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CLINICAL ARTICLE

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# Management of hyperplastic tissue response following connective tissue grafting

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## Abstract

**Objective:** While connective tissue graft shrinkage is a well-documented post-transplantation reaction, there is a literature gap concerning hyperplastic tissue response. Despite its infrequent occurrence, investigation is warranted due to its capacity to compromise esthetics, disrupt lip dynamics, and promote food retention. Moreover, efforts to mitigate hyperplastic tissue response often prove challenging, and there is a potential risk of exacerbating gingival tissue rebound.

**Clinical Considerations:** This report presents a potential solution to managing tissue overgrowth after connective tissue grafting in five clinical cases. The patients underwent corrective surgery involving internal excision of excessive tissue while preserving the overlying mucosa. The surgical approach was tailored to excise hyperplastic tissue with minimal trauma, aiming to optimize esthetic outcomes. Subsequent follow-up assessments spanning 1–5 years demonstrated stable results, with no indications of relapse or recurrence of tissue overgrowth.

**Conclusions:** Within the limitations of this case series, surgical internal excision holds promise as a viable treatment modality for addressing post-transplantation hyperplastic tissue response.

**Clinical Significance:** This case series addresses the challenge of uncontrolled tissue overgrowth following connective tissue grafting, a concern for which previous attempts have proven unsuccessful. Internal in-toto excision emerges as a promising approach for effectively eliminating overgrown tissue, offering potential advancements in the clinical management of this complication.

KEYWORDS

case series, connective tissue graft, esthetic surgery, gingival overgrowth, postoperative complication, tissue transplantation

## 1 | INTRODUCTION

Connective tissue grafting has evolved into a fundamental therapeutic modality for addressing various clinical concerns, including the management of gingival recession, augmentation of keratinized mucosa, alteration of the gingival phenotype surrounding natural teeth and implants, as well as the enhancement of tissue volume at pontic sites.[1–4] Various connective tissue graft (CTG) harvesting techniques and donor sites have been proposed in the dental literature. The selection of the donor site and harvesting technique plays a pivotal role in determining donor site morbidity, graft composition, and, consequently, the clinical behavior of CTG and the likelihood of complications.[5–12] It has been demonstrated that distinct donor sites exert varying impacts on the expression of genes associated with specific cellular functions, including collagen biosynthesis, extracellular matrix organization, and cell signaling.[10]

The palatal subepithelial connective tissue graft (S-CTG) was introduced in 1974 to augment keratinized gingiva. Subsequently, it

  

was used for the coverage of recession sites.[1,2] The harvesting of S-CTGs conventionally involves the deeper soft tissue layers of the anterior and lateral palate, ideally using a single incision to facilitate primary healing of the palatal wound. Upon transplantation to recipient sites, S-CTGs seamlessly integrate during the healing process, mirroring the color and texture of adjacent mucosa. S-CTGs typically exhibit limited lamina propria, accompanied by submucosa rich in glandular and fatty tissue, which can contribute to postoperative volume reduction.[4–7,11–13] Potential graft shrinkage, the sensitivity of the technique, and the need for thicker palatal tissues for S-CTG harvesting constitute the primary drawbacks of this type of graft.[12]

The de-epithelialized free gingival graft (DE-FGG) was introduced in 2003.[13] This technique offers the advantage of harvesting more superficial layers of posterior palatal tissues, primarily composed of lamina propria with dense collagen, ensuring dimensional stability. Despite the palatal wound healing through secondary intention, comparable patient-reported palatal discomfort has been documented following DE-FGG and S-CTG harvesting.[14] Notably, DE-FGG is less technique-sensitive and is applicable when dealing with thin palatal tissues. Nevertheless, this harvesting technique frequently retains epithelial residue within the graft.[15]

The maxillary tuberosity represents an additional potential donor area for connective tissue grafts (T-CTG).[16] The harvested tissue from this region is characterized by exceptional density and contains minimal submucosa. Compared to the palate, the postoperative donor site morbidity is lower. However, graft tissue availability is limited.[9,16]

Unusual graft responses have been discussed in the literature. Free gingival grafts (FGG) and CTGs have been associated with the formation of exostoses.[17–20] Epithelial inclusion cyst formation is also possible following incomplete epithelial removal, particularly in DE-FGG.[21–24] Hyperplastic tissue response (HTR) has received limited attention. This rare response has mostly been observed in connection with T-CTG.[7,25–29] However, tissue overgrowth after DE-FGG and S-CTG have also been reported.[7,13,] Emerging evidence suggests that CTG may induce HTR in various clinical scenarios, such as gingival recession coverage, pontic site augmentation, or after implant surgery.[7,13,25–29]

The understanding of HTR is currently limited. Genes related to specific cellular functions, such as collagen biosynthesis, extracellular matrix organization, and cell signaling, likely contribute to this phenomenon and exhibit varying expression levels depending on the donor site. These genes can increase collagen cross-linking, enhance its stability, and reduce susceptibility to remodeling by matrix metalloproteinases.[10] Tissue structure at grafted sites and LH2b gene expression in fibroblasts with and without HTR were analyzed. LH2b is known to be overexpressed in fibrotic processes, negatively influencing collagen susceptibility to undergo hydroxylation. A tendency was observed for greater LH2b mRNA levels and LH2b/COL-I mRNA ratio in grafted sites with hyperplastic tissue.[26] Interestingly, no significant morphological differences between overgrown tissue and healthy gingiva were found.[26,27]

Unlike skin keloids, which can be likened to HTR, no genetic studies have been conducted to establish evidence supporting a genetically determined, inherited susceptibility to intraoral hyperplastic lesions following connective tissue grafting.[30]

The hyperplastic response can result in undesirable cosmetic outcomes, changes in lip dynamics, food retention, and instill fear in patients who may perceive it as a malignant tumor. HTR poses challenges for removal. Various surgical gingivoplasty approaches have been used to address hyperplastic tissues, including laser therapy, diamond burs, and electrosurgery.[25–27] The reported outcomes exhibited short-term efficacy, marked by regrowth of hyperplastic tissue. In some instances, treatment exacerbated the overgrowth.[27]

This report presents the successful management of tissue overgrowth following connective tissue grafting in five clinical cases, with follow-up from 1 to 5 years. This case series aims to introduce an innovative approach for removing deeper grafted tissue, effectively eradicating hyperplastic tissues to prevent rebound and enhance cosmetic outcomes.

## 2 | CLINICAL CASE PRESENTATION, MANAGEMENT, AND FOLLOW-UP

Five patients, aged 32–53 years, presented with HTR after soft tissue augmentation using CTG, comprising four females and one male. The CTGs had been previously sutured underneath a split-thickness envelope flap on the buccal aspect of teeth or implants. All patients were in good systemic health and not on any medications. The procedures involving CTG-assisted soft tissue augmentation and the management of complications were conducted by two experienced dental surgeons in Croatia and South Africa.

One patient underwent soft tissue surgery employing S-CTG to address gingival recession and enhance the gingival phenotype as a preparatory step for subsequent restorative treatment. In two additional cases, a pedicle S-CTG was used to augment soft tissue and protect the bone graft in hard tissue augmentations. For the remaining two patients, T-CTG and DE-FGG were employed at the time of the post-extraction implant placement. Excessive gingival tissue overgrowth was observed 5–12 months post-graft surgery in four patients, and in one patient, it occurred after 3 years. Table 1 provides an overview of the harvesting sites, techniques, timing of HTR occurrence, and the follow-up duration after corrective surgery. All patients provided informed consent concerning the publication of intraoral images and case details.

## 3 | SURGICAL PROCEDURES

The hyperplastic tissues were meticulously excised from the inner aspect of the gingival tissues using surgical blades while preserving the overlying mucosa. Microsurgical instruments, magnification, and monofilament sutures were employed to minimize tissue trauma. The choice of flap design was based on the site (tooth or implant) and lesion location. A split-thickness envelope flap extending the

**TABLE 1** Harvesting area, technique, HTR timing, follow-up after HTR management, and site specification.

| Case Nr. | Areas[a] | Harvesting area | Harvesting technique | Timing of HTR (months) | Follow-up (years) | Site |
|---|---|---|---|---|---|---|
| 1 | 11, 21, 22 | Palate | S-CTG | 12 | 5 | Tooth |
| 2 | 12 | Palate | Pedicle S-CTG | 36 | 1 | Implant |
| 3 | 21 | Palate | Pedicle S-CTG | 7 | 5 | Implant |
| 4 | 21 | Tuberosity | DE-FGG | 5 | 5 | Implant |
| 5 | 11,21 | Palate | DE-FGG | 6 | 2 | Implant |

Abbreviations: DE-FGG, de-epithelialized free gingival graft; S-CTG, palatal subepithelial connective tissue graft.
[a]FDI numbering system.

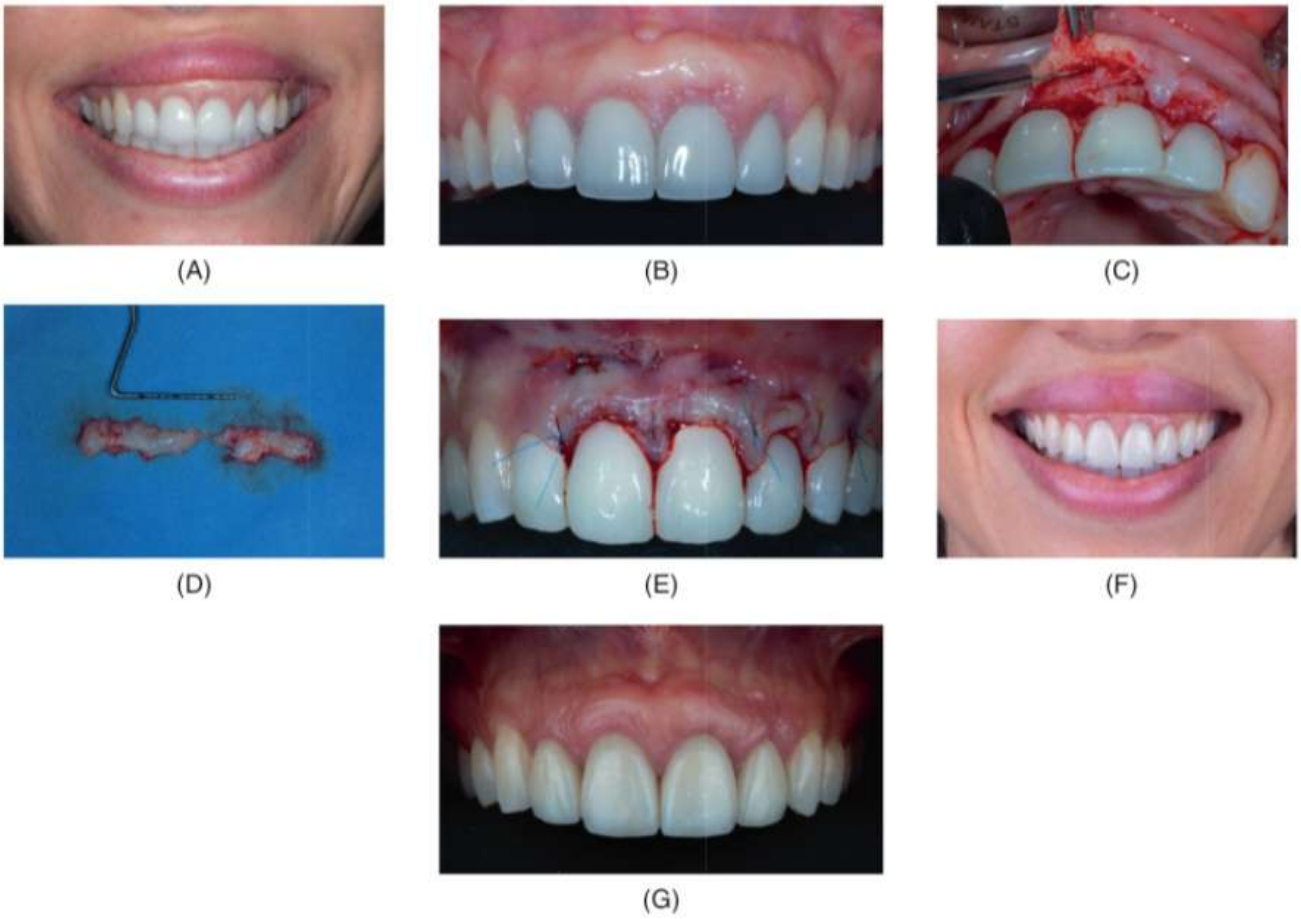


**FIGURE 1** (A) Pre-operative image showing hyperplastic tissue affecting upper lip dynamic. (B) Intraoral view of tissue overgrowth. (C) Split thickness flap was surgically elevated to excise the hyperplastic tissue. (D) Dense excess tissue removed. (E) Immediate wound closure. (F) Postoperative showing restoration of upper lip dynamic. (G) Five-year follow-up after treatment completion with no signs of hyperplastic tissue recurrence.

hyperplastic tissue length was raised around the teeth. In contrast, in implant cases, the marginal gingiva was not elevated; instead, an incision was strategically positioned away from the gingival margin where the lesion was situated apart from it (Cases 4 and 5). In Cases 2 and 3, where HTR was located near the gingiva margin, an internal excision was carried out through the sulcular approach, devoid of flap elevation.

### 3.1 | Case 1

The patient underwent a tunnel technique surgery with a free S-CTG to cover gingival recessions from teeth 12 to 23. Twelve months later, an unesthetic hyperplastic tissue response was observed in the upper maxilla (Figure 1A,B). Under local anesthesia, a split-thickness flap was elevated following a sulcular incision and papilla preservation with no

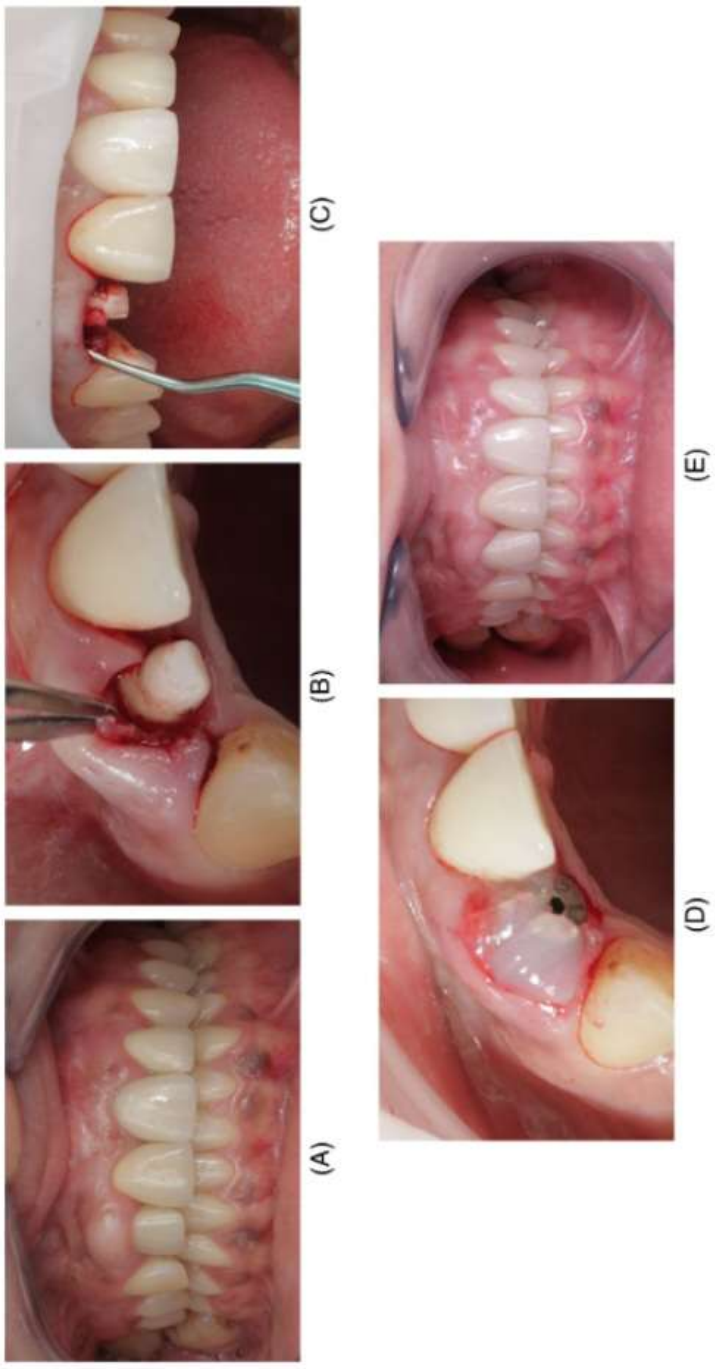


**FIGURE 2** (A) Pre-operative image showing tissue overgrowth. (B) Excess tissue excised in a flapless approach. (C) Removal of hyperplastic tissue. (D) Healing abutment in place and wound protected with an antimicrobial gel (BlueM, Wijhe, Netherlands). (E) Clinical aspect at one-year follow-up.

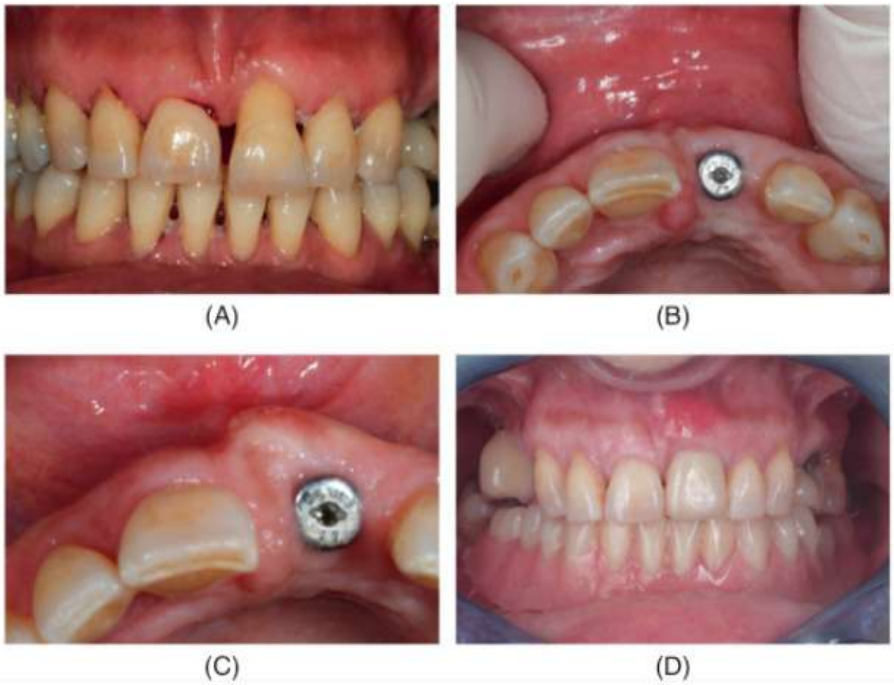


**FIGURE 3** (A) Hopeless tooth 21 with buccal hard and soft tissue deficiency. B) Five months after implant surgery, no tissue overgrowth was detected. (C) Seven months post-surgery, there was a notable hyperplastic response. (D) Five-year follow-up after corrective surgery showing no sign of tissue regrowth.

vertical releasing incisions. The dense, pale-colored tissue was sharply dissected using a 15 surgical blade and removed, leaving a 1-mm thick buccal flap (Figure 1C,D). The wound was closed using a 6.0 monofilament suture (Seralon, Serag-Wiessner, Naila, Germany, Figure 1E). The postoperative course was unremarkable. The 5-year follow-up after the corrective surgery is presented in Figure 1F,G. The gingival tissue volume remained stable, and there was no regrowth.

### 3.2 | Case 2

The hyperplastic tissue response developed 3 years after the implant site augmentation through a pedicled S-CTG in the 12 area (Figure 2A). After local anesthesia, the implant crown was removed, and a sulcular incision provided access to the hyperplastic tissue, which was removed utilizing microsurgical blades (MJK Instruments, Marseille, France), tunneling knives (Deppeler SA, Rolle, Switzerland) and tweezers. (Figure 2B). The periosteum was covered with a thin connective tissue layer, and the superficial mucosal flap was preserved. The marginal gingiva was carefully thinned to 2 mm, and the hyperplastic tissue was excised (Figure 2C). A healing abutment was temporarily installed until the completion of a new provisional crown (Figure 2D). One year post-corrective surgery, the tissue displayed a healthy appearance, with the postoperative dimensions effectively maintained (Figure 2E).

### 3.3 | Case 3

Five months after the extraction of tooth 21 and immediate implant placement combined with hard and soft tissue augmentation using a pedicled S-CTG, the peri-implant soft tissue exhibited a healthy appearance. (Figure 3A,B) Two months later, soft tissue overgrowth was observed in the augmented area (Figure 3C). Internal tissue excision was performed under local anesthesia, as described in Case 2. At the five-year follow-up, the mucosa was reddish without hyperplastic response (Figure 3D).

### 3.4 | Case 4

Soft tissue augmentation using a DE-FGG was conducted concurrently with immediate implant placement to replace tooth 21. The graft, harvested from the tuberosity, underwent extraoral de-epithelialization and was then inserted into the buccal split-thickness envelope flap following the implant placement procedure. Five months post-implant surgery, a rigid tissue mass developed apical to the area between the central incisors (Figure 4A). The patient had a previously placed implant in the adjacent 11 area, dating back 5 years. While the hyperplastic tissue was not evident during smiling, the patient insisted on its surgical removal due to interference with the dynamic movement of the upper lip. Consequently, one year after the implant surgery at the 21 area, corrective surgery was performed under local anesthesia. A horizontal incision in the vestibulum below the enlarged tissue revealed an overgrown tissue mass with the characteristics and consistency of dense connective tissue (Figure 4B). It was carefully and precisely dissected from the underlying periosteum and overlying mucosa, preserving approximately 1 mm of superficial mucosal thickness (Figure 4B). The wound was closed primarily using single interrupted sutures, employing Monofilament 6.0 (Surgicryl Monofast, SMI, St. Vith, Belgium), as illustrated in Figure 4C. A follow-up examination 5 years post-excision revealed no signs of recurrence (Figure 4D,E).

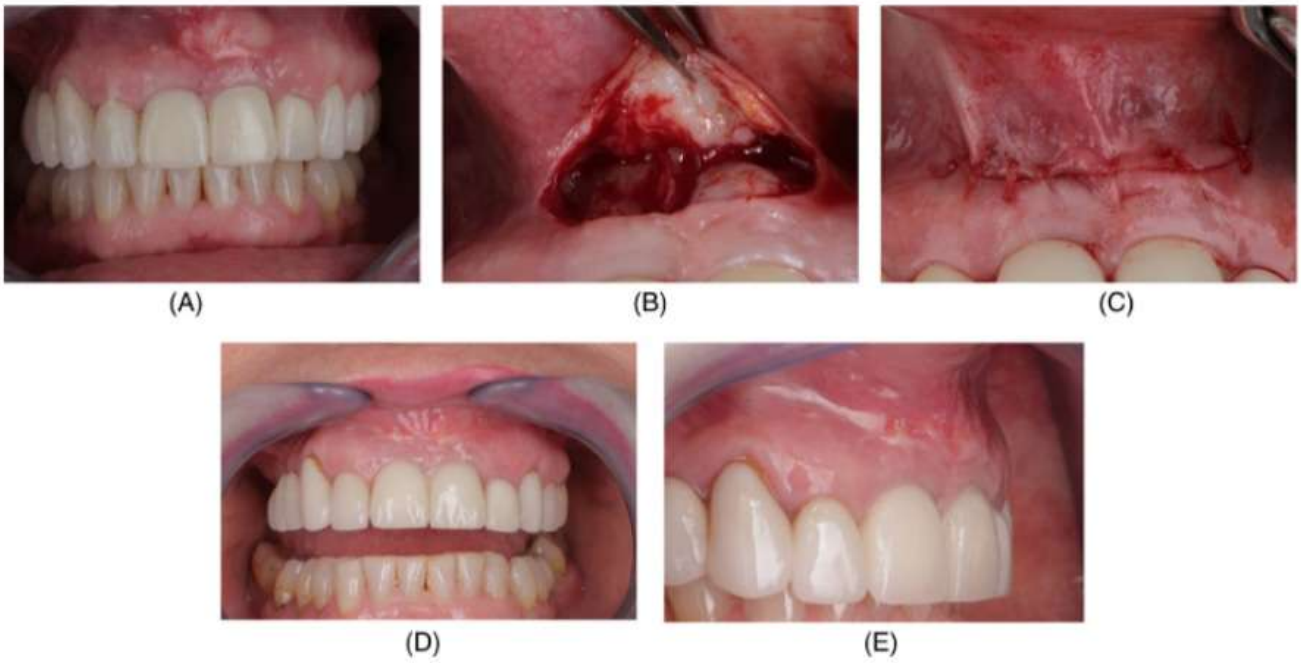


**FIGURE 4** (A) Hyperplastic soft tissue apical to both central incisors 5 months after S-CTG grafting surgery. (B) Horizontal incision coronal to the lesion and split-thickness flap elevated. (C) Wound closure. (D) Tissue appearance 5 years after complication management. (E) Visible scar with no hyperplastic recurrence.

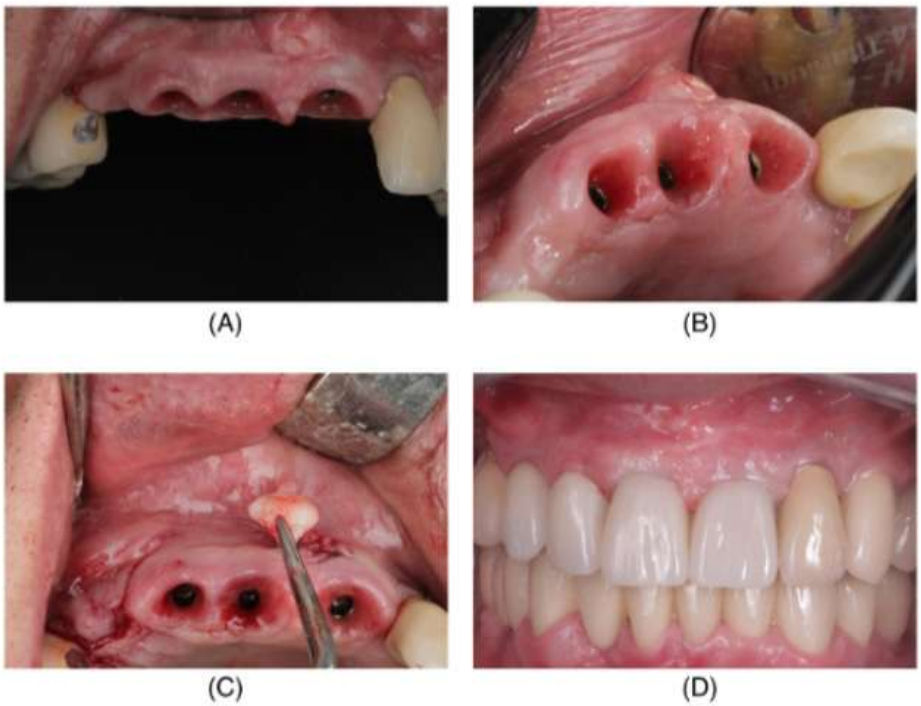


**FIGURE 5** (A) Frontal view of hyperplastic tissue response (HTR) on 21 area. (B) Occlusal view of HTR. (C) Hyperplastic tissue surgically removed. (D) Follow-up 2 years after corrective surgery.

### 3.5 | Case 5

A DE-FGG was placed within the buccal soft tissue tunnel during the immediate post-extraction implant placement in the 21 area. After 6 months, hyperplastic tissue was observed apically to the recipient area (Figure 5A,B). During the corrective surgery, a horizontal incision was executed under local anesthesia, strategically positioned near the coronal aspect of the HTR, utilizing a 15 surgical blade. A split-thickness flap was carefully raised to expose the affected area. The overgrown tissue was removed using micro-surgical instruments. (Figure 5C). A 2-year follow-up assessment revealed no recurrence of the HTR (Figure 5D).

## 4 | DISCUSSION

HTR can lead to unaesthetic gingival appearance, impaired upper lip dynamic, and food retention.[26,27] Considering the critical importance of optimal periodontal and peri-implant soft tissues and recognizing the frequent need for tissue augmentation, especially in implant dentistry, it becomes imperative to enhance our understanding of the triggers and treatment strategies for HTR. This case series presents a novel surgical intervention addressing HTR in four implant cases and one involving natural teeth. The outcomes reveal the successful restoration of esthetics and functional aspects with follow-up extending up to 5 years.

The literature on HTR primarily comprises limited case reports, with a predominant focus on tuberosity grafts and DE-FGG.[7,13,25–29] Dellavia et al. compared tissue composition following augmentation with T-CTG or S-CTG. Tuberosity grafts tended to become hyperplastic after 12 months, and tuberosity graft collagen was more mature and presented higher cross-linking, potentially explaining its higher resistance to metalloproteinase degradation. The authors reported that graft thickness over 3 mm was a potential risk factor for hyperplastic response.[26] In a study evaluating peri-implant soft tissue augmentation in 33 sites using CTG harvested from the lateral palate or tuberosity via a double incision technique, two palatal and three tuberosity grafts exhibited HTR 1 year after the procedure.[7] In some studies, HTR was discussed but not explicitly addressed as a problematic outcome.[7,29]

A human cadaver study investigating palatal grafts revealed that tissue composition was influenced by the harvesting technique but not by donor site location. The study found that DE-FGG contained more lamina propria and less adipose tissue than S-CTG.[5] Lamina propria generally comprises type I and III collagen (COL-1 and COL-III). A biopsy study on graft composition from different locations revealed a higher percentage of COL-I, denser collagen fibers, and more uniformly distributed COL-I in maxillary tuberosity lamina propria compared to palatal lamina propria. The expression of the COL1A1 gene was five times higher in tuberosity tissues, and lamina propria thickness was 1.5–2 times higher in tuberosity when compared to the lateral palate.[10] While tuberosity tissue overgrowth may be advantageous in some cases, such as ridge defect augmentation with composite tuberosity blocks, it often results in esthetically and functionally undesirable outcomes.[31]

Among the five cases outlined in this series, only one involved harvesting from the tuber area. The observed hyperplastic responses were evident regardless of harvesting technique and the fact that grafts were no thicker than 2 mm, suggesting that factors beyond graft thickness, harvest area, and technique may be implicated.

Few studies report on corrective surgery for HTR, and attempts to address these unesthetic lesions have, thus far, proven unsuccessful.[26,27] Dellavia et al. performed plastic surgery to remove the hyperplastic tissue resulting from T-CTG. After 9 months, the sites rebounded, gaining 70% of the volume eliminated by plastic surgery.[26] Gluckman et al. attempted to remove hyperplastic lesions following T-CTG in two patients through gingivoplasty using laser, surgical blades, and diamond burs. Both lesions increased in size after healing, suggesting the ineffectiveness of gingivoplasty at preventing relapse.[27]

An internal in-toto excision of the excess tissue was the treatment of choice in the cases presented in this series. The incision and flap design were carefully chosen to provide adequate access while minimizing the risk of gingival and papilla recession. A split-thickness marginal flap was raised in the natural dentition case, while a flapless approach was chosen for peri-implant tissue hyperplastic response. Lesions located more apical were approached through the horizontal incision below the HTR, avoiding marginal flap elevation. Peri-implant lesions involving marginal gingiva were removed similarly to an internal gingivectomy. The hyperplastic tissue was clearly distinguishable in all cases due to its firm consistency and pale appearance. In implant sites, the marginal mucosa was thinned to 2 mm after lesion removal and to 1–2 mm in natural teeth. The periosteum was retained in the implant cases, while it was removed along with the lesion in the case involving natural dentition. No relapse was observed 1–5 years following corrective surgery.

The primary constraints of this series encompass the limited sample size and the absence of extended follow-up for some instances. Further investigation is required, involving more extensive sample sizes and prolonged follow-ups to deepen our understanding of the causative factors and refine treatment strategies for HTR. Despite the limitations, to our knowledge, this series is the first to report on the internal excision of hyperplastic tissue. The type of flap employed and the presence or absence of periosteum did not appear to influence treatment results significantly.

## 5 | CONCLUSION

Within the constraints of this case series, in-toto excision emerges as a promising and viable treatment modality to effectively manage uncontrolled gingival overgrowth after connective tissue grafting, a concern for which previous attempts at resolution have proven unsuccessful.

### CONFLICT OF INTEREST STATEMENT

The authors declare that they do not have any financial interest in the companies whose materials are included in this article.

### DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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