former and latter comparisons, respectively.

However, when the event "failure rate" as studied outcome was examined, meta-analysis presented an overall risk ratio of 4.00 (95% CI = 1.79 to 8.93) for the comparison among patients with AgP and HPs, and a statistically significant difference ($P < 0.001$) was found (Fig. 3). For the comparison of failure rate between patients with AgP and CP, an overall risk ratio of 3.97 (95% CI = 1.68 to 9.37) and a statistically significant difference ($P < 0.001$) were detected (Fig. 3). The comparisons presented a low heterogeneity among the pooled data.

Effect of GAgP on MBL

Forest plots were used to show the heterogeneity among the pooled data. Three separate bar charts were subsequently generated to present the WMD of each subgroup: HP versus CP, HP versus GAgP, and CP versus GAgP. For HP versus CP, WMD was

Table 1.

Prospective Studies Qualifying for the Data Analysis and Related Extracted Data

Study (year)	Groups	Patients (n)	Implants (n)	Implant System	Implant Length (mm)	Implant Width (mm)	Location	Bone Augmentation Before Implant Placement	Follow-Up Period (months)	Mean age (years)	Smoking	Systemic Disease	Microorganisms During the Follow-Up	Prosthesis Type	Implant Survival (%)	Implant Success Rate (%)	Implant Failure (%)	MBL (mm)
De Boever et al. (2009)[3]	HP	110	261	§	12/10/14	3.3/4.1/4.8	Mandible, 41.1%; maxilla, 58.9%	No augmentation, 77.4%; augmentation, 22.6%	48.1	53.8 ± 15.08	No smokers, 78%	NA	NA	SC/FPD/TIP/	96.94	NA	3.6	NC
	CP	68	193					No augmentation, 70.2%; augmentation, 29.8%			Current smokers, 11.4%	Systemic disorder, 5.8%			96.36		3.62	
	GAgP	16	59								Quit, 10.3%	Systemic disorder, 56.2%			84.75		15.25	
Mengel et al. (2001)[6]	CP	5	12	‖	10/13/15/18	NA	Maxilla/ mandible	NA	12	79	NA	NA	Cocci, 76.15%; non-motile rods, 13.78%; motile rods, 8.05%	SC/FPD	100	100	0	0.13
	GAgP	5	36							37.5			Cocci, 79.57%; non-motile rods, 7.11%; motile rods, 12.10%		88.8	94.4	5.6	0.60
Mengel and Flores-de-Jacoby (2005)[30]	HP	10	11	‖	10/13/15	NA	Anterior maxilla	No augmentation	36	34.5	Non-smokers	None	Cocci, 70.35%; non-motile rods, 12.57%; motile rods, 7.82%	SC	100	NA	0	1.40
	GAgP	10	15					GBR with titanium-reinforced membrane¶ + titanium screws					Cocci, 64.23%; non-motile rods, 11.08%; motile rods, 9.69%		100		0	1.78
Mengel and Flores-de-Jacoby (2005)[31]	HP	12	30	§/‖	NA	NA	Mandible, 43.4%; maxilla, 56.5%	NA	36	31	NA	NA	Cocci, 70.35%; non-motile rods, 12.57%; motile rods, 7.82%	SC/FPD	100	NA	0	0.70
	CP	12	43							34			Cocci, 68.56%; non-motile rods, 14.16%; motile rods, 8.06%		100		0	0.86
	GAgP	15	77							32			Cocci, 67.87%; non-motile rods, 15.40%; motile rods, 7.67%		97.4		2.6	1.14
Mengel et al. (2007)[32]	HP	8	13	§/#	NA	NA	Mandible, 34%; maxilla, 66%	NA	36	31	Non-smokers	None	NC	Removable	100	NC	0	0.71
	GAgP	9	41							34					97.6		2.4	1.29
Mengel et al. (2007)[4]	HP	5	7	‖	NA	NA	Mandible, 41.9%; maxilla, 58.1%	NA	120	35.5	NA	Multiple sclerosis (1 patient)	Cocci, 86.75%; non-motile rods, 8.75%; motile rods, 1.5%	SC/FPD/ removable	100	100	0	NC
	GAgP	5	36							32.5			Cocci, 70.4%; non-motile rods, 13.78%; motile rods, 6.58%		83.33	77.75	16.66	

NA = not available; SC = single crown; FPD = fixed partial denture; TIP = tooth-to-implant prostheses; NC = not clear; GBR = guided bone regeneration.
§ Institute Straumann, Basel, Switzerland.
‖ Nobel Biocare, Gothenburg, Sweden.
¶ W. L. Gore & Associates, Newark, DE.
# Biomet 3i, Palm Beach, FL.

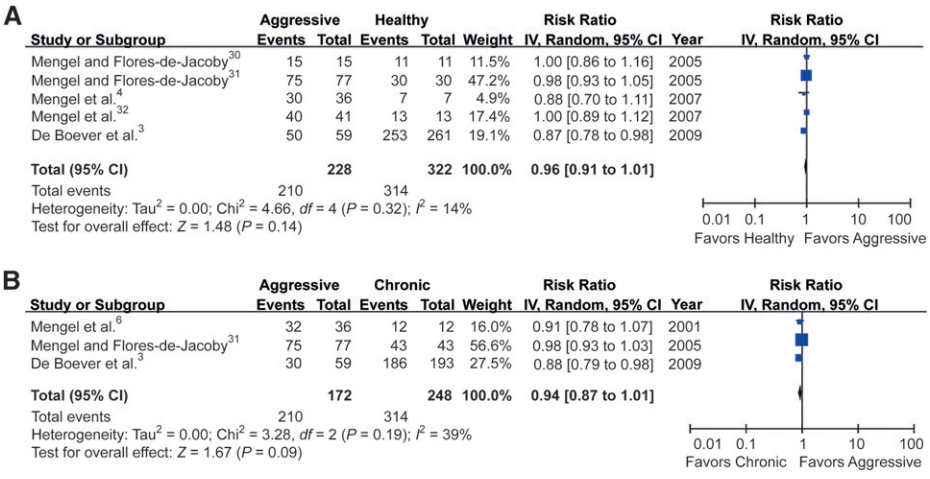

Figure 2.
*Meta-analysis for the comparison of SR between patients with GAgP and HPs **(A)** and between patients with GAgP and CP **(B)**. IV = inverse model.*

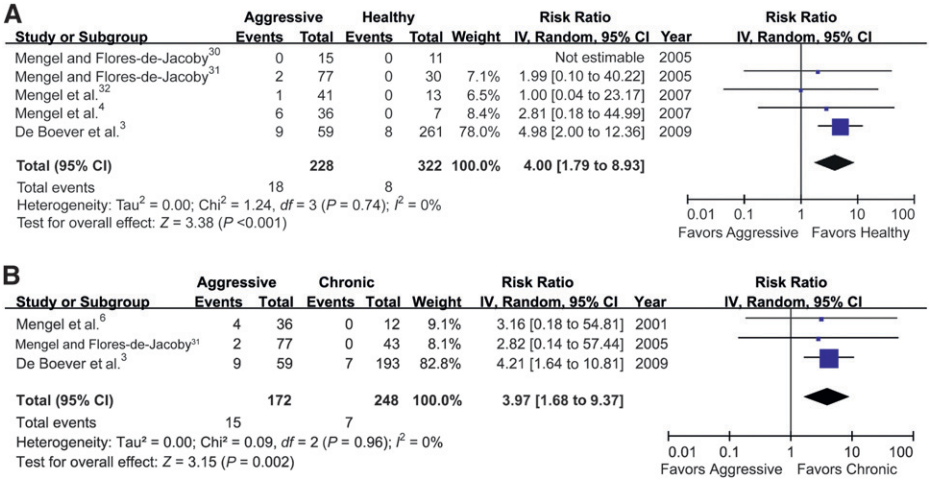

Figure 3.
*Meta-analysis for the comparison of failure rate between patients with GAgP and HPs **(A)** and between patients with GAgP and CP **(B)**. IV = inverse model.*

0.15 mm, with 95% CI = 0.04 to 0.26 mm, favoring the CP group. For HP versus GAgP, WMD was –0.28 mm, with 95% CI = –0.37 to –0.19 mm, favoring the HP group. The WMD for each subgroup was 0.15 mm (95% CI = 0.04 to 0.26, HP versus CP, favoring CP), –0.28 mm (95% CI = –0.36 to –0.19, HP versus GAgP, favoring HP), and –0.43 mm (95% CI = –0.53 to –0.33, CP versus GAgP, favoring CP) (Fig. 4).

Effect of Follow-Up Period on SR of GAgP

The length of follow-up period was analyzed using meta-regression. The results of regression analysis showed that the length of follow-up period did not significantly influence the outcome in either HP versus GAgP ($P = 0.72$) or CP versus GAgP ($P = 0.94$). Additionally, as shown in Figure 5, no statistically significant difference was found regarding the regression of year on logit event rate for implant failure rate in GAgP ($P = 0.38$).

DISCUSSION

Implant survival is no longer considered a challenge; however, in the pursuit of excellence in implant dentistry, longevity must be sought. Many factors were shown to influence the final outcome in implant therapy. Among others, patients with a previous history of periodontal disease and smokers may exhibit higher incidences of implant failure and complications than patients without such conditions.[34] Thus, the current systematic review aims to determine the risk of the aggressive entity of periodontal disease on implant treatment outcome. It was evidenced that, within the limitations, when evaluating SR as the event, patients with a history of GAgP had similar SRs when compared with the CP and HP groups. Nonetheless, when the "failure rate" event was examined, a risk ratio of 4.0 was found compared with HPs and 3.97 when compared with patients with a history of CP. However, because of the small sample size of the "failed implants" group, it is not possible to draw conclusive statements regarding the risk ratio. Furthermore, results from this study must be interpreted cautiously because of the limitations in the studies included.

Many studies displayed the factors that may trigger pathologic bone resorption,[35,36] which can be caused by either biomechanical[37] or biologic/microbial factors.[34] Apse et al.[38] stated that the peri-implant sulcus behaves similar to the periodontal sulcus, and, therefore, an inflammatory process similar to periodontitis occurs around implants, i.e., peri-implantitis. This fact was supported recently by Safii et al.,[39] who showed that higher MBL occurred in patients with a history of periodontal disease than in periodontally

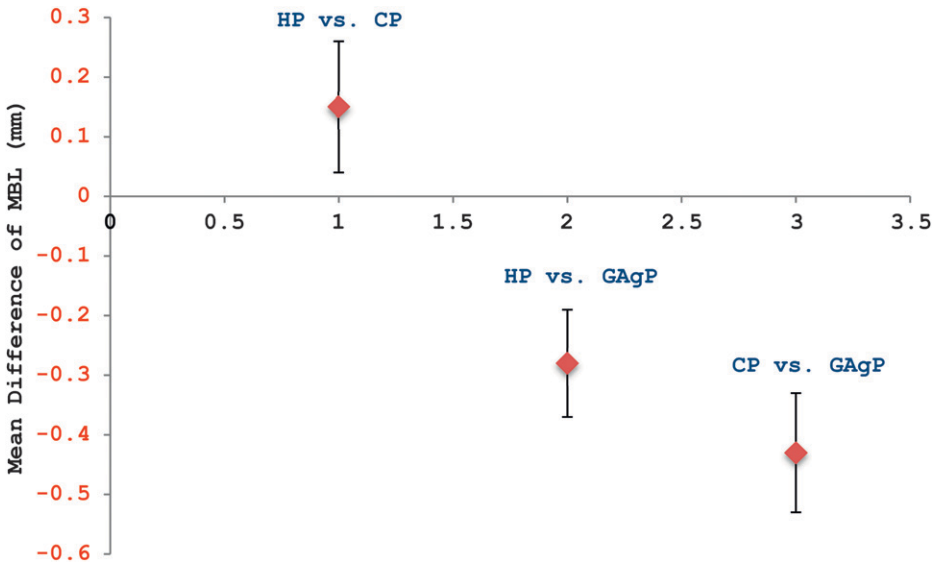

Figure 4.
Funnel plot to show the weighted mean of MBL for HP versus CP, HP versus GAgP, and CP versus GAgP.

Figure 5.
Regression of year on logit event rate for implant failure rate in GAgP.

healthy individuals. Nevertheless, the question remains whether the peri-implantitis is the consequence of a primary infectious process or, on the contrary, whether the infection takes place after the tissue breaks down. Recently, Rasperini et al.[8] found that MBL around implants was correlated with the initial diagnosis of periodontitis and smoking history, in agreement with previous studies.[40-42] Consequently, a higher MBL promotes better environment for bacterial colonization and resulted in a more rapid progression of peri-implantitis. Similarly, Mengel et al.[4] showed that patients with GAgP exhibited MBL of 2.07 mm during the first year after implant placement and 3.37 mm at the end of the 10-year follow-up, and the MBL is more statistically significant than in HPs. De Boever et al.[3] demonstrated that MBL is not only significantly higher in patients with GAgP, but it was also related to bleeding on probing, age, inflammation, presence of plaque, probing depth, and smoking. In agreement with these findings, the present systematic review illustrates that individuals with a history of GAgP displayed higher MBL when compared with HPs or those with CP, but it has to be interpreted cautiously because the amount is relatively small (0.28 to 0.43 mm) and might not have clinical significance. In addition, it is interesting to mention that, as shown by Cho-Yan Lee et al.[43] and validated recently by a systematic review conducted by Atieh et al.,[44] a history of CP does not represent a risk factor for peri-implantitis as long as patients are enrolled in a maintenance program. However, limitations of the present study must be highlighted when interpreting the present results to avoiding misunderstanding. Most of the included studies did not report the standard deviation, so it was not possible to analyze the data obtained for MBL.

Investigations demonstrated the transmission of putative periodontal pathogens from periodontally involved areas to implant sites. Mombelli et al.[45] identified the same pathogens in peri-implant lesions as the ones that were present 6 months before in natural dentition. In this sense, De Boever and De Boever[46] identified periodontal pathogens in the peri-implant sulcus up to 6 months after implant placement in partially edentulous individuals successfully treated for advanced AgP. Regarding the bacterial composition in the peri-implant mucosa, it was shown that an increase of 20% in the proportion of spirochetes and a decrease of cocci from 61% up to 47.5% occurred in the development of peri-implant disease.[47] Mengel and Flores-de-Jacoby[30] showed a higher number of motile rods at the end of the follow-up (3 years),

although this was not statistically significant compared with HPs (7.82% versus 9.69%, respectively). Interestingly, in the follow-up of these results on their prospective study showing long-term results,[4] the difference between HPs and patients with GAgP became statistically significant (cocci, 86.75%; non-motile rods, 8.75%; motile rods, 1.5% versus cocci, 70.4%; non-motile rods, 13.78%; and motile rods, 6.58%, respectively). As stated,[48] a higher peri-implant bone resorption promotes bacterial colonization and a more rapid progression of peri-implantitis; hence, once the initial lesion has taken place, the condition readily worsens by increasing in number the percentage of non-motile microorganisms. Nevertheless, for short- to midterm, relative homogeneity of the microorganism distribution was found. Henceforth, this finding stresses the fact that the etiologic factor of this entity (GAgP) does not depend on the putative pathogenic bacteria but on the host susceptibility.

In addition to this suggestive evidence, it is important to emphasize that patients with GAgP are susceptible for reasons beyond the presence of particular pathogens.[49] As mentioned above, there are a number of factors, often unmodifiable, in patients with AgP that would potentially play a role in implant success. These factors include the following: 1) genetic polymorphisms[23,24]; 2) alterations of the immune system (phagocyte abnormalities and hyper-responsive macrophage phenotype,[20-22] altered PMN transendothelial migration and signaling functions,[25] reduced chemotactic response,[50] and depression in phagocytosis and superoxide production)[26]; 3) depression, stress, and loneliness[50]; 4) oral hygiene; and 5) tobacco consumption. There is much to learn from these studies that might be applied to the study of peri-implantitis and its association with factors influencing the presence of AgP. Furthermore, the SR, success rate, and implant prognosis in those cases remain to be determined. Accordingly, large-scale case-control clinical trials should be conducted.

CONCLUSIONS

The dearth of scientific evidence in this matter does not allow for drawing clear conclusions. However, within the limitations exhibited, it can be concluded that implant placement in patients with a history of GAgP might be considered a viable option to restore oral function with survival outcomes similar to those found in both HPs and those with CP. Nonetheless, when the failure rate event was examined, a risk ratio of 4.0 was found compared with HPs and 3.97 when compared with patients with a history of CP. Therefore, because of the number of unmodifiable conditions that might play a determinant role, both in AgP and peri-implantitis processes, a comprehensive implant maintenance program to identify peri-implant bone loss early is highly encouraged, specifically in patients with a history of generalized aggressive periodontal disease. Moreover, larger and longer follow-up studies and more standardized protocols are needed to validate the current findings.

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