An Adolescent's Acne-Related Lesions: Hypertrophic Scars or Keloids?

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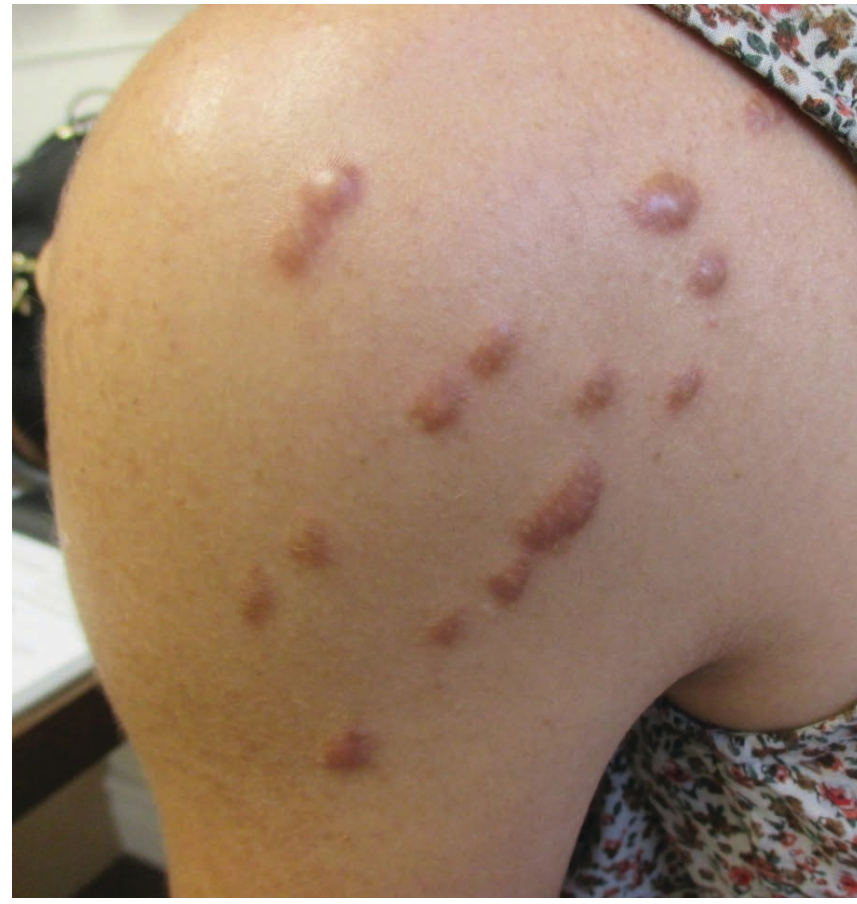
A 17-year-old Asian adolescent girl presented with disfiguring lesions on her shoulders and upper back. The lesions had been first noticed 2 years ago, and they had grown very slowly until reaching their present size a year ago. The lesions are at times pruritic. The patient had had severe acne vulgaris starting at the age of 13. She had been seen by her family physician and had been treated with various topical keratolytics and bactericidals without much success. Her past health was unremarkable. Her 15-year-old and 13-year-old brothers also had severe acne.

On physical examination, multiple, erythematous, well-demarcated nodules were present on the shoulders and upper back. The nodules were smooth, shiny, firm, and nontender. She also had acne and atrophic scars ("ice pick" scars and "boxcar" scars) on the face. The rest of the physical examination findings were unremarkable.



Are these acne-related lesions hypertrophic scars, or are they keloids?

Answer: Keloids

A clinical diagnosis of keloids secondary to previous acne was made. The patient's dermatologist prescribed oral isotretinoin and oral contraceptives in an attempt to clear up her remaining acne so that she did not continue to develop new keloids. The keloids were treated concurrently with a monthly combination of liquid nitrogen cryotherapy and intralesional triamcinolone acetonide, 40 mg/mL, with nice results. The patient is considering laser therapy for the remaining flattened and erythematous scars.

The term *keloid* derives from the Greek word for crab claw, which describes the lateral growth of tissue into the unaffected skin.[1] Keloids are a benign fibroproliferative skin disorder characterized by elevated fibrous scars that extend beyond the borders of the original wound and invade into surrounding normal skin.[2] Keloidal scarring is unique to humans.[3]

EPIDEMIOLOGY

It has been estimated that 4.5% to 16% of the black, Hispanic, and Asian populations develop keloids.[4-6] The condition is 15 to 20 times more prevalent in blacks than in whites.[1,7,8] Keloids do not occur in persons with albinism.[3,8] The peak incidence is between 10 and 30 years of age.[4] Both sexes are equally affected.[4,5,9] Most cases occur sporadically, although some cases are familial.[2,10]

ETIOLOGY

Keloids may arise following any insult to the deep dermis. Acne, as illustrated in the present case, is a very common cause. Other commonly reported causes include lacerations, abrasions, surgical wounds, folliculitis, chickenpox, burns, earlobe piercing, tattooing, insect bites, and vaccinations (particularly the bacillus Calmette-Guérin vaccination).[2,4,5] "Spontaneous" keloid might be a result of an overlooked minor skin trauma.

Keloids may arise after any deep dermal insult, including acne, lacerations, folliculitis, chickenpox, burns, piercings, tattoos, and vaccinations.

There is a genetic predisposition to the development of keloids as evidenced by the increased prevalence in dark-skinned races, familial clustering, and increased concordance among identical twins.[6,8] The susceptibility to keloid formation likely is polygenic. An autosomal dominant mode of inheritance with incomplete penetrance and variable expressivity also has been described.[2,8] In some families, chromosomal keloid susceptibility loci have been mapped to 1q41, 2q23, 3q22.3-23, 7p11, 14q22-23, and 15q21.2-22.3.[8-10] Syndromes associated with keloid formation include Rubinstein-Taybi syndrome (facial abnormalities, brachydactyly, mental retardation, keloid formation, capillary malformations, pilomatricomas) and Goeminne syndrome (congenital muscular torticollis, cryptorchidism, renal dysplasia, multiple keloids).[2]

PATHOGENESIS

It is generally believed that keloids result from a more prolonged inflammatory phase during wound healing.[7] The development of a Th2 response is linked to fibrogenesis in keloids.[7] It has been shown that keloid fibroblasts fail to undergo physiologically programmed apoptosis and continue to produce connective tissue beyond the period expected for normal scars.[8,11] These fibroblasts have increased levels receptors for vascular endothelial growth factor, transforming growth factor-β, insulinlike growth factor, and platelet-derived growth factor-α and respond more briskly to these growth factors.[2,8,12,13] Keloid fibroblasts have a greater capacity to proliferate and produce an increased amount of collagen in an autonomous fashion.[12]

Hypoxia may be responsible for the propagation of keloids.[12] Occlusion of the microcirculation within the lesions due to endothelial proliferation and perivascular myofibroblast contraction results in hypoxia. Moreover, the perivascular myofibroblast contraction might stimulate endothelial hyperplasia and cause further hypoxia, which would lead to excess collagen production.

HISTOPATHOLOGY

Histologically, keloids are characterized by pale-staining, hyalinized, and hypocellular collagen bundles; a tongue-like advancing edge underneath normal-appearing epidermis and papillary dermis; horizontal cellular fibrous bands in the upper reticular dermis; and prominent fascia-like fibrous bands.[6,9] The collagen fibers are larger, thicker, and wavier than those found in hypertrophic or normal scars and are arranged into thick collagen bundles.[4,12] These collagen fibers have a random orientation and are arranged irregularly, whereas those in hypertrophic scars orient parallel to the epidermal surface.[4,12]

CLINICAL MANIFESTATIONS

Keloids are elevated fibrous scars that usually develop within several months after the initial scar.[14] Characteristically, keloids outgrow the boundaries of the original scar, invading surrounding normal tissue.[9,14] They typically grow with time and then stabilize.[3] Keloids rarely regress.[9]

Clinically, keloids present as firm and rubbery nodules that can be flesh- or skin-colored, hypopigmented, or reddish brown (most common).[2] They have a shiny appearance and at certain locations may take on the shapes of a dumbbell (eg, on the earlobe) or a butterfly (eg, on the chest). The lesions often are pruritic and/or painful.[1,7,9] The sites of predilection include the anterior chest, shoulders, back of the neck, jawline, upper back, upper arms, and earlobes.[1,2] The palms and soles are rarely affected.[2,5,7]

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The diagnosis of keloids is mainly clinical. Keloids should be differentiated from hypertrophic scars, which remain within the confines of the original scar border.[4,7] Hypertrophic scars generally arise within 4 weeks after the dermal insult, grow intensely for several months, and then regress.[4] Hypertrophic scars are less likely to be symptomatic. Other differential diagnoses include dermatofibroma, dermatofibrosarcoma protuberans, epidermoid cyst, desmoid tumor, and foreign body granuloma.[4]

COMPLICATIONS

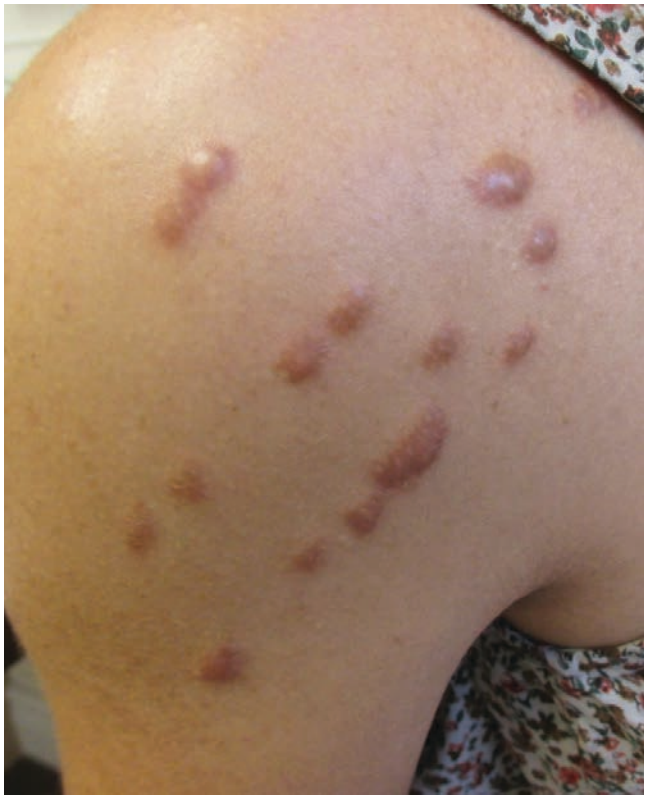
The lesion can be aesthetically unappealing and may adversely affect the patient's quality of life by causing physical and psychological impairment.[15]

PREVENTION

Patients at risk for keloid formation should be advised to consider avoiding contact sports and nonessential cosmetic surgery, particularly in high-risk areas of the skin.[3] If ears are pierced in spite of this advice, pressure earrings can be used to reduce the risk of keloid formation. Dermatologic conditions such as acne vulgaris, folliculitis, and furuncles should be treated aggressively and referral to a dermatologist should be considered in the presence of keloids or other acne scarring.[11] If surgery cannot be avoided in susceptible patients, the wound should be closed with minimal tension.[6] Application of silicone gel sheeting or use of topical imiquimod, use of a pressure garment, and corticosteroid injections are effective prophylactic measures.[1,2,6] Silicone sheets should be avoided on open wounds but can be applied as soon as the skin heals.[1] Semiocclusive dressings for minor abrasions should be considered.

MANAGEMENT

Treatment often is requested to improve cosmesis as well as to resolve pruritus and/or discomfort. The most common treatment modalities include intralesional corticosteroid injection, surgical excision, cryotherapy, and silicon therapy.[1,2] Other treatment options include potent topical corticosteroids, intralesional 5-fluorouracil (5-FU) or bleomycin, topical imiquimod cream, laser therapy, and radiotherapy.[8,16] Often



multimodality therapy is employed, such as liquid nitrogen followed by intralesional steroids, followed by silicone gel sheeting and/or laser therapy.

Intralesional corticosteroid injections are the most effective and widely used treatment for keloids.[4,9,11,12] Corticosteroids work by inhibiting fibroblasts, glycosaminoglycan, and collagen synthesis, increasing collagenase production, and reducing levels of collagenase inhibitors.[9,12] The success rates range from 50% to 100%; higher success rates can be achieved when treating young, proliferative scars.[9] Intralesional corticosteroid injections should be given every 4 to 6 weeks by an experienced injector until the keloid is flat.[16] Because the injections can be painful, pretreatment with a topical anesthetic cream such as eutectic mixture of local anesthetics or the addition of lidocaine (xylocaine) or taking a nonsteroidal anti-inflammatory drug 1 hour prior to treatment may be necessary.[2,3] Complications of intralesional corticosteroids include atrophy of subcutaneous tissue and fat, skin necrosis, hypopigmentation, hyperpigmentation, telangiectasia, and cushingoid features, but these are uncommon in experienced hands.[2,9,12]

If treatment with intralesional corticosteroid monotherapy is not successful, the next option is to consider adding liquid nitrogen cryotherapy prior to corticosteroid injection, or a combination of corticosteroid and 5-FU or bleomycin injections. If these measures result in an insufficient response, surgical excision with preoperative, intraoperative, and postoperative corticosteroid injections, as well as pressure dressing if applicable, can be considered.[1,11] Surgical excision alone has a high recurrence rate and often leads to a longer scar and the potential for a larger keloid.[2,3,11]

Cryotherapy can be used for small lesions.[2,11] Cryotherapy works by inducing vascular damage and tissue necrosis.[15] Multiple treatments often are necessary. Complications can include pain, skin atrophy, and hypopigmentation.[6] The latter is more common in darker-skinned individuals and thus should be employed carefully by clinicians who are experienced in the treatment of this population.[2,6] Cryotherapy can be combined with intralesional corticosteroid injection for enhanced results.[2]

Silicone gel and silicone sheets have been used to soften and decrease the size of keloids.[2] These products work by acting as an impermeable membrane, thereby increasing the temperature and hydration of the occluded keloid.[1] ■

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