Localized juvenile spongiotic gingival hyperplasia: Microscopic variations and proposed change to nomenclature

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Objectives. Localized juvenile spongiotic gingival hyperplasia (LJSGH) is a recently described entity with distinct manifestations. Herein we report a comprehensive histopathologic study of 21 lesions and a literature review. Additionally, we propose a new term that we consider more appropriate.

Study Design. LJSGH cases were retrieved and their clinicopathologic characteristics were assessed. A review of all pertinent literature was also conducted.

Results. Eighteen patients with LJSGH (21 biopsied lesions) were identified. Microscopically, surface morphology was classified into exophytic/papillary, flat, and micropapillary (8, 7, and 6 lesions, respectively). Cases with parakeratinization (n = 9), no prominent spongiosis (n = 5), or epithelial atrophy (n = 4) were recorded. Increased vascularity, mixed inflammation with exocytosis, and cytokeratin-19 positivity were uniformly observed. Less frequent findings included pseudoepitheliomatous hyperplasia (n = 8), bacterial colonies (n = 5), acantholysis (n = 3), and dystrophic calcifications (n = 2). The literature review disclosed 201 patients with a mean age of 14.8 years (range, 3-72; 13.6% affecting adults), similar sex distribution (103:98, female:male), and predominance of the anterior maxilla (≈ 80%). Eighteen cases were multifocal (≈ 10%).

Conclusions. Our data suggest that the terminology could be modified, because LJSGH may be multifocal, affect older individuals, or exhibit epithelial atrophy, and the entity's odontogenic origin (as highlighted by the histopathologic and immunohistochemical findings) needs to be emphasized. (Oral Surg Oral Med Oral Pathol Oral Radiol 2021;131:329−338)

Localized juvenile spongiotic gingival hyperplasia (LJSGH) is a recently described entity manifesting as non-plaque-induced gingival overgrowth with distinct clinical, microscopic, and immunohistochemical features. Darling et al. were the first authors to report reddish gingival enlargements affecting juveniles and displaying microscopic features analogous to spongiotic cutaneous diseases, for which they proposed the term "juvenile spongiotic gingivitis."[1] Subsequently, a clinicopathologic study with a significant number of patients was published characterizing these lesions as LJSGH, which is currently the most widely used term.[2] However, several authors have questioned the current terminology on the basis that it does not accurately describe the clinical phenotype of this entity.[3-5]

Regarding the demographic features of LJSGH, both sexes may be affected, with certain studies showing significant predominance of females,[2] others recording a male predilection,[6,7] and several case series suggesting a similar distribution between the two sexes.[1,5] However, no review of all reported cases has been published to date to disclose which sex is primarily affected. Similar variations have been observed regarding the age of these patients, with several publications recording a mean age between 11 and 13 years,[1,2,6,8] whereas more recent studies report an average age of 18 years or higher.[5,7]

LJSGH has been described clinically as a solitary elevated lesion of red color and smooth, papillary, granular, or pebbly surface affecting the attached and marginal gingiva.[1,2] The upper labial gingiva is the predominant site of involvement in all published clinicopathologic studies.[1,2,5-9] Additionally, even though this entity is usually characterized by solitary lesions, occasional multifocal cases may be encountered.[1,4]

The aforementioned clinical features of LJSGH are usually characteristic; however, diagnostic dilemmas with other known entities involving the gingiva occasionally arise, including plaque-related diseases (e.g., puberty gingivitis), benign reactive or neoplastic overgrowths (such as pyogenic granuloma or hemangioma), human papillomavirus−associated lesions (e.g., papil-

Statement of Clinical Relevance

Spongiotic odontogenic gingivitis could replace the currently used term *localized juvenile spongiotic gingival hyperplasia* because this entity may be multifocal, affect older individuals, or microscopically display atrophic surface epithelium and the entity's odontogenic origin might need to be emphasized.

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loma), or even gingival manifestations of hematologic malignancies (e.g., leukemia).[1,2,6] Establishing the correct diagnosis is essential to ensure that appropriate treatment is followed, which mainly includes excision with proper follow-up,[1,2,5,6] in contrast to reactive or plaque-related entities that also require removal of the causal factor (e.g., periodontal treatment).

Diagnosis of LJSGH should be supported by biopsy and histopathologic examination. The reported microscopic findings exhibit similarities with those observed in the junctional epithelium (JE) featuring lack of keratinization and presence of spongiosis, exocytosis, mixed inflammatory infiltrate, and an occasional papillary surface.[1,2,6] However, to date, no microscopic study has been conducted to reveal the relative frequency of these histopathologic findings or the occurrence of less common features. Additionally, the microscopic variations of LJSGH (e.g., with respect to the morphology of the surface epithelium) have not been emphasized.

Herein we present a clinicopathologic study of LJSGH with emphasis on the microscopic findings and classification of these lesions. Additionally, we review all of the reported cases of the English-language literature in order to collectively present the demographic and clinical features of this entity. Following analysis of the data retrieved from the histopathologic study and literature review, we provide our insights on the nomenclature and propose a term that could appropriately highlight the entity's phenotype and origin.

MATERIALS AND METHODS

Clinicopathologic study of LJSGH

Cases with a diagnosis of LJSGH between the years 2008 and 2020 were retrieved from the histopathologic archives of the Department of Oral Medicine and Pathology, School of Dentistry, National and Kapodistrian University of Athens, Greece. The demographic data (age, sex), provisional clinical diagnosis, and clinical features (number of lesions per patient, site of involvement, size) were documented.

Hematoxylin and eosin−stained slides of all lesions were reevaluated and the diagnosis was confirmed. A histopathologic study was performed and the following features were evaluated (compared with the adjacent normal epithelium, if present, or the epithelium that is normally expected in this anatomic location): (1) epithelial surface morphology (exophytic/papillary vs flat vs micropapillary), (2) spinous layer thickness (normal, acanthosis vs atrophy), (3) keratinization (absence vs focal parakeratin formation), (4) prominent spongiosis (presence or not), (5) exocytosis (presence or not), (6) microabscesses (presence or not), (7) inflammatory infiltrate (acute, chronic, or mixed); (8) vascularity (increased or not); (9) other features. Cytokeratin-19 (CK19) immunohistochemical examination was performed in all cases.

Systematic review of the literature

A literature search of all reported LJSGH cases was conducted. Cases without histopathologic confirmation of the diagnosis or with illustrated clinicopathologic features that were not considered sufficiently diagnostic of LJSGH were omitted. The demographic (age, sex) and clinical (number of lesions per patient, site of involvement and size) features, CK19 status, and outcome (i.e., whether recurrence occurred among excised lesions with at least 6 months of follow-up) were recorded. The reported clinical impressions were not included because a significant number of studies included cases initially diagnosed before the establishment of LJSGH as a recognized entity; hence, this lesion could not be included in the provisional diagnoses by the clinicians.

RESULTS

Clinicopathologic study of LJSGH cases

A total of 18 patients exhibiting clinical and histopathologic features of LJSGH were included (Table I). Among them, 3 patients have already been reported in the literature.[4,10] The patients' mean and median ages were 19.17 and 14 years, respectively; a range from 8 to 57 years was noted, including 6 adults (33%). Most patients were affected by solitary lesions, though 4 of them displayed multifocal LJSGH (in 3 of which it was proven upon biopsy from multiple sites). More specifically, a 12-year-old female patient exhibited 2 solitary LJSGHs (one mandibular and one maxillary) that were excised. Two other patients displayed diffuse multifocal lesions affecting the upper and lower jaw (one of which was previously published[4]) and biopsies were taken from 2 different sites in each patient. Finally, a fourth patient exhibited multiple bright red erythematous lesions affecting the anterior maxilla (areas of teeth 6, 7-8, 9-11) and biopsy was performed from the most representative area disclosing the diagnosis. Overall, among 21 biopsied lesions, the anterior maxillary gingiva was involved in 14 cases, 3 lesions extended to both the anterior and posterior maxillary gingiva, and the remaining 4 lesions involved the mandibular gingiva; in all cases, only vestibular (labial or buccal) gingiva were involved. The lesions exhibited a mean size of 0.7 cm and median of 0.6 cm and ranged from 0.3 to 1.8 cm. The most common clinical impressions were LJSGH and pyogenic granuloma (8 and 7 cases, respectively); granulomatous gingivitis and verruciform xanthoma were speculated in one patient each, and in one case no provisional diagnosis was submitted by the referring doctor.

Table I. Clinical and demographic features of the current series

Clinical features	*Value*
No. of patients*	18
Sex	
Female	10
Male	8
Age (17 cases with available data)	
Mean	19.17
Median	14
Range	8-57
Lesions per patient	
Single	14
Multifocal	4
Clinical impression	
LJSGH	8
Pyogenic granuloma	7
Granulomatous inflammation	1
Verruciform xanthoma	1
None	1
Number of biopsied lesions*	21
Site of involvement	
Anterior labial maxillary gingiva	14
Anterior labial−posterior buccal maxillary gingiva	3
Anterior labial mandibular gingiva	2
Anterior labial−posterior buccal mandibular gingiva	1
Posterior buccal mandibular gingiva	1
Size (cm)	
Mean	0.7
Median	0.6
Range	0.3-1.8

LJSGH, localized juvenile spongiotic gingival hyperplasia.
*Three of the cases (4 of the lesions) presented herein have previously been reported (Kalogirou et al.[10] and Siamantas et al.[4]).

Table II. Microscopic findings in biopsied cases of SGH

Histopathologic features[e]	*No. (n = 21)*
Subtype (surface)	
Exophytic (papillary)	8
Flat	7
Micropapillary	6
Spinous layer thickness	
Acanthosis	14
Atrophy	4
Normal	3
Keratinization	
Nonkeratinized epithelium	12
Focal parakeratin	9
Prominent spongiosis	
Yes	16
No	5
Exocytosis	
Present	21
Neutrophilic	7
Lymphocytic	6
Mixed	8
Microabscesses	
Yes	13
No	8
Inflammatory infiltrate	
Mixed acute and chronic inflammation	21
Mostly lymphocytic	13
Equal distribution	7
Mostly acute (mimicking abscess)	1
Vascularity	
Increased	21
Other features	
Pseudoepitheliomatous hyperplasia	8
Bacterial colonies	5
Acantholytic changes	3
Dystrophic calcifications	2
CK19 immunohistochemistry	
Positive in all epithelial layers	21

SGH, spongiotic gingival hyperplasia; *CK19*, cyokeratin-19.
[e]Three of the cases (4 of the lesions) presented herein have previously been reported (Kalogirou et al.[10] and Siamantas et al.[4]).

The results of the microscopic study of all cases are presented in Table II. First, the lesions were categorized based on their overall morphology as exophytic/papillary (8 cases; Figure 1A), flat (7 cases; Figure 1B), and micropapillary (6 cases). The latter variant displayed small papillary projections of the surface epithelium, although their overall arrangement was not exophytic (Figure 1C). The thickness of the spinous cell layer was also evaluated: 14 LJSGHs showed acanthosis (with or without elongated rete ridges; Figure 2A), in contrast to 4 lesions demonstrating epithelial atrophy (Figures 2B and 2C), and the remaining 4 cases displayed normal thickness of the spinous layer. Regarding keratinization, most cases (12) were nonkeratinized (Figure 2D), and 9 lesions showed focal areas of parakeratinized epithelium (Figure 2E). Spongiosis, a characteristic feature of LJSGH, was detected in the majority of cases (Figure 2D), though 5 lesions lacked prominent spongiotic changes (Figure 2F). In contrast, 3 lesions exhibited spongiosis in an increased intensity that led to acantholysis (Figure 2G), one of which displayed separation of the epithelium from the connective tissue and a tombstone appearance (Figure 2H). Exocytosis was also noted in every case, varying from neutrophilic (7 cases) to lymphocytic (6 cases) or mixed (8 cases), and microabscesses were detected in 13 lesions (Figure 2E). The presence of a highly vascular connective tissue with varying degrees of acute and chronic inflammation was also seen in all cases. Most lesions exhibited mainly chronic inflammatory infiltrate (13 cases; Figure 3A) or an almost equal distribution between lymphoplasmacytic cells and neutrophils (7 lesions). However, one LJSGH displayed edematous connective tissue with predominance of acute inflammation mimicking abscess (Figure 3B). Finally, other features that have not been reported to date were noticed, including pseudoepitheliomatous hyperplasia (8 lesions; Figure 3A), bacterial colonies (5 cases;

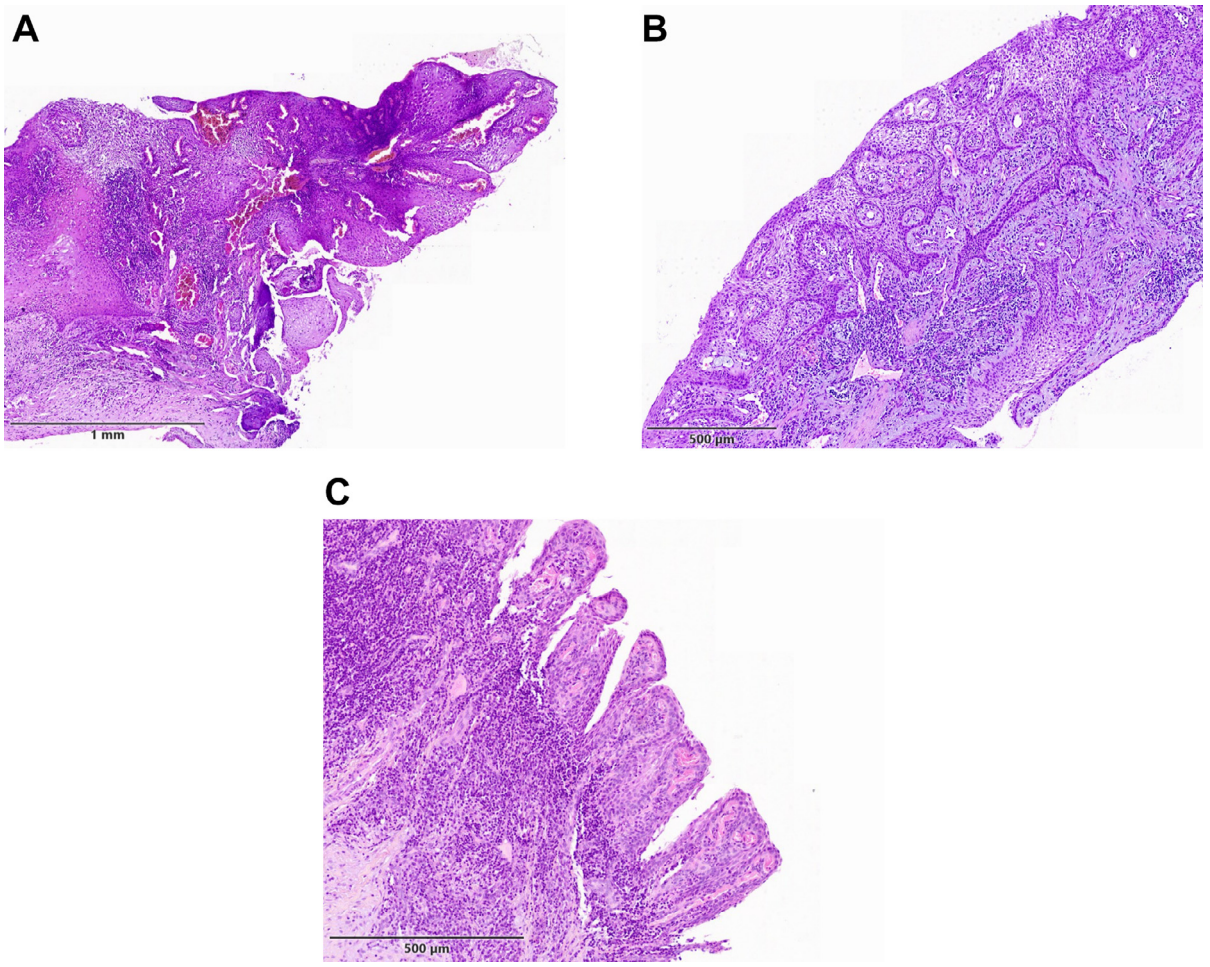

Fig. 1. Histopathologic features of LJSGH. Regarding the surface characteristics of the lesions, 3 different morphologic patterns were observed: **(A)** exophytic, showing a papillary configuration; **(B)** flat, exhibiting a smooth surface without irregularities; and **(C)** micropapillary, demonstrating small papillary projections of the surface epithelium. Hematoxylin and eosin stain, initial magnification 200 ×.

Figure 3C), and dystrophic calcifications (2 cases; Figure 3D).

Immunohistochemical analysis for CK19 was positive in all cases. Lesional epithelium showed cytoplasmic expression of this marker within all epithelial layers, in contrast to normal adjacent epithelium that expressed CK19 only within the basal cell area (Figure 2C), which is in agreement with previously reported studies.[1,8]

Review of the reported cases of LJSGH

A total of 15 previously published LJSGH studies were reviewed; 2 publications were excluded because of the presence of clinical and/or histopathologic features that were not convincingly consistent with LJSGH.[11,12] The total number of reported cases was 201, including our series of 15 additional patients (as well as 3 previously reported cases, as mentioned before; Table III).[1-10,13-17] An almost equal distribution between the 2 sexes was noted (103:98), with a mean age of 14.8 years (range, 3-72). The vast majority of patients were within the first 2 decades of life (≈ 90%); however, 13.6% were adults (18 years old or older). Among the 181 patients with documented information regarding number of lesions, 18 presented with multifocal LJSGH (9.9% of cases). CK19 immunohistochemical examination was performed in 98 of 209 biopsied lesions and was diffusely positive in the whole thickness of epithelium in all of them. The maxillary gingiva were most commonly affected compared to the mandibular gingiva (175:30); among 177 lesions with fully specified site of involvement, the maxillary labial gingiva was affected in almost 80% of cases. The documented size of LJSGH was similar to that presented in our study (0.66 cm; range, 0.3-1.8 cm). Finally, 16 total recurrences have been reported, though when taking into

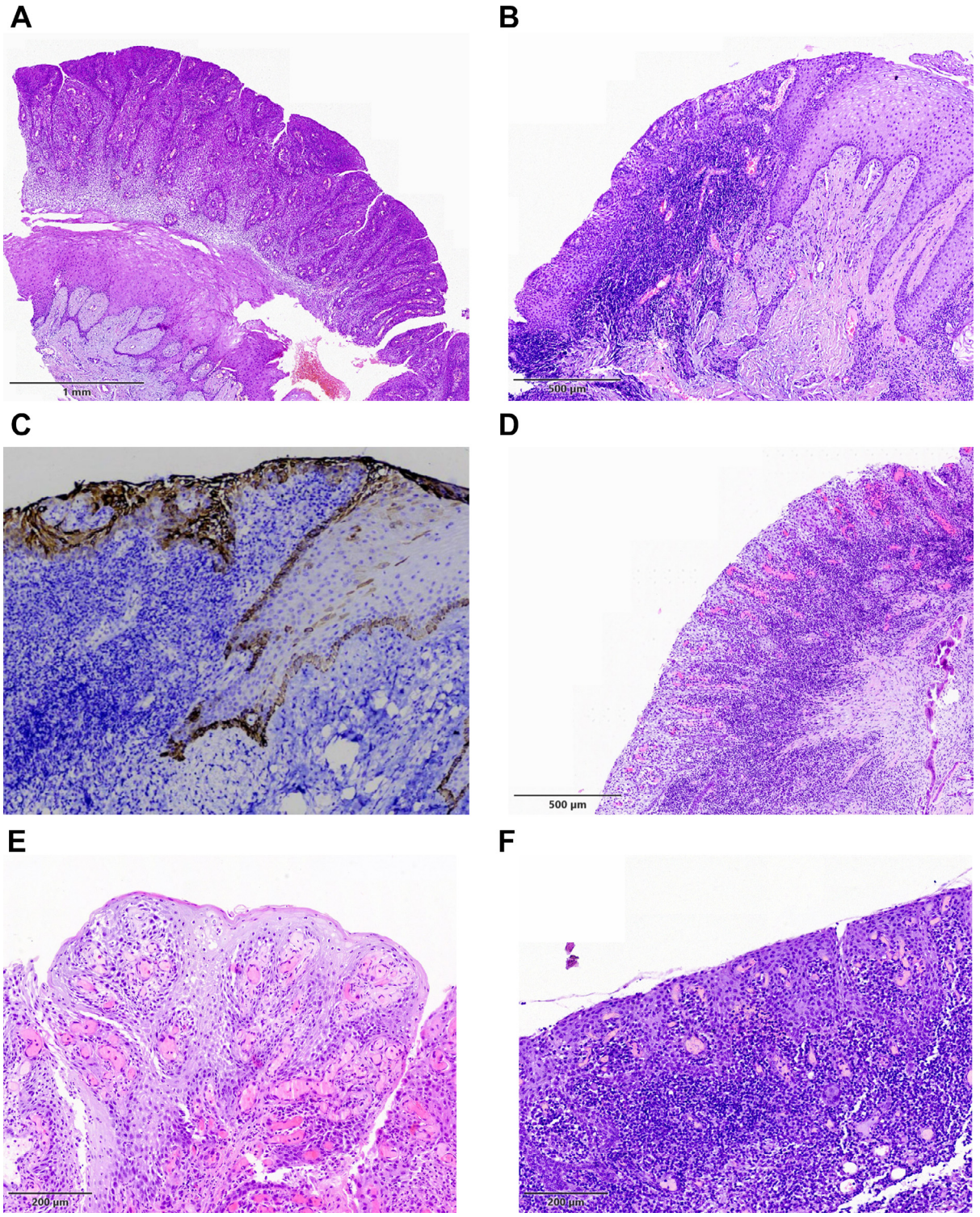

Fig. 2. Microscopic findings in the surface epithelium. **(A)** Micropapillary lesion showing marked acanthosis. **(B)** Flat LJSGH exhibiting epithelial atrophy. **(C)** Immunohistochemical examination for CK19 of the same lesion highlighting the transition from normal gingival epithelium (showing positivity in the basal cell layer) to atrophic epithelium of LJSGH (showing diffuse immunoreactivity). **(D)** Lesions may most commonly show a nonkeratinized surface epithelium with spongiosis. **(E)** Papillary LJSGH displaying focal parakeratinization. In addition, the presence of neutrophilic exocytosis with microabscesses may be observed. **(F)** Flat LJSGH lacking prominent spongiotic features. **(G)** Intense spongiotic changes may be observed, and one lesion **(H)** displayed a suprabasal separation of the epithelium from the underlying connective tissue, resulting in a tombstone appearance. **(A), (B), (D)-(H)** Hematoxylin and eosin stain, initial magnification 200 ×.

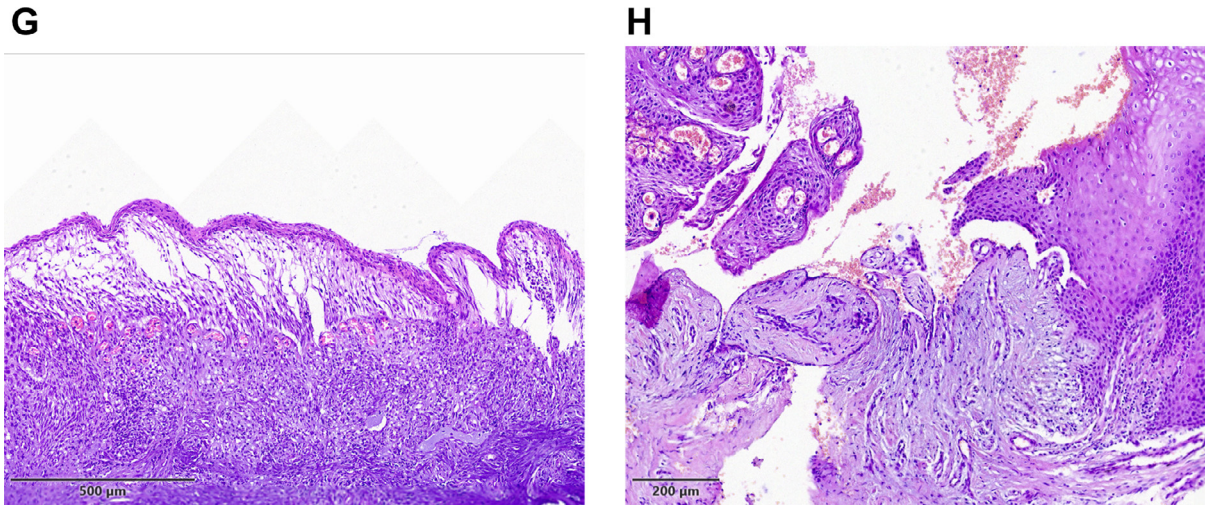

Fig. 2. Continued

consideration studies with excised lesions and a minimum follow-up of 6 months, 10 of 40 cases displayed recurrence (25%).

DISCUSSION

This is the first study to systematically evaluate and record the frequency of several histopathologic features of LJSGH. Previously, certain microscopic findings, such as spongiosis and nonkeratinized surface, have been considered as highly indicative of LJSGH.[1,2] Although prominent spongiosis and lack of keratinization were observed in the majority of our cases, too, a substantial number of lesions exhibited either absence of prominent spongiosis or focal parakeratin. The presence of parakeratinized epithelium has previously been described.[6] A hypothesis could be drawn that focal parakeratin formation or absence of spongiosis may be a manifestation of older lesions. Other known predominant features of LJSGH (exocytosis, inflammatory infiltrate, and increased vascularity) were present in all cases.

Less common findings that have not been reported to date were also observed. Pseudoepitheliomatous hyperplasia, a feature indicative of various pathologic lesions of the oral cavity,[18] was seen in almost half of the cases. Bacterial colonies were also occasionally encountered and may be attributed to the common presence of local microbial factor in this anatomic location. Finally, dystrophic calcifications exhibited mainly an intravascular localization and could be associated with the degenerative changes caused by the increased inflammation. This phenomenon could be mistaken for other developing gingival overgrowths that might display calcifications as well as share according clinical and demographic features, including peripheral ossifying fibroma or peripheral odontogenic fibroma. However, the main finding in these entities is a hyperplastic, cellular fibrous connective tissue, whereas marked inflammation with exocytosis and spongiosis of the overlying epithelium is rarely encountered.

Because LJSGH usually exhibits characteristic features (solitary reddish patches affecting the maxillary gingiva of juveniles and exhibiting nonkeratinized epithelium with spongiosis and exocytosis), histopathologic examination is usually sufficient for diagnosis. However, we recommend performing CK19 immunohistochemical examination to further support the diagnosis in cases with atypical clinical or microscopic features such as the ones that we previously described or in patients who do not match the demographic features of LJSGH, as was suggested by a recent study.[5]

Noteworthy was our microscopic observation that the overall surface morphology of the studied lesions fell into one of three major categories: exophytic/papillary, flat, and micropapillary. We also suggest that the aforementioned microscopic classification of LJSGH could correspond to their clinical appearance. Though a clinicopathologic correlation was not possible in all cases (because of the lack of detailed clinical information and/or clinical photos in some of the cases submitted as biopsies to our oral pathology laboratory), we were able to ascertain in a number of cases that microscopically flat lesions were clinically associated with slightly elevated bright red plaques (Figures 4A and 4B), papillary histopathologic morphology was a feature of more exophytic, occasionally pedunculated lesions with surface projections (Figures 4C, 4D, and 4E), and micropapillary configuration was clinically correlated with flat granular lesions (Figures 4F and 4G).

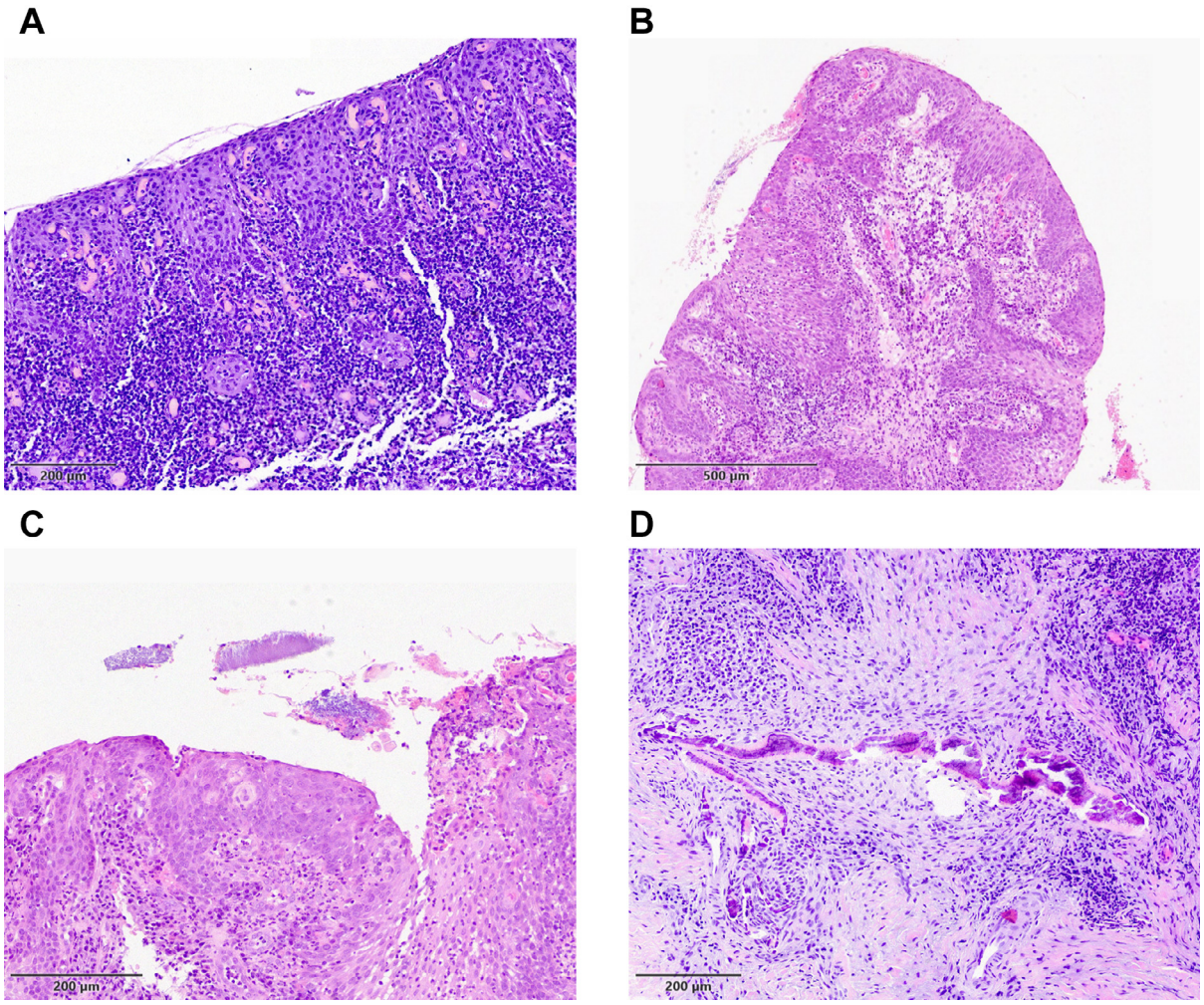

Fig. 3. Microscopic features of the connective tissue and other findings. **(A)** Flat LJSGH with pseudoepitheliomatous hyperplasia and a subepithelial dense, predominantly chronic, inflammatory infiltrate. **(B)** Edematous connective tissue with predominance of an acute inflammatory component mimicking abscess; prominent neutrophilic exocytosis is also evident. **(C)** Papillary LJSGH with bacterial colonies adjacent to the surface epithelium. **(D)** Amorphous basophilic calcified material consistent with dystrophic calcification. Hematoxylin and eosin stain, initial magnification 200 ×.

The data retrieved from this microscopic study in addition to a thorough review of all reported cases (with sufficient justification of the diagnosis) provide the opportunity to acquire broader knowledge about these lesions that could lead to a reappraisal of the current terminology. The reported clinical and demographic features suggest that the terms *localized* and *juvenile* could be omitted, as previously suggested.[5] The first suggestion is supported by the occurrence of multifocal LJSGH in approximately 10% of the cases and by the occasional presence of diffuse lesions.[1,14] A previous study has already highlighted the necessity of omission of the word localized, though their review included solitary lesions affecting the gingiva of multiple teeth in addition to multifocal cases.[4] Regarding the term juvenile, as previously reported, LJSGH may involve patients in their 50s or older and does not exclusively affect young patients.[5] In our review, adults were included in 13.6% of published cases; although this may be considered a relatively low percentage, it could be speculated that the use of the term juvenile may have led to underrecognition or underreporting of such lesions in adults. Based on most recent studies that included older patients, a higher prevalence in individuals of 18 years of age or higher was observed (10/28 in the study by Vargo and Bilodeau [35.7%][5] and 6/18 in our study [33.3%]).

The selection of the most appropriate term for these lesions could also be facilitated by a careful reappraisal of the observed microscopic findings.

Table III. Demographic and clinical and features of published cases of SGH

Demographic and clinical features	*No. (%)*
Publications[b]	16 (including the present study)
No. of patients	201
Gender	
Female	103 (51)
Male	98 (49)
Age (197 patients with available data)	
Mean	14.8
Range	3-72
Adults (≥18 years) (182 patients with available data)	25 (13.6)
Age distribution (187 patients with available data)	
≤10	62 (32.8)
11-20	109 (57.7)
21-30	4 (2.1)
31-40	4 (2.1)
41-50	3 (1.6)
51-60	3 (1.6)
≥61	4 (2.1)
Lesions per patient (181 patients with available data)	
Single	163 (90.1)
Multifocal	18 (9.9)
Number of biopsied lesions*	209
Involved jaw (202 lesions with available data)	
Maxilla	175 (85.4)
Mandible	30 (14.6)
Site of involvement (177 fully specified lesions)	
Maxillary labial gingiva	141 (79.78)
Mandibular labial gingiva	23 (13)
Anterior-posterior maxillary buccal gingiva	5 (2.8)
Posterior maxillary buccal gingiva	5 (2.3)
Anterior-posterior mandibular buccal gingiva	1 (0.6)
Posterior mandibular buccal gingiva	1 (0.6)
Posterior mandibular lingual gingiva	1 (0.6)
Size (cm) (65 lesions with available data)	
Mean	0.66
Range	0.3-1.8
CK19 immunohistochemistry (performed in 98 lesions)	
Positive in all the epithelial layers	98 (100)
Recurrences	
Total	16
Recurrences (studies with at least 6 months of follow-up; 40 cases)	10 (25)

SGH, spongiotic gingival hyperplasia; *CK19*, cyokeratin-19.
[b]Only studies with microscopic confirmation of diagnosis and illustrated clinicpathologic features that were considered diagnostic of LJSGH were included.

Though spongiosis is not very prominent in all cases, it is always seen in varying degrees, ranging from mild interepithelial vacuolization to intense changes causing acantholysis. Hence, the term *spongiotic* is considered acceptable. In contrast, *hyperplasia*, which describes an increase in the number of cells in a tissue, does not always characterize these lesions, some of which may histopathologically exhibit epithelial atrophy. Additionally, the marked inflammatory component that is present in every single case might be highlighted by the word *gingivitis* (i.e., inflammation of the gingiva), which was also used in the first term proposed by Darling et al.[1]

Another essential consideration for the establishment of a new appropriate term would be highlighting the entity's pathogenesis and origin. It has been convincingly argued that these overgrowths display an odontogenic origin as highlighted by the expression of cytokeratins that are implicated in physiologic or pathologic odontogenic epithelia,[8] in addition to the microscopic similarities with the JE and other odontogenic lesions (such as radicular cysts). More specifically, the histopathologic findings of our study in addition to the pattern of uniformly detected CK19 positivity (which has also been described by previous publications[1,8]) strengthen this hypothesis. However, the speculated origin from the JE has not been completely proven, because other remnants of odontogenic epithelium (epithelial rests of Malassez in the periodontal ligament or rests of Serres in the alveolar mucosa) may exhibit similar immunophenotypic features.[19-21] Additionally, the fact that these lesions occur mostly during young ages when these rests predominate[22] could support a hypothesis that LJSGH originates from epithelial rests that are left behind during odontogenesis and not the JE. Further studies investigating the expression of markers that differentiate the JE from other odontogenic epithelia (such as CK17[20]) could answer these equivocal questions.

In summary, this is a comprehensive microscopic study of LJSGH and review of all previously reported cases. Based on the collective data retrieved, we suggest that the terms localized, juvenile, and hyperplasia may be misleading, in agreement with other authors.[3-5] Additionally, we argue that the odontogenic origin and inflammatory component of the entity deserves more emphasis. Herein, we propose the term "spongiotic odontogenic gingivitis" or "spongiotic gingivitis with odontogenic metaplasia (SGOM)" as more appropriate to describe these lesions.

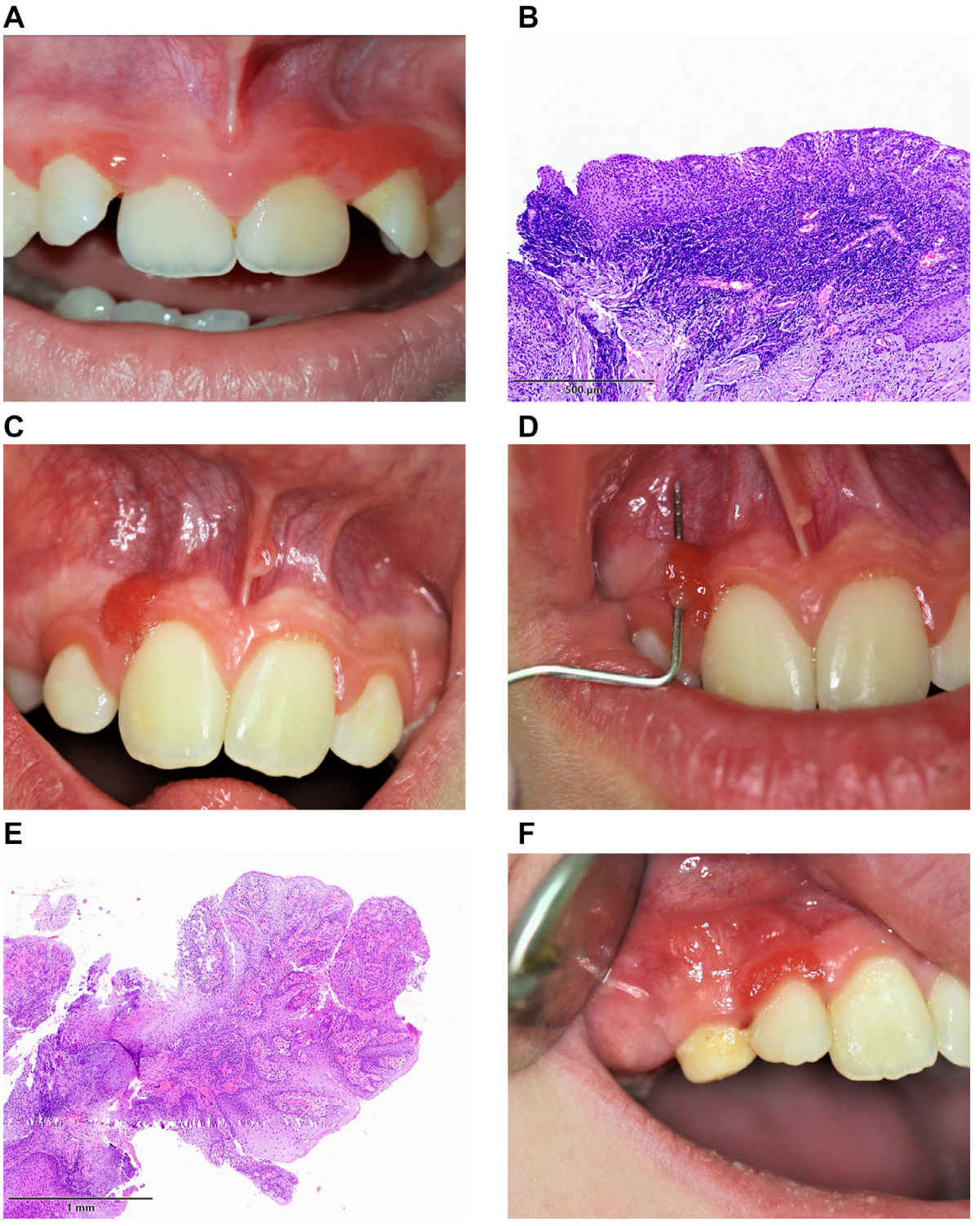

Fig. 4. Correlation between clinical and microscopic features of LJSGH and suggestion for classification into 3 subtypes. **(A), (B)** Flat variant: Patient with multifocal bright red and smooth-surfaced lesions exhibiting a flat surface microscopically. **(C)-(E)** Exophytic/papillary variant: Exophytic and pedunculated overgrowth showing microscopically a papillary morphology. **(F), (G)** Micropapillary variant: Granular, slightly elevated gingival enlargement with a micropapillary microscopic architecture. **(B), (E)**, and **(G)** Hematoxylin and eosin, initial magnification 200 ×.

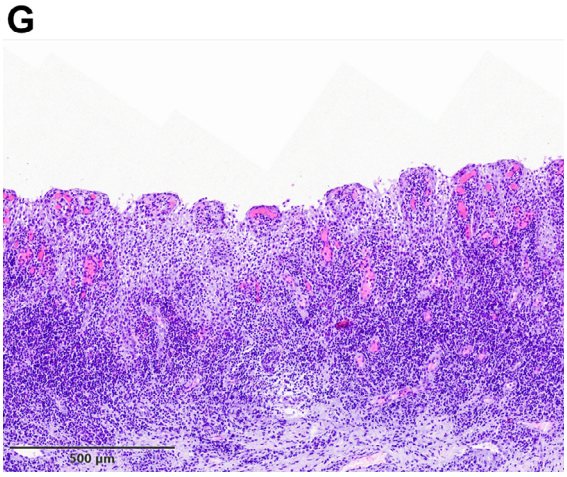

Fig. 4. Continued

CONCLUSIONS

Though LJSGH is an entity that has already been explicitly described, certain parameters, including the detailed description of its microscopic findings as well as the overall assessment of its clinical and demographic features, had not been previously reported. Based on our microscopic findings, a subclassification into 3 variants, namely, exophytic (papillary), flat, and micropapillary, may be useful. Additionally, the current nomenclature is questioned and, on the basis of critical assessment of the lesions' overall features, the new term "spongiotic odontogenic gingivitis" or "spongiotic gingivitis with odontogenic metaplasia (SGOM)" is proposed.

PRESENTATION

Part of this work was presented (poster presentation) at the Annual Meeting of the American Academy of Oral and Maxillofacial Pathology (Virtual Meeting), April 24-28, 2020.

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