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CASE REPORT

Clinical features and management of spongiotic gingival hyperplasia in Kuwait: a case series

Abdulhameed Alsarraf[1,2,3]*, Abdullatif Burahmah[1], Marwah Alshatti[4]

Abstract:

This study aims to present a series of cases of spongiotic gingival hyperplasia treated at the oral medicine and pathology service of Jahra Dental Specialty Centre, Kuwait Ministry of Health, with emphasis on its clinical features and management approaches. Patients diagnosed with SGH between January 2024 and June 2026 were retrospectively identified from clinical and histopathology records based on established clinical diagnostic criteria, with biopsy performed where consented. Six patients (five females) aged 5–16 years (mean 12.2 years) were identified, with lesions predominantly involving the anterior maxillary gingiva—solitary in four cases and multifocal in two. Common symptoms included mild tenderness and bleeding on brushing. Five cases were managed with clinical monitoring alone, showing no progression over 12–24 months; one case underwent excisional biopsy confirming the diagnosis, with no recurrence at 11 months. SGH is a rare, benign gingival condition affecting the anterior gingiva in children and adolescents, refractory to conventional periodontal treatment. Histopathological diagnosis guides appropriate management, while clinical monitoring may be a reasonable alternative in stable cases.

Keywords: Gingival hyperplasia; Biopsy; Oral medicine; Oral pathology.

INTRODUCTION

Juvenile spongiotic gingivitis is a benign gingival lesion that was initially reported by Darling et al.[1]. As additional cases were reported, most of them are characterized by localized gingival erythema accompanied by epithelial proliferation, rather than representing a purely inflammatory condition[2]. Furthermore, later studies proposed the term localized juvenile spongiotic gingival hyperplasia to better reflect the clinical and histopathological features of this entity[3,4]. This lesion has also been a subject of nomenclature debate, as it has been reported in patients of all ages. Initially, it was described predominantly in children and adolescents, hence the use of the term "juvenile"[3]. However, recent case series and reviews confirm occurrences in adults, supporting that the lesion is not strictly age-dependent thus changing the nomenclature of the condition to spongiotic gingival hyperplasia (SGH)[4–6].

Statement of Clinical Significance
Spongiotic gingival hyperplasia is a rare, benign condition mimicking more aggressive gingival lesions and resists conventional periodontal therapy. Recognizing its distinct clinical features helps clinicians avoid unnecessary aggressive surgery, supports histopathological diagnosis when needed, and confirms that clinical monitoring is a reasonable, effective option in stable cases.

SGH is considered an inflammatory hyperplasia with distinctive clinicopathologic features and unknown etiology[4]. Clinically, it presents as a well-demarcated erythematous area typically associated with a granular, velvety, or pebbly overgrowth.

The etiology of SGH remains uncertain and controversial. Its negligible association with plaque and calculus argues against a bacterial origin[6,7], and reviews have emphasized that SGH is not a plaque-induced condition,

[1]Oral Medicine Clinic, Jahra Dental Specialty Center, Ministry of Health – Al Jahra, Kuwait.[2]Kuwait Oil Company Hospital, Oral Medicine & Pathology Clinic, Dental Department – Ahmadi, Kuwait.
[3]Oral Diseases Research Academy – Kuwait.
[4]Dental Administration, Ministry of Health – Kuwait.
***Correspondence to:** E-mail: abdulhameed.alsarraf@hotmail.com
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further distinguishing it from common gingivitis[4,5]. The absence of sex hormone receptor activity and negative HPV testing suggest that neither hormonal influences nor viral infections are significant etiological factors[6]. Also, SGH has been linked to a possible odontogenic developmental origin, with some authors proposing that these lesions may represent an ectopic junctional epithelium (JE). This is based on both histological and immunohistochemical similarities to JE, which is particularly susceptible to inflammation from minimal irritants[3,8].

Recent reports also highlight the role of microenvironmental triggers, such as local trauma from tooth brushing, orthodontic brackets, or malpositioned teeth, which may act on a predisposed JE to initiate lesion development[9]. The recurrent or persistent nature of some lesions further supports a model where both developmental susceptibility and environmental irritation are necessary for lesion expression. Thus, SGH is best regarded as a reactive inflammatory hyperplasia of odontogenic origin, triggered by multifactorial local irritants, and distinct from plaque-induced gingival diseases[4,7].

Histopathologically, the lesion is characterized by non-keratinized epithelial hyperplasia with prominent spongiosis and neutrophilic exocytosis[4]. In addition, features such as epithelial acanthosis, vascular congestion, and a dense inflammatory infiltrate in the lamina propria support the diagnosis[8]. Moreover, SGH demonstrates histopathological and immunohistochemical characteristics that show differences from plaque-induced gingival diseases[3,8]. Immunohistochemically, SGH displays cytokeratin expression including CK7, CK14, and CK19, with diffuse CK19 positivity across the entire epithelial thickness, a feature rarely observed in other reactive gingival lesions. Molecular and immunohistochemical markers further support this developmental hypothesis. Studies have shown increased proliferative activity (Ki-67 index) and inflammatory cell infiltration (CD3, CD20), indicating that SGH is both a reactive hyperplasia and a lesion with intrinsic epithelial predisposition[8]. Reporting this case series aids clinicians in recognizing the clinical features of SGH and avoiding unnecessary aggressive surgical management. This study aims to present a series of cases of spongiotic gingival hyperplasia treated at our oral medicine and pathology service.

MATERIAL AND METHODS

Patients were identified retrospectively through health records and histopathology archives of the oral medicine and pathology service at Jahra Dental Specialty Centre, Kuwait Ministry of Health, from January 2024 to June 2026. All consecutive patients presenting within this timeframe were evaluated to minimize selection bias, ensuring that all eligible cases encountered during the study period were fully included. Patients were initially identified based on primary clinical presentation of localized, bright red, velvety, or granular exophytic gingival enlargements unresponsive to plaque control or through initial diagnostic coding for gingival overgrowths. Inclusion criteria mandated a complete clinical record, photographic documentation, and an available tissue biopsy specimen for microscopic review. Exclusion criteria comprised clinical cases inconsistent with SGH such as drug-induced gingival overgrowths or inflammatory pyogenic granulomas lacking characteristic clinical features of SGH. SGH clinical diagnosis was made based on the literature and only when all the following criteria[1,3–5,7,8] were met for all cases:

a) well demarcated pebbly, velvety, papillary or granular bright red gingival lesion;
b) location of the lesion on facial/labial marginal gingiva and/or attached gingiva of anterior region;
c) onset during childhood or adolescence;
d) unresponsiveness to professional plaque control and reinforced oral hygiene;
e) absence of features suggest for other pathologies such as ulceration or rapid enlargement; and
f) clinical stability without progression for at least 12 months from follow-up.

Definitive histopathological diagnosis was established for the case underwent biopsy, which was sent for subsequent histopathological assessment and diagnosis.

RESULTS

This case series presents the characteristic clinical and histopathological features of SGH in six patients diagnosed and managed in our Oral Medicine and Pathology service from January 2024 to June 2026. Our six patients were aged 5 to 16 years (mean 12.2 years, median 12.5 years). Five of our six patients were females. Table 1 shows patients and clinical data of SGH cases with emphasis on their clinical features and management approaches.

Figure 1 shows generalized, well-circumscribed gingival erythema and hyperplasia involving the upper anterior gingiva in a 5-year-old female who presented with bleeding during brushing, as noted by her parents. The erythema was noted for 2 years, and clinically it did not show any progression during her follow-up appointments every 6 months for 2 years. Oral hygiene instructions were re-enforced to address bleeding while brushing. Figure 2 shows a solitary, well-circumscribed gingival hyperplastic lesion involving the upper right canine detected in a 13-year-old male with a 6-month history of its presence. He had mild tenderness while brushing, which gradually resolved after clinical follow-up every 6 months for 2 years. Figure 3 shows generalized multifocal gingival hyperplasia involving the lower anterior gingiva from the lower right canine to the lower left lateral incisor, presenting in a 15-year-old female. The patient noticed the lesions 12 months ago and was complaining of bleeding while brushing. During follow-up appointments scheduled every 6 months for

Table 1. Patient and clinical data of SGH cases presented to our oral medicine and pathology service with emphasis on their clinical features and management approaches.

Case	Age	Gender	Site	Size (mm)	Duration	Symptoms	Clinical Appearance	Differential Diagnosis	Treatment	Outcome / Follow-up
1	5	F	Maxillary gingiva	40×10	~2 years	Bleeding during brushing	Well-defined erythematous gingival hyperplasia with oedema Figure 1)	SGH, PCG	Monitor	No progression – followed up every 6 months for 2 years
2	13	M	Maxillary attached gingiva (#13)	5×5	~6 months	Mild tenderness while brushing	Solitary, well-defined, erythematous lesion (Figure 2)	SGH, PG	Monitor	No progression – followed up every 6 months for 2 years
3	15	F	Mandibular anterior gingiva (#32–43) region	10×2	~1 year	Bleeding during brushing, gingival tenderness	Multiple lesions of gingival hyperplasia (Figure 3)	SGH	Monitor	No progression – followed up every 6 months for 1 year
4	12	F	Maxillary gingiva (#11–13 region)	12×8	~8 months	Tenderness while eating and occasional bleeding	Gingival hyperplasia with a papillary/ pebbly surface (Figure 4)	SGH, PG	Monitor	No progression – followed up every month for 3 months then every 6 months for 1.5 years
5	16	F	Maxillary marginal gingiva extending to attached gingiva (#21)	4×5	~1 year	Asymptomatic, cosmetic concern	Erythematous sessile lesion with oedema (Figure 5)	SGH, PG	Monitor	No progression– followed up every 6 months for 1.5 years
6	12	F	Maxillary mesial marginal gingiva extends to the alveolar mucosa (#13)	7×7	~2 years	Mild tenderness while eating	Erythematous nodular lesion, pedunculated (Figure 6)	SGH, PG, POF	Excisional biopsy	Healed well, no recurrence after 11 months review post-operatively

SGH: Spongiotic gingival hyperplasia; PCG: Plasma cell gingivitis; PG: Pyogenic granuloma; POF: Peripheral ossifying fibroma.



Figure 1. Generalized well-circumscribed gingival erythema and hyperplasia involving the upper anterior gingiva.



Figure 2. Localized well-circumscribed gingival hyperplasia involving the upper right canine.



Figure 3. Generalized multifocal well-circumscribed red gingival hyperplasia involving the lower anterior facial gingiva.

12 months, no clinical progression was seen, and oral hygiene instructions were reinforced, which addressed her symptoms. Figure 4 shows a gingival hyperplastic lesion with a papillary/pebbly surface involving the upper left anterior gingiva extending from the upper left central incisor to the upper left canine in a 12-year-old female. Poor oral hygiene can also be seen. The lesion has been present for 8 months with symptoms of tenderness while eating and occasional bleeding. There has been no clinical progression of the lesion, however, the oral hygiene improved significantly following initial periodontal therapy and oral hygiene instructions. The patient was monitored closely to ensure optimum oral hygiene for the first 3 months, followed by 6 months follow-up appointments for 18 months. Figure 5 shows a sessile, well-circumscribed erythematous lesion involving the upper



Figure 4. Gingival hyperplasia with a papillary/pebbly surface involving the upper left anterior gingiva.

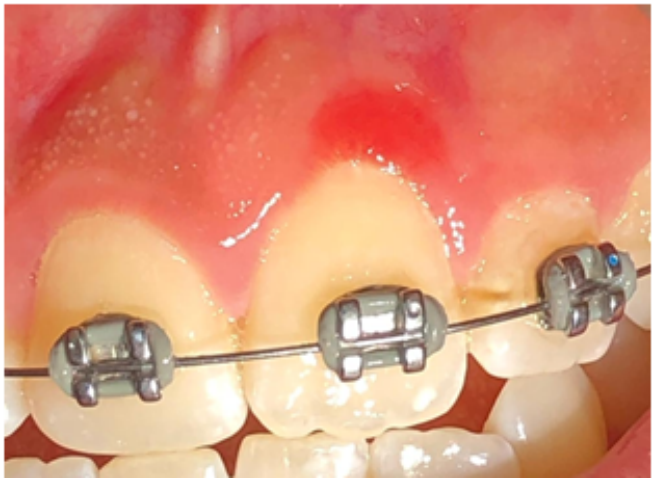


Figure 5. Localized well circumscribed gingival hyperplasia involving the upper left central incisor.

left central incisor in a 16 year-old-female undergoing orthodontic treatment. The patient was asymptomatic, however, she expressed her aesthetic concerns regarding the clinical presentation. The patient wishes to discuss the removal of the lesion once orthodontic treatment is completed, therefore she was monitored clinically every 6 months for 18 months with no clinical progression seen.

Figure 6 shows a solitary nodular lesion on the mesial marginal gingiva extending to the alveolar mucosa of the upper right canine in a 12-year-old female. The lesion has been present for 2 years, and it was agreed to excise the lesion under local anesthesia. The biopsy site healed well with no clinical recurrence after 11 months.

An excisional biopsy with histopathological examination and immunohistochemical testing was performed for one patient (case 6). The remaining five cases were diagnosed based on clinical features, as they declined biopsy at the time of consultation, and a mutual decision was made with the patients' parents to monitor the lesion clinically, with biopsy to be offered if the lesion progressed, became symptomatic, or developed atypical features.

Management

The clinical management of SGH involved surgical or laser excision while some cases were seen to regress spontaneously[6]. A total of six cases were managed over a period of 2 years since the initial consultation. Five cases clinically monitored every 6 months, mainly due to parents refusing biopsy at the time of consultation. One of the 5 cases wishes to discuss the removal of the lesion once orthodontic treatment is completed (Figure 5). The remaining case underwent excisional biopsy was a 12-year-old female patient, referred to the Oral Medicine Clinic for evaluation of a localized gingival enlargement in the left anterior maxillary region. The swelling had been present for approximately two years with minimal change in size. The patient reported mild tenderness and discomfort during mastication but denied bleeding, discharge, or systemic symptoms. Her medical history was unremarkable, with no known allergies. Extraoral examination was within normal limits, and no cervical lymphadenopathy was detected. Intraoral examination revealed a solitary nodular lesion on the mesial marginal gingiva extending to the alveolar mucosa of tooth #13, measuring approximately 7×7 mm (Figure 6). The lesion appeared erythematous with a pedunculated base and pebbly surface texture. Oral hygiene was fair.

Initial working diagnosis included SGH and excisional biopsy was planned to establish a definitive diagnosis. Local anesthesia was administered using 2% lidocaine with 1:100,000 epinephrine, followed by complete excision of the lesion. Hemostasis was achieved using silver nitrate (Figure 7). On histopathological examination (Figures 8–9), superficial fragments of mucosa covered by stratified squamous epithelium with irregular acanthosis, focal papillomatosis, elongated and focally thinned rete pegs, along with focal spongiosis and vascular dilatation with transmigration of neutrophils within the epithelium. Submucosal stroma shows



Figure 6. Single red nodule on the mesial marginal gingiva extending to the alveolar mucosa of tooth #13, measuring approximately 7×7 mm.



Figure 7. Post excisional biopsy of the lesion and hemostasis achieved with silver nitrate.

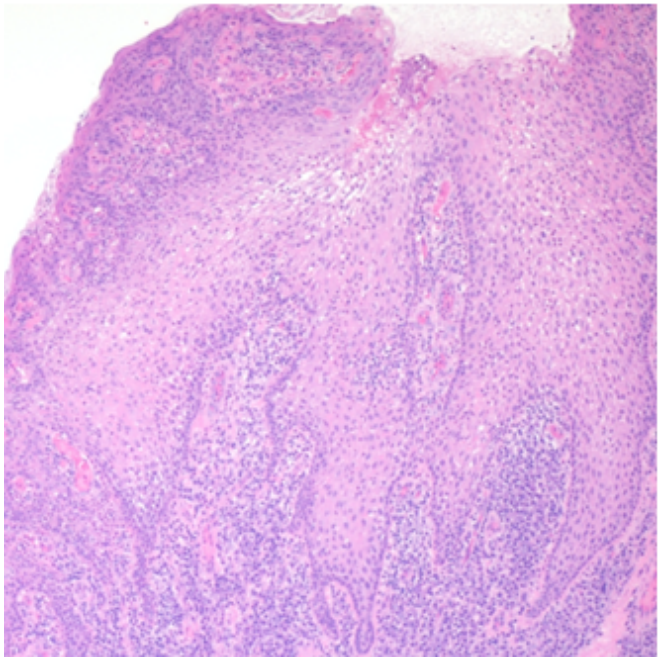


Figure 8. Hyperplastic surface epithelium with irregular acanthosis, elongated rete pegs, and spongiosis, overlying a densely inflamed lamina propria. The epithelial–stromal interface and full lesional architecture are appreciable at scanning power. (H&E, original magnification ×40).

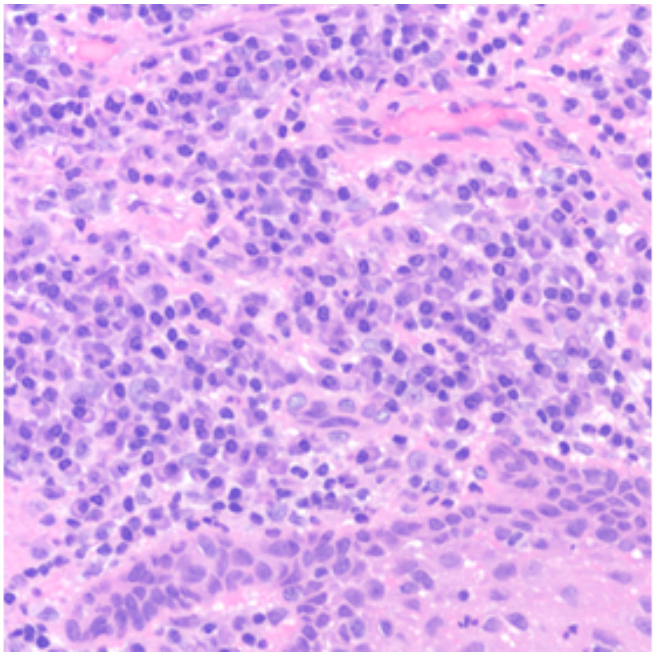


Figure 9. Higher-power view of the lamina propria showing a dense mixed inflammatory infiltrate composed predominantly of plasma cells and lymphocytes, with dilated, congested capillaries. No cytological atypia or dysplasia is identified. (H&E, original magnification ×400).

moderate to moderate-to-dense infiltrate of plasma cells and lymphocytes. Also noted is thinned-out adjacent mucosa; focal bacterial colonies. No abnormal full-thickness proliferative pattern to suggest dysplasia. Thus, the diagnosis of spongiotic gingival hyperplasia is formulated.

DISCUSSION

SGH presents as well-circumscribed, linear erythematous gingival lesion, most commonly affecting marginal gingiva of the upper anterior gingiva (Figure 1)[5]. The condition may present as solitary (Figure 2) or multifocal (Figure 3). The clinical appearance is typically described as exophytic gingival hyperplasia with a papillary, granular, or pebbly surface (Figure 4). It predominantly occurs in children and adolescents and is often asymptomatic, although mild discomfort or bleeding may be reported (Figure 5). Clinically, it can mimic plaque-induced gingivitis or pyogenic granuloma but characteristically shows minimal response to conventional plaque control measures[7]. SGH is considered a rare lesion, representing only 0.069% of 31,469 biopsies submitted to an oral pathology laboratory over a seven-year period[5]. Over the past decade, an increasing number of case reports and case series have broadened understanding of this entity, highlighting diverse clinical presentations such as multifocal involvement and cases occurring in older adults[5,9].

The upper labial gingiva of the maxilla is consistently the predominant site of involvement across clinicopathologic studies, although occasional multifocal lesions have also been reported[4,7]. Clinically, SGH typically presents on the attached gingiva, often sparing the marginal gingiva, and is not directly associated with dental biofilm, although plaque accumulation may worsen the clinical picture[6,7]. Lesions are usually bright red, painless papules or plaques with subtle papillary or granular surfaces, and they may occur either as focal or multifocal overgrowths[10].

The demographic profile of the presented case series is consistent with published literature. Our six patients were aged 5 to 16 years (mean 12.2 years, median 12.5 years) which is compatible to clinical case series of Leite et al.[10]; patient ages ranged from 6 to 24 years (mean 11.6 years)[10]. Although our youngest patient was 5 years old and none of our patients were adults, SGH is increasingly recognized[3,5].

For sex predilection in our case series, five of our six patients were female, in contrast to the equal sex

occurrence in Leite et al.[10] and the continuous absence of sex predilection in other studies with bigger sample sizes[4,5]. Thus, this finding of sex predilection is not representative due to the small sample size.

Lesion locations and site distributions mirrored the literature, with six cases presented in the anterior maxilla similar to the published cases[4,7] and one case in the anterior mandible. Clinical presentation of the lesions: four of the six were solitary, measuring between 4 and 12 mm, consistent with localized small lesions in the literature[4,8]. On the other hand, two cases (Case 1 and Case 3) presented with infrequent multifocal/generalized involvement, as it is increasingly being reported[5,9]; the lesions measured around 40x10 mm, which is larger than the reported ones.

Symptoms reported by the patients were consistent with the literature, including bleeding on brushing and painless growth[4,10]; in our case series, four patients reported mild tenderness while eating or brushing, three bleeding on brushing, and one asymptomatic with an esthetic concern only.

All showed papular or patch-like lesions on the vestibular gingiva of the anterior maxilla[10]. Reviews and larger surveys confirm a distinct clinicopathologic profile with no consistent sex predilection, supporting its recognition as a separate disease entity[4,5]. No lesion regressed despite improved and reinforced oral hygiene and plaque control, consistent with SGH resistance to conventional periodontal measures and management[6,7]. Thus, clinical management of SGH should be guided by definitive histopathological diagnosis to ensure appropriate treatment and follow-up planning[6].

Regarding the management of SGH, our case series differ from the published reports. Surgical excision remains the most common treatment as reported in the literature, but recurrence occurs in up to 25% within one year, and spontaneous resolution has also been documented[7,10]. In this case series, only one case provided parental consent to perform an excisional biopsy for definitive histopathological assessment and diagnosis (Figures 6–9), and no recurrence was seen at 11 months follow-up (Figure 10); however, the follow-up period is shorter than the usual time of 12 months at which recurrence is reported[7,10]. The remaining five patients were monitored without intervention from 12-24 months, which offers an uncommon opportunity to observe the natural course of untreated lesions. Spontaneous resolution has been documented in a minority of cases[6]; however, we noted no spontaneous resolution in our cases, which showed persistent but non-progressive lesions. Thus, our observation is suggestive that active monitoring is a reasonable option in stable patients with SGH lesions.



Figure 10. Post-operative clinical presentations after 11 months.

Other treatment modalities described include laser excision, trichloroacetic acid, cryotherapy, and photodynamic therapy[10,11]. The latter is considered effective due to its selective cytotoxicity, minimal invasiveness, and low incidence of adverse effects[8,12]. Fernandes et al.[2] described long-term follow-up cases showing lesion persistence even in older patients[2]. Lafuente-Ibáñez de Mendoza et al.[7] demonstrated through immunohistochemical analysis that SGH possesses a cytokeratin expression pattern consistent with junctional epithelium, strongly supporting an odontogenic developmental origin[7].

More recently, case reports have emphasized the esthetic and psychosocial impacts of SGH, especially in adolescents, where the conspicuous maxillary anterior location causes patient anxiety and treatment-seeking behavior[11,12].

CONCLUSION

Spongiotic gingival hyperplasia is a rare pathology that predominantly affects the anterior maxillary gingiva and is refractory to conventional

periodontal treatment and intervention. Histopathological examination is warranted for definitive diagnosis and proper treatment planning, and to avoid mismanagement. Surgical excision remains the main treatment modality, with potential for recurrence. Given its conspicuous anterior location and the esthetic and psychosocial burden it may impose, particularly on children and adolescents, greater awareness of this entity among dental and pathology practitioners is warranted to ensure timely and appropriate care.

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AUTHORS' CONTRIBUTIONS

AA: Conceptualization, Investigation, Methodology, Project Administration, Validation, Writing – review & editing. AB: Investigation, Validation, Writing – review & editing. MA: Writing – original draft.

CONFLICT OF INTEREST STATEMENT

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Ethics approval: This study was reviewed and approved by the Standing Committee for Coordination of Health and Medical Research of the Ministry of Health, protocol approval number: 2278/2023

DATA AVAILABILITY STATEMENT

The authors confirm that the data supporting the findings of this case series are contained within the article, including Table 1 and Figures 1–10.

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