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ORIGINAL ARTICLE

Effect of desquamative gingivitis on periodontal status: a pilot study

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OBJECTIVE: Desquamative gingivitis (DG) represents the gingival manifestation associated with several mucocutaneous disorders and systemic conditions. Little is known of whether or not DG could influence the onset or progression of plaque-related periodontitis. In this study, the potential impact of DG on plaque-related attachment loss and pocket formation has been evaluated.

METHODS: A cross-sectional evaluation of 12 patients with DG [eight oral lichen planus (OLP), four mucous membrane pemphigoid (MMP)], never treated for DG lesions or plaque-related periodontitis, was carried out. Probing depth (PD), clinical attachment loss (CAL), full-mouth plaque (FMPS), and bleeding (FMBS) scores were evaluated at six sites per tooth. Clinical parameters of sites with DG lesions were compared with that of DG unaffected sites.

RESULTS: Median PD and CAL, as well as FMPS and FMBS, were not significantly different ($P > 0.05$ Mann–Whitney test) for both OLP and MMP patients. However, a negative association between DG lesions and PD < 4 mm (OLP: OR = 0.26; MMP: OR = 0.47), and a positive association with PD 4–6 mm (OLP: OR = 3.76; MMP: OR = 2.68) and with PD > 6 mm (only for OLP: OR = 3.83) were found to be significant.

CONCLUSIONS: The potential interference between DG lesions and periodontitis needs further prospective investigation; nonetheless, a higher level of attention might be prudent.

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Keywords: desquamative gingivitis; periodontal status; oral lichen planus; mucous membrane pemphigoid

Introduction

The presence of epithelial desquamation, erythema, and erosive and/or vesiculobullous lesions on gingiva characterizes the so-called 'desquamative gingivitis' (DG). This is neither a specific disorder, nor does it recognize a single etiopathogenesis, but represents the gingival manifestation associated with several mucocutaneous disorders and systemic conditions. Oral and systemic implications of disorders causing DG have been highlighted together with the need for accurate diagnosis and adequate management (Lo Russo *et al*, 2008). Nonetheless, there is only little systematic information about the impact and the potential influence of DG on the onset and/or progression of plaque-related periodontitis.

It has been suggested that DG lesions may indirectly increase the long-term risk for plaque-induced periodontal disease via plaque accumulation when symptoms associated with such lesions impede proper oral hygiene (Lo Russo *et al*, 2008). However, evidence is lacking; in fact, in a few studies addressing this issue in oral lichen planus (OLP) (Ramon-Fluixa *et al*, 1999) and mucous membrane pemphigoid (MMP) (Tricamo *et al*, 2006), the main causes of DG (Leao *et al*, 2008; Lo Russo *et al*, 2009), no significant differences were found between cases and matched control groups. On the other hand, it has been recently reported that periodontal status is worse in patients affected by pemphigus vulgaris (PV) (Akman *et al*, 2008), another disorder associated with DG lesions.

In this study, we have examined a series of patients affected by DG to evaluate the potential impact of DG lesions on plaque-related periodontal attachement loss and pocket formation; in particular, clinical parameters relevant to plaque-induced periodontitis were analyzed and results from sites where DG lesions were present were compared, in the same patient, with that from sites not affected by DG.

Patients and methods

Patients selection

Among patients referred in the last 2 years (2007–2008) to the Oral Medicine Section of the Department of Oral Sciences of Palermo University for the diagnosis and the management of desquamative gingival lesions, a group

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of 12 cases was selected. The selection was made by fulfilling the following criterion: (i) presence of erythematous/erosive and vesiculo-bullous lesions on gingiva consistent with the clinical picture of DG. Exclusion criteria were: (i) history of previous and/or current treatment for desquamative gingival lesions; (ii) inclusion in previous and/or current oral hygiene maintenance programs; (iii) previous and/or current treatments for plaque-related periodontal disease; and (iv) pregnancy.

A total of 8 (66.6%) patients were diagnosed as affected by OLP, while in the remaining four cases (33.3%), the diagnosis of MMP was made using a standardized diagnostic workflow (Lo Russo *et al*, 2008).

All patients provided informed consent to participate in the study.

Periodontal examination

Periodontal examination was conducted by the same examiner, specifically trained in periodontology, throughout the study and for all patients, using a dental chair and with adequate light conditions; during the examination, no patient had received treatment for the gingival disease and/or plaque-related periodontal disease. Measurements included probing depth (PD), clinical attachment loss (CAL), full-mouth plaque score (FMPS: percentage of sites with plaque on the total number of probed sites), and full-mouth bleeding score (FMBS: percentage of sites with bleeding on probing on the total number of probed sites). The number of missing teeth for each patient was determined by subtracting the number of teeth present from 32.

All periodontal measurements were performed at six sites per tooth; all teeth in all quadrants were carefully examined. All measurements were performed with a periodontal probe (PCPUNC15: Hu-Friedy®, Chicago, IL, USA) and the readings were recorded to the nearest 1 mm. PD was measured from the gingival margin to the base of the probeable pocket. The presence or absence of plaque and bleeding on probing was simultaneously recorded for each subject. CAL represented the distance between the cementoenamel junction (CEJ) and the base of the probeable pocket; the CEJ was determined by the anatomic CEJ or the most apical extent of a restoration margin. CAL was estimated by adding PD measurement and the location of gingival margin in relation with CEJ and/or the restoration margin.

A standardized digital sheet was used for systematic recording of the above-mentioned periodontal parameters as well as the presence and the exact location (site by site) of DG lesions.

During clinical history collection, particular attention was paid to ascertain the presence of the main recognized risk factors for periodontitis (i.e. smoking and poorly controlled diabetes mellitus).

Statistical analysis

For each patient, periodontal data of probed sites were stratified according to the presence or absence of desquamative gingival lesions; mean values of the individual measurements of affected and unaffected sites were calculated and regarded as representative values for that case. Probed sites were also classified into three different categories of PD (<4 mm, 4–6 mm, >6 mm) and CAL (<3 mm, 3–4 mm, ≥ 5 mm). Statistical analyses were performed with the GRAPHPAD PRISM software (version 5). Quantitative measurements (PD, CAL, FMPS, FMBS) were not normally distributed (Kolmogorov–Smirnov test). Therefore, these data were described by median and quartile values (inter-quartile ranges) in tables.

For each specific diagnosis, differences between sites where DG lesions were present and sites without DG were evaluated using the Mann–Whitney test. In addition, the prevalence of different categories of CAL and PD was described in contingency tables and analyzed with Fisher's exact test and odds ratios (OR), including 95% confidence intervals (CI).

All statistical tests were performed with a 5% level of significance.

Results

Ten patients (Table 1) were women and two were men with a mean age of 59.66 years (range 40–71 years). No patient smoked; only one reported to have quit cigarette smoking 5 years earlier.

Seven patients were affected by hypertension; it was associated with hypercholesterolemia and asthma in three and two cases, respectively. One patient showed serum positivity for Hepatitis C virus antibodies, but had no liver dysfunction. None of the drugs used for the above-mentioned disorders was deemed responsible for potential drug-associated oral lesions.

Four patients (3 OLP and 1 MMP) had no symptoms, two OLP patients referred severe oral pain, while the remaining six patients (3 OLP and 3 MMP) had only mild oral discomfort. Based on clinical history, the beginning of symptoms occurred, on average, 14.5 months (range 6–19 months) before inclusion in this study, and all but one case had an intermittent course.

On average, patients had 65.2 sites (range 6–162 sites) affected by DG, which corresponded to a mean of 47% (range 9.1–100%) of the total sites. In fact, patients had a mean of 8.5 missing teeth (range 4–16), so probeable sites ranged between 92 and 168. On the other hand, in four patients (2 OLP and 2 MMP), extensive DG lesions involved all teeth; thus, in the remaining eight patients, a mean of 113.75 sites without DG were probed (range 93–150). Periodontal data analysis for patients affected by OLP and MMP is detailed in Tables 2 and 3, respectively. As regards OLP, median PD was 2.18 mm and 2.08 mm in DG affected and unaffected sites, respectively; median CAL was 3.1 mm in DG sites and 2.86 mm in unaffected sites. Median PD in MMP was 2.33 mm and 2.45 mm in DG affected and unaffected sites; median CAL in MMP was 3.88 mm in DG sites and 3.78 mm in unaffected sites. Median values for PD and CAL were not significantly different (Mann–Whitney test; see Tables 2 and 3 for details) for both OLP and MMP patients. There were no significant differences

Table 1 Demographic features of patients, diagnosis, and extent of DG lesions

Case	Age (years)	Gender	Diagnosis	Missing teeth	No of probed sites	Sites with DG		Sites without DG	
						n	%	n	%
1	69	F	OLP	11	126	33	26.2	93	73.8
2	40	F	MMP	5	162	162	100	0	0
3	73	M	MMP	6	156	56	35.9	100	64.1
4	54	F	OLP	5	162	60	37	102	63
5	55	F	OLP	4	168	24	14.3	144	85.7
6	71	F	OLP	16	96	96	100	0	0
7	67	F	OLP	14	108	6	5.57	102	94.3
8	54	F	OLP	10	132	12	9.1	120	90.9
9	59	F	MMP	7	150	150	100	0	0
10	59	F	MMP	10	132	33	25	99	75
11	48	M	OLP	4	168	15	10.7	153	89.3
12	67	F	OLP	10	132	132	100	0	0

DG, desquamative gingivitis; OLP, oral lichen planus; MMP, mucous membrane pemphigoid.

Table 2 Clinical parameters of OLP patients in DG affected and unaffected sites

				Percentiles			
		Min	Max	25	50 (median)	75	P-value* (Mann–Whitney test)
PD (mm)	DG sites	1.45	3.61	1.94	2.18	2.74	0.754
	NO DG sites	1.94	2.22	2	2.08	2.22	
CAL (mm)	DG sites	1.89	5.5	2.3	3.1	4.8	0.851
	NO DG sites	2.14	6.19	2.24	2.86	4.3	
FMPS (% sites)	DG sites	52	100	83	94	100	0.601
	NO DG sites	66	100	69.75	89	97.75	
FMBS (% sites)	DG sites	24	100	62.75	88.5	97.5	0.795
	NO DG sites	45	100	56.25	86	94.75	
PD < 4 mm (% sites)	DG sites	16.67	100	64.58	85.99	94.6	0.106
	NO DG sites	88.24	98.92	91.44	95.06	97.13	
PD 4–6 mm (% sites)	DG sites	0	66.67	1.04	9.09	33.02	0.331
	NO DG sites	1.08	7.84	2.87	5.94	7.58	
PD > 6 mm (% sites)	DG sites	0	16.67	0	1.04	3.06	0.328
	NO DG sites	0	3.92	0	0	0.98	
CAL < 3 mm (% sites)	DG sites	0	60.61	5.42	21.36	55.95	0.949
	NO DG sites	0.98	65.36	6.49	26.47	54.25	
CAL 3–4 mm (% sites)	DG sites	16.67	83.33	33.14	43.34	62.76	0.949
	NO DG sites	28.43	59.8	29.17	49.35	58.08	
CAL ≥ 5 mm (% sites)	DG sites	0	83.33	0	10.23	51.56	0.559
	NO DG sites	2.15	70.59	4.46	17.94	43.28	

*Significance level: $P < 0.05$.

also in median plaque (FMPS) and bleeding score (FMBS). Tables 2 and 3 also represent the results for median values of percentages of sites falling within different categories of PD (<4 mm, 4–6 mm, >6 mm) and CAL (<3 mm, 3–4 mm, ≥5 mm): there were no significant differences in median percentage of sites with the above-mentioned PD and CAL categories in both OLP and MMP patients.

The prevalence of sites falling within different categories of PD and CAL, stratified according to DG presence, is shown, for OLP, in Table 4 and, for MMP, in Table 5. Comparing DG affected sites with unaffected sites, the following associations were found to be significant: a negative association between DG lesions and PD <4 mm, with an Odds Ratio (OR) of 0.26 (CI 95%: 0.17–0.38; $P < 0.0001$) and 0.47 (CI 95%: 0.27–0.79; $P = 0.004$) for OLP and MMP, respectively; a positive association between DG lesions and PD ranging between 4 and 6 mm, with an OR of 3.76 (CI 95%: 2.47–5.73; $P < 0.0001$) and 2.68 (CI 95%: 1.49–4.81; $P = 0.0005$) for OLP and MMP, respectively. Only for OLP lesions, a positive significant association was found with PD >6 mm (OR: 3.83; CI 95%: 1.14–12.8; $P = 0.0291$).

Discussion

From a theoretic point of view, disorders causing DG may have potential harmful outcomes on the development and progression of plaque-related periodontal

Table 3 Clinical parameters of MMP patients in DG affected and unaffected sites

				Percentiles			
		Min	*Max*	*25*	*50 (median)*	*75*	P-*value* (Mann–Whitney test)*
PD (mm)	DG sites	2.02	2.57	2.08	2.33	2.52	0.533
	NO DG sites	2.45	2.46	2.45	2.45	2.46	
CAL (mm)	DG sites	3.82	4.15	3.83	3.88	4.08	0.638
	NO DG sites	3.67	3.9	3.67	3.78	3.9	
FMPS (% sites)	DG sites	95	100	96.25	100	100	0.218
	NO DG sites	85	97	85	91	97	
FMBS (% sites)	DG sites	48	100	55.25	88.5	100	0.814
	NO DG sites	78	96	78	87	96	
PD < 4 mm (% sites)	DG sites	26.79	91.36	41.31	87.76	91.19	0.533
	NO DG sites	88	92.93	88	90.47	92.93	
PD 4–6 mm (% sites)	DG sites	8.02	71.43	8.34	12.24	57.36	0.133
	NO DG sites	7.07	8	7.07	7.53	8	
PD > 6 mm (% sites)	DG sites	0	1.79	0	0.31	1.49	0.805
	NO DG sites	0	4	0	2	4	
CAL < 3 mm (% sites)	DG sites	5.36	11.33	5.53	6.42	10.2	0.266
	NO DG sites	11	15.15	11	13.08	15.15	
CAL 3–4 mm (% sites)	DG sites	52.67	82.14	55.09	64.51	78.27	0.800
	NO DG sites	60	64.65	60	62.33	64.65	
CAL ≥ 5 mm (% sites)	DG sites	12.5	36	16.19	29.07	34.72	0.800
	NO DG sites	20.2	29	20.2	24.6	29	

*Significance level: $P < 0.05$.

Table 4 Prevalence of sites falling within different categories of PD and CAL in OLP patients; stratification has been made according to DG presence

			Pairwise comparison			P-*value**
		Number	*%*	*Odds ratio*	*95% CI*	*(Fisher's exact test)*
PD < 4 mm	DG sites	304	31.1	0.26	0.17–0.38	**<0.0001**
	NO DG sites	672	68.8			
PD 4–6 mm	DG sites	66	63.4	3.76	2.47–5.73	**<0.0001**
	NO DG sites	38	36.5			
PD > 6 mm	DG sites	8	66.6	3.83	1.14–12.8	**0.0291**
	NO DG sites	4	33.3			
CAL < 3 mm	DG sites	129	36.3	1.21	1.12–1.91	0.1687
	NO DG sites	226	63.6			
CAL 3–4 mm	DG sites	161	33.3	0.99	0.77–1.28	1.00
	NO DG sites	322	66.6			
CAL ≥ 5 mm	DG sites	68	29	0.77	0.56–1.06	0.1174
	NO DG sites	166	71			

CI, Confidence Interval.
*Significance level: $P < 0.05$.
Statistically significant values are given in bold.

disease. These potential injuries may be related to both direct and indirect relationships. In fact, DG lesions are typically chronic and often associated to a wide range of oral symptoms: when symptoms are present, the efficacy of daily oral hygiene procedures may be impaired and the consequent plaque accumulation may enhance the long-term risk for periodontal disease. In the instance of such an indirect effect (plaque accumulation), it seems reasonable to speculate that the distribution of the potential periodontal breakdown may be unrelated to the specific location of DG lesions.

On the other hand, a direct effect of DG lesions on periodontitis may also be plausible based on the possible shared pathogenetic mechanisms/mediators; in fact, the pathogenesis of plaque-related periodontal disease involves a local inflammatory reaction and the activation of the immune system stimulated by bacterial factors (Kornman, 2008). The elicited pro-inflammatory molecules and the activated cytokine networks play an essential role in this process, with interleukin-1 and tumor necrosis factor-alpha (TNF-α) being key molecules (Cochran, 2008). Now, immune-inflammatory mechanisms are also critical for the pathogenesis of most of DG-associated disorders (Lo Russo *et al*, 2008), which often involves common molecules/cytokine networks [e.g. TNF-α for OLP (Sugerman *et al*, 2002; Sugermann *et al*, 1996; Khan *et al*, 2003)]. Thus, it is possible to speculate that some interference could exist

Table 5 Prevalence of sites falling within different categories of PD and CAL in MMP patients; stratification has been made according to DG presence

		Pairwise comparison				
		Number	*%*	*Odds ratio*	*95% CI*	P-*value** *(Fisher's exact test)*
PD < 4 mm	DG sites	327	64.4	0.47	0.27–0.79	**0.004**
	NO DG sites	180	35.5			
PD 4–6 mm	DG sites	72	82.7	2.68	1.49–4.81	**0.0005**
	NO DG sites	15	17.3			
PD > 6 mm	DG sites	2	33.3	0.24	0.04–1.34	0.0974
	NO DG sites	4	66.6			
CAL < 3 mm	DG sites	33	55.9	0.60	0.34–1.02	0.0797
	NO DG sites	26	44.1			
CAL 3–4 mm	DG sites	248	66.6	0.98	0.69–1.39	0.929
	NO DG sites	124	33.3			
CAL ≥ 5 mm	DG sites	120	71	1.30	0.88–1.92	0.178
	NO DG sites	49	29			

CI, Confidence Interval.
*Significance level: $P < 0.05$.
Statistically significant values are given in bold.

and that the potential effect arising from these shared mechanisms/mediators should be strictly localized to sites where DG lesions are present.

The analysis of evidence available in the literature provides very few information, as periodontal status of patients with DG has received very limited attention. Ramon-Fluixa *et al* (Ramon-Fluixa *et al*, 1999) observed that no significant differences were present between an OLP group of patients and a control group with regard to different periodontal indices (Silness and Löe plaque index, the simplified calculus index, and the loss of attachment component of the Ramfjord periodontal disease index), even if patients with gingival involvement exhibited significantly higher plaque and calculus indices. Tricamo *et al* (Tricamo *et al*, 2006) showed that patients with MMP exhibit higher gingival index than controls, suggesting that more gingival inflammation was present; however, this conclusion should be regarded with caution as index evaluation may have been biased by the morphology of MMP lesions. Recently, it has been reported that patients affected by PV, another disorder associated with DG, have worse periodontal status (Akman *et al*, 2008) than healthy matched controls; however, it should be underlined that these results have been obtained by evaluation and comparison of Community Periodontal Index of Treatment Needs (CPITN). The partial periodontal data recording on which CPITN is based may significantly alter the precise definition of periodontal status (Baelum *et al*, 1993a,b); moreover, CPITN scores are not adequately correlated with attachment loss (Baelum *et al*, 1995).

Other potential biases may affect available studies: e.g. gingival lesions were not present in all cases, and some cases were already receiving some treatment at the time of evaluation (Tricamo *et al*, 2006; Akman *et al*, 2008). In addition, in the available reports, the matching between cases and controls is limited to age, gender, and smoking habits; however, this may be not adequate as the complexity of factors (genetic and epigenetic) that may influence the biologic phenotype contributing to the pathogenesis and clinical expression of the periodontal disease (Offenbacher *et al*, 2008) can make obtaining an adequate matching very difficult, at least based on the current body of knowledge.

Thus, in our study we have compared, in the same patient, sites where DG lesions were present with unaffected sites; this approach has the advantage of focusing on DG and using as controls intra-oral sites of the same patient, which make it possible to overcome difficulties in matching cases and controls because of still unknown and/or uncontrollable determinants of the individual susceptibility to periodontitis. This seems much suitable to provide insights with regard to the influence of DG immunopathogenic mechanisms upon plaque-related periodontal disease. Furthermore, in this study, periodontal measurements and indices relevant to plaque-induced periodontitis have been recorded for all teeth, at six sites per tooth. The full-mouth evaluation is more adequate to provide a precise definition of periodontal attachment status, which is mandatory for a reliable comparison.

Our results (Tables 2 and 3) indicate that in sites where DG lesions are present, on average, PD and CAL, as well as FMPS and FMBS, are not significantly different from sites where DG lesions are absent. This seems to rule out a possible interference of DG disorders on the formation of pockets and attachment loss. Nonetheless, it should be borne in mind that a suitable period of time is required for any potential effect to take place; under this point of view, we have no data regarding the duration of DG lesions: we only know that symptoms began, on average, 14.5 months (range 6–19 months) earlier, but symptoms are not always present in DG-associated disorders, as in four of our cases, and may not be synchronous with the disease onset. In addition, the extent of DG lesions may change over time (Lo Russo *et al*, 2008), thus, it is not possible to estimate the disease duration and whether or not it is sufficient to produce clinically measurable modifications of periodontal parameters.

On the other hand, the analysis of our data (Tables 4 and 5) shows that in DG affected sites, there is a significant negative association with PD < 4 mm and a positive association with PD ranging between 4 and 6 mm, for both OLP and MMP; a positive association was also found for PD > 6 mm, but only in OLP patients. This seems to suggest that the tendency to a deeper probing depth may exist in sites where DG lesions are present. Taken together, these observations do not allow drawing any robust conclusion regarding a potential interference between DG lesions and the plaque-related periodontal damage. Long-term prospective studies are required for such an important issue; but based on these preliminary data, maybe a higher level of attention in patients with DG lesions seems prudent.

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Conflict of interest

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Author contributions

Lo Russo and Campisi designed the study and organized patients recruitment; Guiglia and Pizzo examined patients and collected data. Fierro and Ciavarella revised the literature and participated in data analysis and paper drafting; Lo Muzio, Campisi and Lo Russo analyzed data, drafted and edited the paper.

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