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Keloids: A Retrospective Review and Treatment Algorithm

*Pooja SY, Kevin MK, Jordan G, Andres M, Carisa MC and Damon SC**

Department of Plastic and Reconstructive Surgery, The Johns Hopkins University School of Medicine, USA

Abstract

Introduction: Keloids cause substantial functional and psychological impairment, but effective treatment remains elusive. This study comparatively investigated treatment efficacy and recurrence rates associated with a variety of pharmacologic and procedural treatments intended to remove or reduce keloid scars.

Methods: We retrospectively reviewed patients aged 18 to 100 years with documented keloid diagnoses at our institution between January 1st, 2017 to January 1st, 2019. Study outcomes included improvement in keloid-related pain and pruritis, volumetric scar improvement, and keloid recurrence rates.

Results: 214 patients encompassing 391 scars met inclusion criteria. Most patients were African American (n=118, 55%), and the most common scar etiologies were surgery (n=125, 32%) and burns (n=117, 30%). Most keloids were on the head/face (n=106, 27%) and the chest/abdomen (n=78, 20%). Thirty-five percent (n=75) of patients failed treatment prior to presenting at our institution. Volumetric improvement 15 months post-treatment was greatest in burn-related keloids treated with laser therapy and intralesional steroid injection (80% with improvement, $p<0.01$) and for surgical scars treated with surgical excision and intralesional injection (63% with improvement, $p=0.03$); recurrence rates were similarly lower in these etiology/treatment pairings [burn scar recurrence: 30% ($p=0.03$); surgical scar recurrence: 26% ($p=0.01$), compared to 75% and 68% with intralesional injections alone, respectively]. These data were used to develop a management algorithm by scar etiology to optimize keloid treatment/minimize recurrence.

Conclusion: By compiling retrospective data from our dedicated keloid clinic, we identified a treatment algorithm that optimizes keloid volume reduction and minimizes recurrence rates, based on scar etiology. Such pathways can help to standardize keloid treatment.

Keywords: Keloid; Recurrence rates; Laser therapy; Intralesional injections; Surgical excision

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Correspondence:
Damon S Cooney, Department of Plastic and Reconstructive Surgery, Johns Hopkins University School of Medicine, Baltimore, MD, 21287, USA, Tel: (443) 287-6012; Fax: (410) 614-1296

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Introduction

Keloid scars are benign, fibroproliferative tumors known to cause pain, pruritus, contractures, and cosmetic disfigurement, resulting in both functional and psychological impairment [1,2]. Keloid scars are thought to form as a result of dysregulated wound healing, with disorganized deposition of type I and type III collagen bundles that can extend beyond the original incision or injury site [3,4]. To date, no singular, definitive factor behind keloid development has been identified, although a number of pathogenic mechanisms have been proposed in the literature [5]. Ultimately, a combination of environmental factors (e.g., trauma, inflammation) and genetic predispositions are thought to underlie keloid formation.

Although our understanding of keloid pathophysiology has grown, effective treatment remains a challenge [1]. In 1919, the first plastic surgeon, John Staige Davis, described a number of keloid treatments including excision and closure, excision and skin grafting, partial gradual excision, radiation, injections with mixtures of fibrinolysins, and freezing with carbon dioxide [6]. While similar strategies continue to be used today, we have also added more novel therapies such as corticosteroids, 5-fluorouracil, bleomycin, rapamycin, mitomycin C, laser therapy, silicone bandaging, and fat grafting [7-9]. However, treatment failures continue to be an issue, with some reports of up to 100% recurrence rates among treated keloid scars [10].

Given the complex, multifactorial etiology of keloids combined with their high recurrence rates, there is no well-established, universally-effective strategy for these challenging scar types [5]. This poses a tremendous challenge given that keloid scars confer substantial morbidity, and can worsen with every recurrence. Our institution has a high-volume clinic specifically dedicated to the treatment of patients with keloid scars. This study retrospectively investigated our institutional

experience, by reporting treatment efficacy and recurrence rates associated with different pharmacologic and surgical treatment regimens intended to remove or reduce keloid scars. As a secondary aim, this study attempted to identify a management algorithm to minimize keloid recurrence.

Methods

Study design

This was an Institutional Review Board-approved retrospective cohort investigation of patients who received keloid-related therapy at Johns Hopkins Hospital, Johns Hopkins Bayview Medical Center, and/or Johns Hopkins Green Spring Station (IRB00188758). Throughout the study we adhered to the Strengthening the Reporting of Observational studies in Epidemiology (STROBE) guidelines [11].

Study population and variables of interest

Eligible patients were those (1) aged 18 to 100 years, with a (2) documented diagnosis of keloid [International Classification of Diseases (ICD) 9 code of 701.4 or ICD 10 code of L90.1] and who (3) received therapy at Johns Hopkins Medicine for their keloid scar between January 1st, 2017 to January 1st, 2019. Keloid scar diagnoses were confirmed via pathology reports when available.

Demographic information, treatment details, and post-treatment outcomes were abstracted from the medical record for all included patients, from the first-reported treatment sought for their keloid scar throughout the course of their keloid treatment history. Follow-up in months was collected for all included patients. Patients were stratified into cohorts by keloid scar etiology and treatment modality: Surgery (i.e., radical resection of the scar, sometimes in combination with scar remodeling techniques such as z-plasty), intralesional injection (steroids, 5-fluorouracil), topical steroids, radiation, laser resurfacing therapy, and any other therapy intended to remove or reduce keloid scars. Patients who underwent combination therapy (e.g., injection and surgery) were evaluated separately.

Study outcomes included subjective improvement in keloid-related pain and pruritis symptoms after treatment, volumetric improvement in keloid scarring (e.g., response to therapy), and keloid recurrence rates (e.g., treatment failure). Patients were evaluated for pain and pruritus at each clinical visit, with each variable measured on a 10-point patient-reported scale where 0 indicated no pain/pruritis and 10 indicated the worst possible pain or pruritus. Scar response to therapy was defined as a binary variable (1 – scar responded to therapy, 0 – scar did not respond to therapy), where a positive treatment response was defined as a reduction in volumetric size of the keloid as documented in the clinical record, and as confirmed by 3D Vectra analysis of patient keloid photographs when available. Among keloids that initially responded to therapy, we used clinical records to determine scar recurrence rates, defined as recurrence of keloid scarring in the same location as the original scar without new insult/injury to the area. Keloid recurrence was also reported as a binary outcome variable. Study outcomes were compared between treatment cohorts.

Statistical analyses

All study analyses were completed using StataCorp version 15.1 (StataCorp, College Station, Texas). Statistical analyses were completed by scar, with hierarchical adjustments for clustering by patient. Patients with missing outcomes data were excluded from study analyses. All outcomes were two-tailed, with a significance level set at α of 0.05. Post-hoc power analyses were completed using G*Power Software (HHU, Dusseldorf, Germany).

Statistical analyses were aimed at comparing keloid scar treatment response and recurrence rates between different therapeutic modalities. Schapiro-Wilk testing was used to determine whether continuous variables were normally distributed. Chi-square and ANOVA analyses were used as appropriate to compare keloid treatment regimens, while Kruskal-Walli's testing was used to compare non-normally distributed variables (represented by median values and interquartile ranges) and Fischer exact testing was used for variables with low cell counts (less than 5). The association between treatment modality and keloid response/recurrence rates was investigated using univariable logistic regression. Variables found to be significant upon univariable analyses were then entered into a multivariable-adjusted logistic regression using stepwise forward selection (threshold for inclusion: $p<0.2$), to identify significant predictors of response/recurrence and to determine adjusted odds ratios. Kaplan-Meier and Cox proportional-hazards analyses were used to study time to first keloid recurrence.

We proposed a treatment algorithm aimed at minimizing keloid recurrence based on our institutional data. We used a logistic regression model to evaluate the algorithm, by predicting odds of keloid recurrence based on algorithm adherence versus nonadherence. Concordance statistics were used to evaluate model robustness.

Results

Study population and keloid scar characteristics

In total, 214 patients, encompassing 391 scars, met inclusion criteria during the study period. Table 1 presents patient demographics at the initial presentation to our institution. More than half of patients identified as African American (n=118, 55%). The most common scar etiologies were surgery (n=125, 32%) and burns (n=117, 30%), (Table 2). Keloid scars were most commonly found on the head/face (n=106, 27%) followed by the chest/abdomen (n=78, 20%).

Keloid treatment

Overall, around one third of keloid patients (n=75, 35%) had failed treatment prior to presentation at our institution, while the remainder presented with treatment-naïve scars. On average, treatment-naïve patients presented to our institution for initial keloid treatment 4 months (SD ± 3 months) after the initial occurrence of their scar. The distribution of treatments (both monotherapies and combination therapies) across the study population are demonstrated in Table 3.

At our institution, scar etiology was associated with treatment choice: Burn scars were significantly more likely to be treated with laser and adjuvant intralesional steroid injections, while trauma and surgical scars were significantly more likely to be treated with intralesional steroid injections followed by surgical excision if there was no improvement or in case of recurrence (post-hoc Chi-square analyses: $p<0.00001$). Among surgically-managed patients, the mean number of intralesional steroid injections prior to index surgery was 2 (SD ± 1) and the mean number after index surgery was 7 (SD ± 3), administered at intervals of 4 to 7 weeks. Mean length of follow-up for keloid scar treatment at our institution was 15 months (SD ± 5 months).

Treatment outcomes

With regards to pain and pruritis treatment outcomes, no

Table 1: Patient demographics at initial presentation (n=214 patients).

Demographic Factor	Value
Age in years, mean ± SD	39 ± 12
	Number of Patients
Sex, Female, n (%)	140 (65)
Race, n (%)	
Caucasian	83 (39)
African American	118 (55)
Other	13 (6)
Payer Status, n (%)	
Private Insurance	135 (63)
Public Insurance	79 (37)
Ethnicity, n (%)	
Not Hispanic/Latino	182 (85)
Hispanic/Latino	15 (7)
Unknown	17 (8)
BMI, mean ± SD	29 ± 7
Smoking history, n (%)	
Current	32 (15)
Former	36 (17)
Never	146 (68)
Family history of keloid scarring, n (%)	6 (3)
Comorbidities, n (%)	77 (36)
Hypertension	66 (31)
HLD	38 (18)
Anxiety/depression	25 (12)
CAD/PVD	18 (9)
Asthma/COPD	15 (7)
Autoimmune disorder	11 (5)
DM	14 (7)
Genetic syndrome*	1 (1)
Length of follow-up in months, median (IQR)	10 (7)

SD: Standard Deviation; BMI: Body Mass Index; HLD: Hyperlipidemia; CAD: Coronary Artery Disease; PVD: Peripheral Vascular Disease; COPD: Chronic Obstructive Pulmonary Disease; DM: Diabetes Mellitus
*Rubinstein-Taybi, Feminine

Table 2: Keloid scar etiologies and locations by frequency (n=391 scars).

	Number of Scars (%)
Etiology	
Surgery	125 (32)
Burn	117 (30)
Idiopathic	39 (10)
Piercing	39 (10)
Infection/folliculitis/acne	35 (9)
Trauma	16 (4)
Other	12 (3)
Insect Bite	4 (1)
Pregnancy	4 (1)
Location	
Face/head (including ears)	106 (27)
Chest/abdomen	78 (20)
Hand	59 (15)
Neck	47 (12)
Upper arm/forearm	39 (10)
Pelvic/Perineal/Gluteal Region	27 (7)
Thigh/Leg	8 (2)
Foot	4 (1)

Table 3: Keloid scar treatments by frequency (n=391 scars).

	Number of Scars (%)
Monotherapy	
Corticosteroid Injection	94 (24)
Excision	47 (12)
Topical Therapy (Steroid, Pressure Dressing, Silicone)	8 (2)
Radiation	4 (1)
Combination Therapy	
Excision + Steroid Injection	125 (32)
Laser + Steroid Injection	70 (18)
Excision + Steroid Injection	23 (6)
Excision + Steroid and 5-Fluorouracil Injection	8 (2)
Excision + Radiation	8 (2)
Excision + Laser	4 (1)

individual treatment type (e.g., surgical excision, intralesional injection, laser, etc.) or treatment combination (e.g., surgical excision with intralesional injection) was clinically superior across all keloid scar etiologies. When comparing baseline pain/pruritis scores to post-treatment pain/pruritis scores among the most common therapies that patients received (surgical excision alone, intralesional injection alone, surgical excision with intralesional injection, and laser therapy with intralesional injection), all treatments resulted in modest improvements in pain and more substantial improvements in pruritis across the keloid patient cohort (Table 4).

When considering keloid scar treatment response (i.e., improvements in the volumetric size of the keloid after treatment) and keloid recurrence, however, different treatments had differing levels of success. Treatment success in these two domains was found to depend on a number of scar-level factors.

With regards to keloid scar appearance (i.e., keloid size), long-term treatment response varied by scar etiology. When considering the treatments used for the two most common scar types (burn and surgical scars), laser therapy with adjuvant intralesional steroid injection was found to best improve the long-term appearance of burn-related keloid scars: 80% of patients with burn scars who received laser therapy and intralesional injection reported improvement in keloid appearance 15 months post-treatment compared to 41% with intralesional injection alone ($p<0.01$). On the other hand, surgical excision combined with intralesional injection was found to best improve the long-term appearance of post-surgery scars: 63% of patients with surgical scars who received excision and intralesional injections reported improvement in keloid appearance 15 months post-treatment compared to 45% of patients who received intralesional injection alone ($p=0.03$).

For keloid scar recurrence, which was defined as return of

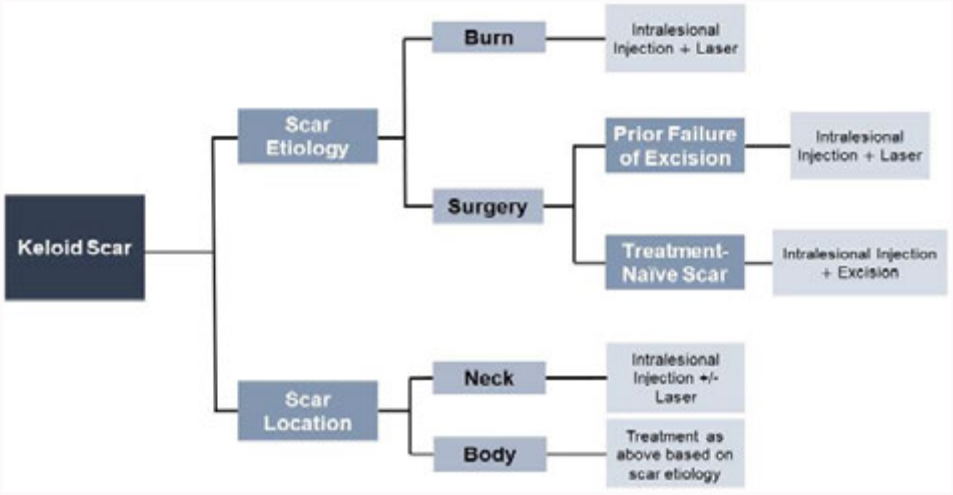

Figure 1: Keloid scar management algorithm designed to minimize scar recurrence.

fibrous scar after an initial improvement in scarring, we identified a number of possible contributing factors: Scar etiology, history of prior treatment, and scar anatomic location. We used the results of our scar recurrence analyses to generate a management algorithm designed to minimize recurrence among keloid scars that initially respond to therapy (Figure 1). Overall, our patient cohort recurrence rate for scars that initially responded to therapy was 46% with a mean time to keloid recurrence of 13 months (SD ± 2 months) after initial treatment.

First, we investigated scar etiology, with specific consideration for the most common keloid etiologies (surgery, burn; Table 5). We found that patients with burn scars had a significantly lower recurrence rate with laser therapy and intralesional corticosteroid injections (recurrence rate: 30%, p=0.03). In contrast, among keloid patients who had surgical scars, those treated using a combination of excision and intralesional steroid injections demonstrated significantly lower rates of scar recurrence when compared to other treatment options such as intralesional injection alone (Figure 2; recurrence rate: 26%; p=0.01). Conversely, keloid patients with surgical scars who were treated only with intralesional injection had the greatest rates of scar recurrence when compared to other treatment options (recurrence rate: 68%; p=0.01). Furthermore, among patients with surgical scars, Cox proportional-hazards modeling demonstrated that patients treated with excision and intralesional steroid injections had significantly longer intervals to first keloid recurrence compared to patients treated with intralesional injection alone (mean time to recurrence 10 months for surgery with injections versus 6 months for injections alone; p=0.04; Figure 3 and Table 6).

Among patients with surgical keloid scarring, we found that history of prior treatment failure impacted recurrence rates. After adjusting for demographic and baseline treatment factors, patients with surgical scars who failed prior surgical excision had significantly decreased odds of recurrence, if they underwent treatment with laser and intralesional steroid injections rather than repeat excisional procedures, (adjusted odds ratio: 0.7, 95% CI 0.5-0.9, p=0.03).

Lastly, for keloid scar recurrence, we found that it was important to consider anatomic location. Among patients with keloid scars on the neck, often due to ingrown hairs or shaving wounds, surgical excision was incidentally found to increase odds of recurrence after

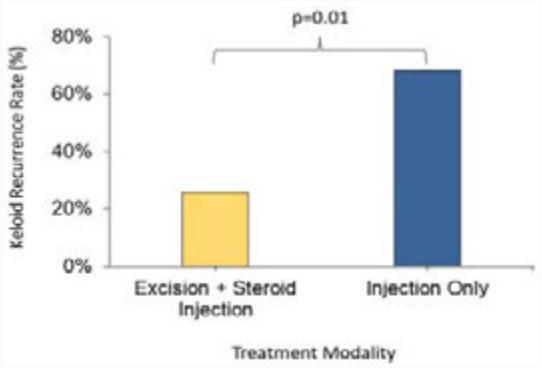

Figure 2: Keloid surgical scars treated with a combination of excision and intralesional steroid injections demonstrated significantly lower recurrence rates compared to intralesional injection alone.

adjusting for patient demographics and scar etiology (adjusted odds ratio: 1.3, 95% CI 1.1-1.5, p=0.03). Intralesional steroid injection alone or laser therapy with intralesional injections were found to be more efficacious for keloids in this location, although these therapies did not result in a significantly decreased adjusted odds of recurrence (adjusted odds ratio for steroid injection alone: 0.8, 95% CI: 0.6-1.2, p=0.08).

Across the study population, treatment-related adverse events included hypopigmentation in those who received intralesional steroid injections (n=15, 5%) as well as among those who received laser ablation (n=8, 7%). Among the few patients whose keloids were treated with radiation, there were no reports of radiation therapy-related malignancies.

Treatment algorithm validation

After generating an algorithm to minimize scar recurrence using our study results, we validated it using logistic regression. Patients with keloid scars that initially responded to therapy were stratified by whether or not their scars were treated in accordance with the aforementioned algorithm based on their etiology, history of prior therapy, and anatomic location. The recurrence rate amongst scars treated in adherence with the algorithm was 27%, compared to 55% amongst those nonadherent to the algorithm. Adherence to the algorithm significantly decreased odds of keloid recurrence (OR: 0.5;

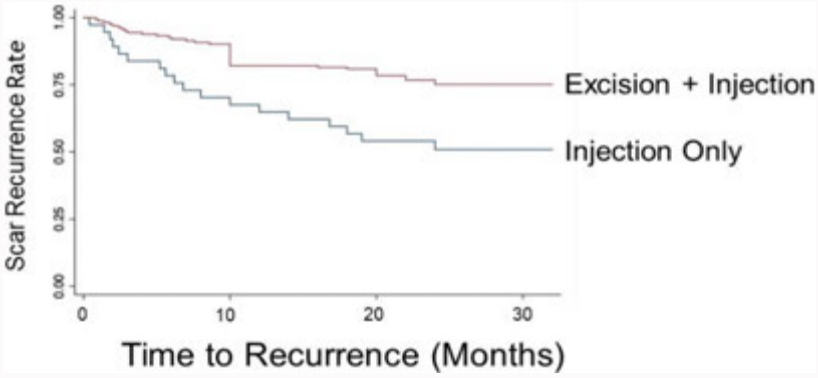

Figure 3: Kaplan-Meier curves demonstrating that patients treated with excision and intralesional steroid injections had significantly longer intervals to recurrence compared to those treated with intralesional injection alone.

Table 4: Keloid treatment outcomes: Changes in pain and pruritis by treatment modality (n=391 scars).

	Excision Alone	Excision + at least 2 Intralesional Injections	Intralesional Injection Alone	Laser + at least 1 Intralesional Injections	p-value
Change in pain*, median (IQR)	-1 (2)	-2 (2)	-1 (2)	0 (2)	0.4
Change in pruritis*, median (IQR)	-3 (2)	-4 (3)	-3 (3)	-4 (2)	0.26

*Based on a 0-10 scale where 0=none and 10=worst imaginable, IQR: Interquartile Range

Table 5: Treatment failure (Recurrence) rates by scar etiology and treatment modality.

	Intralesional Injection Alone	Excision Alone	Laser + Intralesional Injections	Excision + Intralesional Injection	Surgery + Radiation	Radiation Alone	P value*
Post-Surgery Scar	68%	58%	50%	26%	30%	41%	0.01
Burn Scar	75%	50%	30%	44%	N/A	N/A	0.03

N/A: Not Applicable (this treatment combination was not used for this particular scar etiology); *p values calculated using chi square testing amongst treatment modalities used

Table 6: Mean (± SD) time in months to first scar recurrence by scar etiology and treatment modality*.

	Intralesional Injection Alone	Excision Alone	Laser + Intralesional Injections	Excision + Intralesional Injection	Surgery + Radiation	Radiation Alone	P value
Post-Surgery Scar	6 (2)	7 (4)	7 (3)	10 (3)	10 (5)	6 (2)	0.04
Burn Scar	5 (2)	8 (3)	12 (5)	8 (4)	N/A	N/A	0.02

*Data reported as Mean (Standard Deviation), SD: Standard Deviation; N/A: Not Applicable (this treatment combination was not used for this particular scar)

95% CI: 0.4-0.7; p=0.02). Figure 4 demonstrates model validation using area under the Receiver Operating Curve (ROC).

Post-Hoc power calculations

Given the retrospective nature of this investigation, post-hoc power analyses were undertaken after the study cohort was defined [12]. Based on the study sample size for each respective keloid etiology, only burn scars and post-surgical scars were adequately powered for multivariable investigations (power >0.8); therefore, only these sub-cohorts were investigated and the results of their multivariable analyses presented.

Discussion

In this retrospective review of our high-volume institutional keloid clinic, we found several factors associated with keloid scar treatment success rates. These included (1) keloid scar etiology and subsequent treatment, (2) whether the keloids were recurrences or treatment naïve, and (3) keloid scar location. Keloids resulting from burns treated with laser therapy and intralesional corticosteroid injections were less likely to recur. Keloids resulting from surgery treated with a combination of excision and intralesional steroid injections were less likely to recur and experienced longer intervals to keloid recurrence compared to other treatments. Surgical scar keloid patients who failed prior excision had lower odds of recurrence when treated with laser and intralesional steroid injections compared to those treated

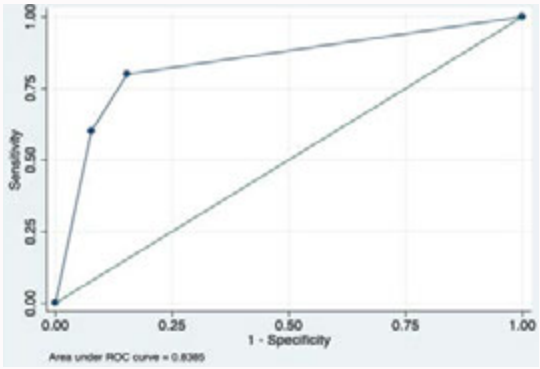

Figure 4: Treatment algorithm model validation using area under the Receiver Operating Curve (ROC).

with repeat excision. Finally, in treatment-naïve patients, considering the location of keloid scars can inform type of treatment; notably, keloids located on the neck recurred less often when treated with intralesional steroid injection alone or steroid injection combined with laser therapy. To best summarize these treatment pathways, we created and validated a keloid treatment algorithm (Figure 1), with the goal of assisting other physicians serving patients with these challenging scars.

Reasons for these different responses to treatment are unknown

and may be driven by varying pathogenic mechanisms behind each keloid type's formation. Surgical excision has the benefit of removing some or all of the keloid and may be preferred depending on keloid size, an acute need to remove some or all or the scar, or a patient's limited ability to return to clinic for serial treatment. Pairing excision with corticosteroids is useful as following keloid removal, subsequent steroid injection blocks the proliferative processes incited by excision [13]. Our data supported this approach when treating treatment-naïve keloids resulting from surgery, demonstrating lower recurrence rates and longer keloid-free intervals between recurrences.

Among burn keloids and keloids that failed prior excision, laser therapy paired with intralesional corticosteroid injection was found to be the most effective. Keloid recurrences are often seen in more severe scars and can result in larger keloid scars than what was initially present [14,15]. Thus, as demonstrated in our retrospective review, alternative treatment modalities may be more efficacious. Lasers rely on photochemical and thermal reactions to denature collagen and other structural proteins, also reducing erythema and pruritis through their effect on scar microvasculature [16]. Unlike re-excision which may stimulate additional collagen synthesis, Nd:YAG lasers may selectively suppress fibroblast collagen production [17]. Pulsed-Dye Lasers (PDL) and Nd:YAG and Nd: Van lasers are thought to selectively damage blood vessels to the scar, inducing hypoxia in the keloid [18]. While, CO_2 lasers ablates the keloid scar tissue creating a microthermal zone; these zones modulate the immune response and can induce tissue repair [19]. Pairing laser treatment with topical corticosteroids appears to enable the steroid to access more keloid tissue *via* microchannels created by the laser, increasing the drug's bioavailability [20]. Thus, laser therapy with adjuvant corticosteroid injection or laser therapy alone may prevent additional recurrence in scars that demonstrate excessive collagen synthesis and fibrosis in response to surgical excision.

With regards to keloid location, we found neck scars were at significantly greater risk for treatment failure (i.e., scar recurrence) after excision. In our cohort, neck keloids comprised around 10% of all keloids, and were largely the product of ingrown hair or shaving wounds. It is known that neck skin is different in composition than skin in other anatomic locations of the body, with a different collagen construct [21]. Thus, the re-injury to the neck tissues during surgical excision may result in different healing patterns than in other areas of the body. A 2016 study by Tirgan similarly noted neck keloids' poor response to surgical excision [22]. Given the importance of appropriate management of neck keloid scars because they can be difficult to conceal and can cause functional and cosmetic distress, our findings suggest avoiding excisional therapy if possible. Prior literature reports success with mitomycin-C, a chemotherapeutic agent that inhibits fibroblast proliferation, for neck keloid scars [8]. Further work should investigate the relative utility of alternative therapies for this challenging subset of keloid scars.

An additional outcome of interest was pre- to post-treatment change in patient-reported pain/pruritis scores. We found that all treatments demonstrated modest improvements in pain and greater improvements in pruritis, although no treatment demonstrated superiority. This is similar to other studies' findings in that patients treated with laser therapy and intralesional corticosteroid injections demonstrated modest improvements in pain while those treated with laser therapy and topical corticosteroids demonstrated significant improvement in pain scores [20].

This study has limitations. As a single-institution retrospective investigation, our findings' external validity and ability to determine causation is limited. However, our institution has a high-volume keloid clinic run by the senior author (DSC) which draws patients from around the country; as such our findings may provide further insight regarding successful treatment of these challenging scars. Additionally, not all available therapies for keloids were represented and evaluated in this investigation. However, a range of the most common therapies (e.g., intralesional injections, surgical excision, laser) were evaluated and are represented in the study results. Additionally, our post-hoc power analysis indicated sufficient power to detect differences between surgical and non-surgical treatment of keloid scars.

While data in the current study were derived from clinical reports in the medical record, using keloid-specific patient reported outcome measures may better characterize treatment response and delineate between treatment types. Future prospective investigations should include validated scar scales and patient quality-of-life measures. Additionally, future investigation should focus on long-term outcomes in patients with keloid scars. The average length of follow-up in this investigation is 10 months, but keloid scarring has chronic implications for patients' health. Thus, more longitudinal follow up can help determine whether treatment efficacy is maintained over time.

Conclusion

Despite advances in patient care, keloid scars remain challenging to treat. Upon review of data from our institution's keloid patients, we identified treatment patterns and outcomes by scar etiology, recurred versus treatment-naïve keloid status, and location to improve treatment success in keloid patients as measured by improved keloid scars and reduced recurrence. We used these data to create a treatment algorithm from our institutional experience to help inform other practitioners' management of these challenging and highly-morbid scar types.

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