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The Utility of Open-Book Enucleation of Keloids (OBEK), a Novel Procedure in Reducing Wound Burden in Tension-Prone Keloid Surgery

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Abstract

Keloids and hypertrophic scars are the overgrowth of scar tissue at the site of injury. Common keloid treatments such as surgical excision and corticosteroid administration yield unsatisfying results due to keloids exhibiting high recurrence rates. The authors propose a novel surgical procedure to treat keloids, called Open-Book Enucleation of Keloids (OBEK). In this new surgical technique, an incision within the keloid itself is made creating two skin flaps. The keloid nucleus is then excised, and the skin flaps are used to provide a tension-free closure. The authors treated a series of 25 keloids of the chest and/or shoulders with the OBEK procedure. The authors observed partial skin necrosis which ranged from none to twenty percent of the wound surface area. Patients treated with the OBEK procedure completely healed and showed no evidence of post-surgical keloid recurrence for as long as they were followed. In the authors' opinion, the OBEK procedure proved to be the optimal treatment for keloids, relieving both physicians and patients from the burden of post-surgical keloid recurrence and excessive adjuvant therapy.

Keywords: Platelet rich plasma • Androgenetic alopecia • Evaluation• Minoxidil

Introduction

Keloid scars and hypertrophic scars are a result of increased chronic inflammation in the reticular dermis which fails to subside due to the presence of internal/external stimuli. Along with the intensity, frequency and duration of the pro-inflammatory stimuli, a variety of local, systemic and genetic factors influence the development of keloids and hypertrophic scars.

There are a multitude of local factors that contribute to the development of keloidal and hypertrophic scarring. The first and most significant factor is local mechanical force. The diverse shapes of keloids indicate that their growth around the wound site depends on the direction of tension applied to the skin, as seen in horizontal contraction of the anterior chest wall and the development of horizontal keloids [1,2]. Clinical observations also show that keloids display a proclivity towards tension-prone locations on the body as opposed to areas where tension rarely occurs. Another local factor that promotes inflammation of the reticular dermis during wound healing is rewounding. The repetitive insertion and removal of earrings to and from earring holes, for instance, results in multiple injuries which dreadfully increases the risk of keloid formation at this specific site. Correspondingly, another local factor that contributes to pathological scar development is infection. In the case of acne and folliculitis, prolonged inflammation results in an increased risk of pathological scarring. Similarly, the duration of inflammation and the size of the wound may also contribute to the development of keloids. Burn wounds that healed in less than 10 days had a 4% chance of developing hypertrophic scarring whereas burn wounds that persisted longer than 21 days resulted in a 70% risk of developing hypertrophic scarring [3].

Adolescence and pregnancy display greater risks of developing keloids and hypertrophic scars. It is possible that sex hormones such as estrogen and androgen promote vasodilatation which amplifies the existing inflammation and enhances the development of new or existing pathological scars. This suggests that hypertrophic scar and keloid development is susceptible to hormone variations. Hypertension is also associated with the development of pathological scars. In a study which involved 304 consecutive patients with keloids, ordinal logistic regression analyses displayed a significant positive correlation between blood pressure and keloid size/number (both $p<0.0001$) [4]. This study provides epidemiological evidence that hypertension increases the severity keloid formation, which may be the result of damaged blood vessels and therefore, an increased inflammation in local tissue and at the wound site. Additionally, systemic inflammation is found to be responsible for keloid aggravation. Patients that undergo reconstructive surgery of extensive burns or injuries have higher levels of inflammatory cytokines that greatly increases the likelihood of developing pathological scars.

Patients who present with a family history of pathological scarring exhibit an increased risk of keloid and hypertrophic scar formation. Clinical evidence also demonstrates that patients with dark skin are 15 times more likely to develop keloids than patients with lighter skin [5]. Furthermore, pathological scars were found to be absent in albino patients [6]. We believe that Single Nucleotide Polymorphisms (SNPs) may be the genetic cause of pathological scar formation. In fact, a genome-wide association study distinguished 4 SNP loci in 3 different chromosomal regions that were positively linked to keloid development in the Japanese population [7]. Another study singled out one SNP (rs8032158) which displayed a significant correlation with keloid severity [8].

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From a clinical point of view, the distinction between keloids and hypertrophic scars lays in their boundaries; hypertrophic scars do not grow beyond the boundaries set by the original wound while keloids tend to originate from the wound, spreading into the surrounding normal skin.

From a pathological point of view, the distinction is drawn by the amount of thick hyalinizing collagen bundles, which are present in abundance in keloids but a lot less in hypertrophic scars. While keloids are characterized by abundance in hyalinized collagen and blood vessels, hypertrophic scars display low levels of hyalinized collagen and relatively few blood vessels. By comparison, we believe that hypertrophic scars and keloids result from the same fibroproliferative skin disorder but differ in the duration and the intensity of the inflammation.

Keloids often develop in areas of high-tension, such as the shoulders, chest, and torso; making it prohibitively difficult for a surgeon to reconstruct the defect in a tension-free space after surgical excision.

In the authors' clinical practices, we have developed a method of Open-Book Enucleation of Keloids to reduce keloid volume and promote a tension-free closure after surgical excision. This method facilitates 1) near-complete surgical excision of keloids, 2) tension-free closure of the wound, and 3) reduces the need for post-excision reconstruction with skin grafting or flap closure.

Methods

Unlike traditional keloid surgery which removes the entire surface footprint of a keloid during excision, the OBEK procedure uses an incisional approach within the keloid itself to deepithelialize and separate the overlying skin of the keloid from the keloid core. Once skin flaps are created circumferentially with sharp excision, the keloid nucleus is surgically excised. The resulting skin flaps are then used to 'close the book' and provide tension-free closure.

Bolster sutures are then used to compress the keloid upon the wound floor in order to facilitate adherence and tissue neovascularization of the overlying skin.

In essence, the perfused skin of the keloid is used, rather than being discarded as specimen, in order to reconstruct the defect. In the authors' opinion, the keloid skin provides for satisfactory closure of the wound and is suitable for long- term reconstruction. Minimal adjuvant therapy was necessary in order to reach optimal results. Post-surgical treatments included corticosteroid administration as well as radiotherapy.

Results

A series of 25 consecutive patients with centrally-located keloids on the chest and/or shoulders were evaluated. The mean surface area of the initial keloids was 5.8 cm^2 (Range 4.1 cm^2 to 9.4 cm^2). Mean time to reepithelialization and complete wound closure was 12 days (Range 4 to 26 days). Partial skin necrosis overlying the wound repair was common and ranged from none to twenty percent of the wound surface area. These areas completely reepithelialized within several weeks of surgery.

Discussion

Certain surgical methods reduce the risk of post-surgical reoccurrence of keloids. As opposed to skin grafts, local flaps tend to extend naturally post-surgery, thereby avoiding post-surgical contractures. Similarly, Z-plasties followed by zig-zag sutures significantly reduce the risk of keloid recurrence. Segmented scars tend to heal better and faster than a single linear scar and reduce the risk of keloid reoccurrence, especially in the case of a scar that crosses a joint. The choice of sutures is also crucial in reducing the tension in the wound area. Is it important to note that dermal sutures do not alleviate the tension on the dermis? For this reason, deeper structures in the hypodermic area need to be sutured, in order to elevate the wound edges while placing minimal tension on the reticular layer (Figure 1).

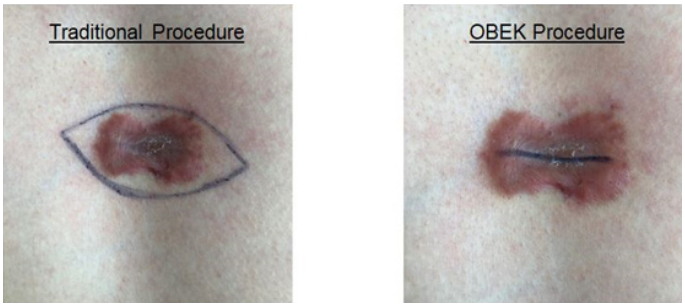

Figure 1. Minimal adjuvant therapy was necessary in order to reach optimal results. Post-surgical treatments included corticosteroid administration as well as radiotherapy.

Radiotherapy has proven to be very effective in treating keloids. While radiotherapy is mainly considered to suppress fibroblast activity, it also suppresses angiogenesis. When patients with severe keloids are subjected to radiation therapy, their skin color improves instantly and the skin flattens out progressively. This is indicative of both angiogenesis suppression due to the reduced blood vessels numbers which in turn lead to a reduction in inflammation, as well as fibroblast activity suppression.

Corticosteroids suppress the inflammatory response and may also induce decreased blood flow to the wound *via* vasoconstriction. In fact, upon administration of corticosteroids, keloids whitened. This whitening was also accompanied by a reduction in pain and itching.

Compression therapy can compress the blood vessel in the scar, reducing the process of inflammation. Stabilizing therapy permits the stabilization of wounds with mechanical supports such as tapes or sheets, thus reducing the inflammatory process of the reticular dermis. Laser therapy reduces the number of blood vessels at the site of the wound and reduces the inflammatory response. 5-FU therapy, which functions by blocking angiogenesis, has also been successful at treating keloids [9]. Finally, cryotherapy has also been used to treat keloids in combination with intralesional triamcinolone injection [10].

Conclusion

The management of centrally located keloids on the body often involves a combination of treatment modalities, including surgical extirpation, and non-surgical laser therapy, radiation therapy, or intralesional injections. Surgical decision making depends on the feasibility of surgical removal and the options available for reconstruction of the wound. Skin grafting or soft-tissue flaps place a keloid patient at additional risk of developing keloids at the harvest site. The OBEK procedure allows surgeons the option of near-complete excision of a keloid while minimizing the post-surgical demands for reconstruction. In the authors' opinion, the preservation of host keloid skin provides an excellent biologic dressing to facilitate wound healing in keloid surgery and completely prevents post-surgical recurrence of keloids when combined with minimal adjuvant therapy.

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