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Review Article

Auricular keloids: a literature review

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ABSTRACT

Keloids represent a complex pathological response to the wound healing process, characterized by an overgrowth of dermal fibrosis that exceeds the boundaries of the original wound. The auricle, particularly the earlobe, is a region of heightened susceptibility, owing to increased skin tension and frequent injury, most commonly resulting from ear piercing. The persistent nature of keloids, coupled with their high rate of recurrence, poses a significant therapeutic hurdle for medical professionals.

Keywords: Keloid, Auricular, Earlobe, Pathogenesis, Fibroproliferative, Triamcinolone, Brachytherapy, Surgical excision

INTRODUCTION

Keloid disease exemplifies the intricate nature of human wound healing. When this process becomes aberrant, a lesion can result in both cosmetic and functional complications. Keloids arise from the overproduction of dense fibrous tissue that extends beyond the original wound. They are distinct from hypertrophic scars, which are characterized by their tendency to spread and not spontaneously regress.[1] A significant proportion of head and neck keloids are located on the auricle and earlobe (Figure 1). This predilection is attributable to heightened skin tension, a more delicate dermis, and recurrent medical trauma, particularly from piercings. Patients frequently contend with social anxiety, diminished self-esteem, and, in more severe cases, physical pain or functional limitations associated with hearing aids.[3]

Auricular keloids present a challenge for plastic surgeons. While surgical excision is feasible, these lesions are resistant to physiological resolution. This review will examine the molecular mechanisms underlying auricular keloids and their therapeutic applications, thereby providing a comprehensive resource for achieving enduring outcomes. Keloids have distinct epidemiological characteristics by genetics and

demographics. The condition is particularly common in individuals with darker skin, and affects 4.5% to 16% of individuals of African, Asian, and Hispanic descent, while the incidence is approximately 0.1% in the Caucasian population.[4] The discrepancy underscores the role of genetic factors, which are typically associated with skin type. The prevalence is greatest amongst individuals in their twenties and thirties. This age group frequently socializes more, empathizing their likelihood of sustaining ear piercings or other trauma. There is typically a small predominance in females as we know women generally have a higher rate of earlobe piercings.[5]

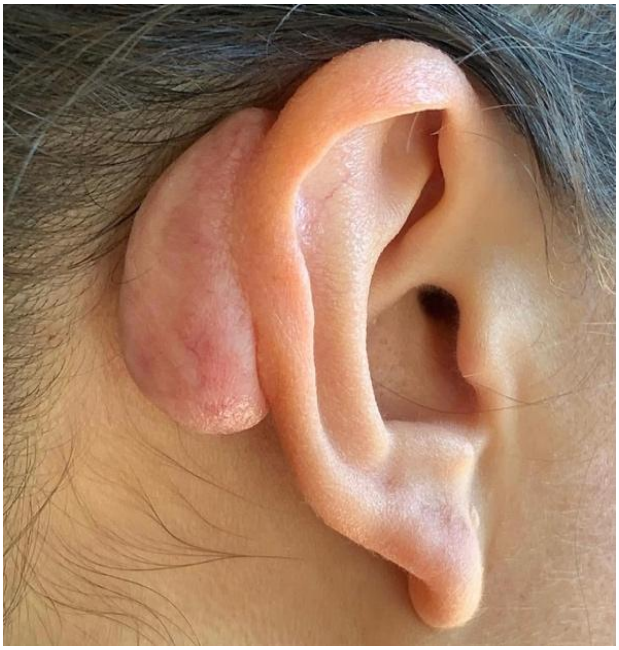

Figure 1: Posterior auricular keloid (3.5×4 cm).

PATHOPHYSIOLOGY: THE MOLECULAR PERFECT STORM

Keloids arise due to a number of biological problems. These problems start with an unusually protracted and intense inflammatory response. This response then leads to uncontrolled cell growth and a significant increase in collagen production.[6] People who are prone to keloids show a quicker and longer-lasting influx of platelets and inflammatory cells after an injury. This leads to an unbalanced environment of cytokines. Transforming growth factor-beta (TGF-β) is the main cytokine that drives fibrosis. When the pro-fibrotic isoforms, TGF-β1 and TGF-β2, are more common than the anti-fibrotic isoform, TGF-β3, this process stimulates fibroblasts and enhances collagen synthesis.[7] Furthermore, the chronic inflammatory condition characteristic of fibrosis is sustained by increased levels of platelet-derived growth factor (PDGF), a strong mitogen and chemoattractant that promotes fibroblast proliferation, together with pro-inflammatory interleukins including IL-6 and IL-13.[8,9] As a result, fibroblasts from keloids show specific differences, such as excessive growth when exposed to substances that encourage cell division, along with increased activity in creating new proteins. Keloid fibroblasts produce more type I and III collagen, fibronectin, and glycosaminoglycans. This leads to the creation of disordered extracellular matrix bundles that are up to twenty times more prevalent than those formed by normal fibroblasts.[10]

These fibroblasts also show great resistance to apoptosis due to a shift in pro-apoptotic and anti-apoptotic proteins, which allows their persistent presence and fibrillar matrix production well beyond the normal time frame for wound healing.[11] Epigenetic and environmental factors are ultimately important in this pathophysiological process; hypoxia in the keloid microenvironment stimulates hypoxia inducible factor-1α which promotes collagen production. At the same time, mechanical tensile stress activates signaling through Yes-associated protein (YAP) and Transcriptional co-activator with PDZ-binding motif (TAZ) to promote proliferation and inhibit apoptosis.

MANAGEMENT STRATEGIES

Caring for auricular keloids requires careful consideration, as single treatment plans are unsuccessful the majority of the time, and even in combination, multiple treatments are typically more beneficial.[12] Corticosteroids injected directly into the lesion are the most common and effective non-surgical treatment for keloids. Triamcinolone acetonide is used nearly exclusively because it provides anti-inflammatory properties, decreases collagen production, and induces fibroblast death. TAC is injected in concentrations of 10-40 mg/mL every 4-6 weeks, making the exact position and injection site of the lesion quite critical to minimizing the frequent adverse effect of dermal atrophy.[5] Second-line chemotherapeutic treatments include 5-fluorouracil, a pyrimidine analog that inhibits fibroblast proliferation, which is more effective at decreasing keloid size and recurrence when combined with TAC instead of utilized alone; despite being a potential complication, myelosuppression with intralesional delivery is rare.[13] In addition, bleomycin is another antineoplastic agent that requires multiple punctures for delivery into the keloid and acts by inducing breaks in the DNA strand; while effective, it can also cause hyperpigmentation and induce dermal atrophy.[14]

Cryotherapy with liquid nitrogen promotes the penetrative ability of the medication into the keloid by softening the keloid; however, it may also impart the risk of skin lightening.[15] Silicone gel sheeting usually works better with raised scars, as it may hydrate skin, alter cytokine levels, and lessen itching in smaller, newer keloids, while it tends to be limited in its effects. Although surgery is significant in the removal of excess tissue, its use is the first step before any additional treatment to consider. This is because surgery resets the body's healing process and creates another "wound" that must be managed to prevent keloid recurrence.[16]

Different types of surgical techniques exist. The elliptical excision, or "surgical resection" is the most common type of surgery because it allows careful management of the tissue and closure of the skin under no tension--at times, a subcuticular technique is used, to help facilitate less inflammation.[17] There is the core excision, which is when physicians remove the middle mass of the keloid, but keep the epithelial layer on, and it is believed the nudging affects recurrence, although the efficacy remains debated There is the shave or flush cut excision for larger, pedunculated lesions, likely effective with immediate coagulation and combination treatment.

What matters the most is just starting the necessary adjunctive treatments following surgery in the first 24-96-hour window, before the reformation of the fibrotic process begins. The delay may lessen any benefit you may have with surgery. The adjuvant treatment of choice is postoperative radiotherapy, superficial X-ray or electron beam therapy, at 12-20 Gy, is very effective. The reduction from destruction of quickly growing fibroblasts--the recurrence risk falls to between 10% and 25%. Also, the use of modern, calibrated systems greatly decreases chance of cancer.[18]

Intralesional corticosteroids continue to be the mainstay of treatment; the usual method is to inject triamcinolone acetonide (TAC) into the margins of the wound perioperatively, which is followed by a continuous schedule of injections postoperatively for 3 to 6 months. In addition, laser therapy is available as an adjunct. Pulsed-dye laser (PDL) is commonly used to target the microvasculature to reduce the effects of erythema, but CO_2 and Erbium lasers are also used. YAG lasers may be used for precise excision or ablation, sometimes in combination with intralesional corticosteroid therapy.

DISCUSSION

The reason auricular keloids (Figure 2) have never been easy to treat is the inherent difference between the surgeon's ability to technically excise an external entity as opposed to our developing, but incomplete, understanding of a patient’s own biological healing response. Would excision, by itself, be considered an adequate therapy? As we know, the answer is no, especially when the patient is not treated effectively with even simple adjunctive modalities. Requiring excision alone to manage this kind of medical problems is akin to demanding successful weeding of a pumpkin patch, and in these instances, we predict recurrence as probable, but in all actuality, we can demand recurrence by ignoring the biology of the plant in its given natural soil conditions.[19] In this regard, the clinician cannot adopt a "paint by numbers approach, they can adopt a bespoke, multistep treatment regimen that considers lesion specific disease features, including size, history of recurrence and symptom aggravation, as well as patient directed risk tolerance, expected compliance and access to financial resources to treatment."

The management's art and science result from its gradual implementation. For a new, small earlobe keloid, a less aggressive, non-surgical intervention that involves serial intralesional corticosteroid injections would be a reasonable approach, or a minimal excision with immediate intraoperative and postoperative triamcinolone acetonide would result in excellent results with minimal morbidity. The substantial challenge that a large, recurring keloid poses, in a high-tension environment like the presternal region of a young, genetically predisposed patient, suggests a more assertive and definitive management plan. In complex contexts, surgical excision followed immediately by postoperative radiation is considered the cornerstone of therapy, creating the most significant reduction in potential recurrence.[20] The surgeon must act as a master conductor, having the technical ability to perform the resection and the intellectual insights to coordinate the timing, sequencing, and the aggressiveness of any adjunctive therapies (Figure 3).

We must remember that the immediate postoperative period, especially the first critical week, is a narrow therapeutic window when a recovering scar is most amenable to biological alteration, giving a fleeting opportunity to influence the healing toward an optimal aesthetic and functional outcome rather than toward fibrosis.

The future of managing keloids looks optimistic, largely due to the possible use of targeted biological agents. These agents come from emerging information about the biology of keloids. Newer modalities of mediation, such as topical or injected TGF-β inhibitors, interferon therapy, or the injection of botulinum toxin A to relieve skin tension, would signal a change towards the utilization of methods that aim to target underlying 'mechanisms'.[21]

These modalities have the potential to shift what we consider management from simply 'managing' keloids to a more exact manipulation of keloid biology. That being said, until there is larger validation through randomized controlled trials of the efficacy of any of these promising methods with adoption into a clinical pathway, the mainstay of effective keloid management remains the careful and appropriate use of one' currently available management options: the careful use of surgical procedures in combination with the right adjunctive evidence-based therapy.

Besides surgical and adjunct therapy, it is of utmost importance to include this education in an approachable manner, covering expectations and the time commitment to many keloid management protocols.[22] Successful treatment of auricular keloids is not seen as being driven by a single treatment of intervention; rather management is a continuing therapeutic collaboration involving a complex, multimodal approach to a chronic biological phenomenon.

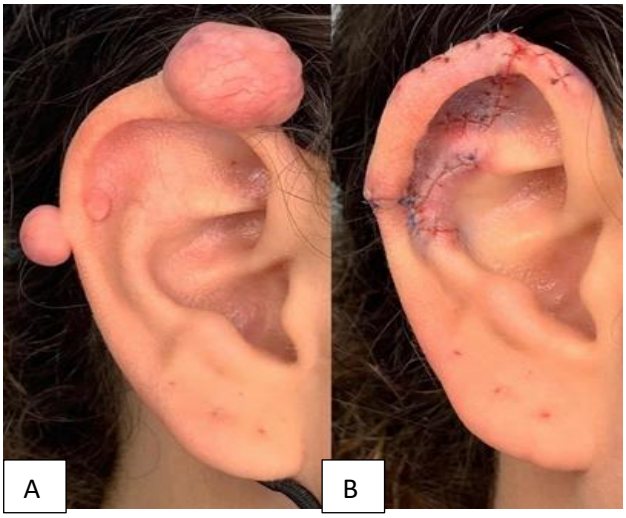

Figure 2 (A and B): Pre and post surgical resection, multiple auricular keloid.

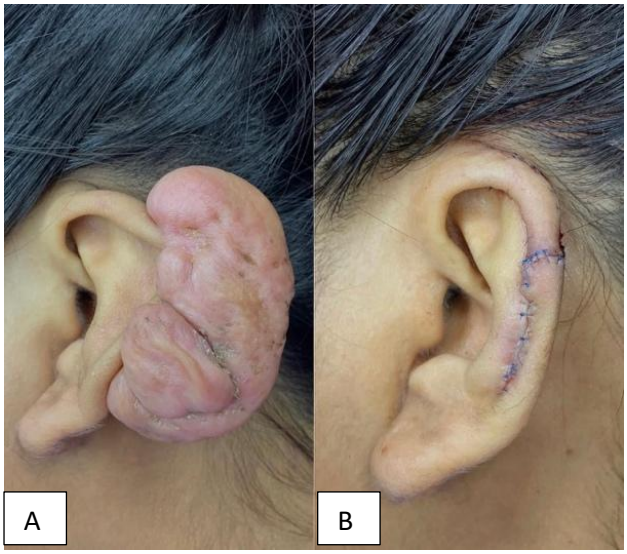

Figure 3 (A and B): Surgical resection, auricular helix reconstruction.

CONCLUSION

While surgical resection is an option, the body's inherent wound healing processes are beyond our control, recurrence following surgical intervention is a possibility, particularly in the absence of adjunctive non-surgical treatments, owing to the biological predisposition of keloids to reform. A personalized, multi-faceted therapeutic strategy is crucial for managing auricular keloids. A variety of treatment modalities are available. For a small, first-time earlobe keloid, corticosteroid injections or post-surgical injections may be a suitable approach. Conversely, the management of larger, recurrent keloids necessitates a more aggressive therapeutic intervention, especially in areas subject to high tension, such as the chest. Surgical excision, followed by radiotherapy, is the most effective way to prevent keloids from coming back. The surgeon must closely monitor the combined treatment. The healing process after surgery shapes the scar and helps it heal over several days. Other potential treatments include blocking growth factors like TGF-β or using botulinum toxin to reduce skin tension. Although these methods aren't widely used in clinical practice or well-studied, they could help treat keloid scars. Currently, the most reliable options are professional surgical procedures and established non-surgical therapies, along with patient education and monitoring. Recurrent keloids, especially those on the ear, require careful management.

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