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Keloid Formation in Scars Secondary to Measles Exanthem Managed with Surgery followed by Adjuvant Radiation Therapy

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Abstract

Background

Keloids are benign fibroproliferative dermal tumors that arise from dysregulated wound healing, with a higher prevalence in individuals with darker skin phototypes. They typically develop at sites of trauma including surgical wounds, acne, piercings, burns, or vaccination sites. Keloid formation in measles rash scars has not been previously described in the literature.

Case Presentation

A 33-year-old African American female presented with multiple extensive keloids (the largest measuring ~10 cm) distributed across the posterior thorax, neck, anterior chest, breasts and upper extremities. The lesions emerged approximately six years prior, specifically localized to areas of previous scars from measles rash. Management involved a multidisciplinary approach; surgical resection with adjuvant radiotherapy was recommended due to the high risk of recurrence.

Conclusion

This case identifies a novel association between measles-induced cutaneous inflammation and subsequent keloidogenesis. Understanding this potential trigger is essential for clinicians to optimize early intervention and management strategies.

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Introduction

Keloids are pathological scars characterized by exuberant fibroblast proliferation and excessive extracellular matrix deposition, primarily type III collagen, extending beyond the boundaries of the original wound [1]. Unlike hypertrophic scars, keloids do not regress spontaneously and can be symptomatic (itching, pain) or cause functional and cosmetic impairment.

The etiopathogenesis of keloids remains elusive, but it is understood to involve abnormal wound healing with persistent inflammation, fibroblast dysregulation, and aberrant collagen synthesis [1]. Genetic predisposition plays a significant role, with higher prevalence in individuals of African, Hispanic, or Asian descent. Established triggers include surgical incisions, lacerations, acne, burns, body piercings, and intramuscular or subcutaneous vaccinations (e.g., BCG, smallpox); however, keloidogenesis secondary to a measles viral exanthem remains unreported. There are reports of keloids after chickepox [2]. This report describes a unique case of widespread keloids arising specifically within the distribution of a prior measles rash.

Case Report

A 33-year-old African American female presented to the dermatology clinic with complaints of multiple raised scars on her chest, neck, arms, and breasts that had progressively enlarged over the past six years. She denied significant past medical history, allergies, or familial history of keloids. The patient reported an episode of measles at age 27, which was confirmed clinically by a characteristic rash and systemic symptoms. She recalled dark marks in the areas where the rash had been most intense. Over the ensuing months and years, these areas began to develop firm, raised scar tissue. She denied any intervening trauma, surgery, or procedures in these regions. The largest keloid, located on her back, was approximately 10 cm in its greatest diameter (Figure 1). Smaller lesions were noted on the right lateral neck, anterior chest, breasts, and bilateral arms, corresponding precisely to the distribution of her prior rash (Figure 2). Significant pain and hyperesthesia associated with the posterior keloids rendered lying down difficult; additionally, the patient noted pruritus in the majority of the lesions. She also reported significant cosmetic distress impacting her quality of life.

Figure 1: Multiple keloids on the back of chest

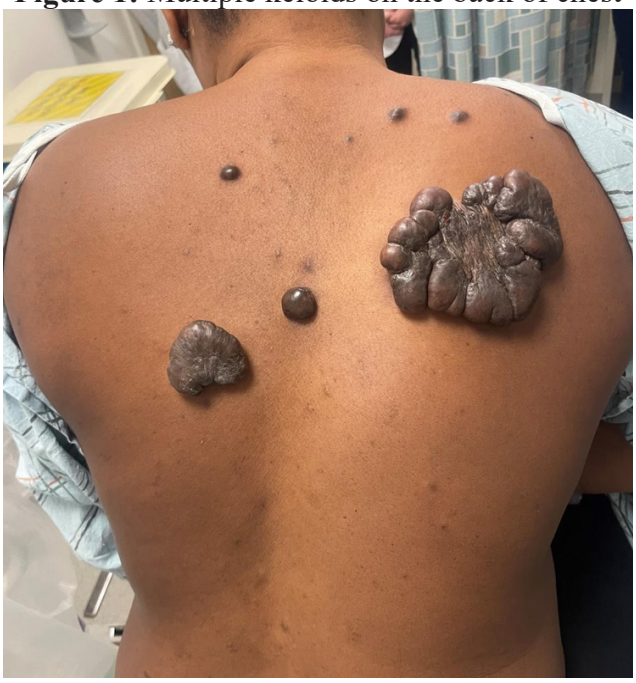
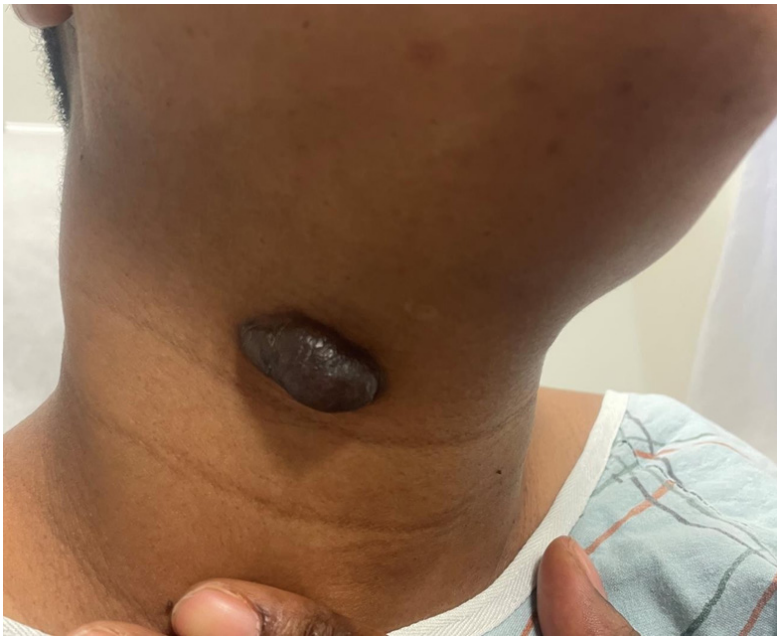


Figure 2: Keloid on right Antero-lateral neck



Examination revealed multiple hypertrophic, firm, rubbery nodules consistent with keloids. Lesions extended way beyond the margins of the initial rash sites with variable thickness and surface irregularity. The largest lesion gave a foul smell and a very small area of ulceration as shown in Figure 1. However, no signs of infection were present. Diagnosis was made clinically given the typical appearance, distribution, and history.

Given the extent of the lesions, a multimodal treatment plan was initiated. The patient was evaluated by Plastic Surgery. Given the size of the lesions and high risk of recurrence, surgical excision was planned. She was referred to Radiation Oncology to discuss adjuvant radiation therapy (RT) post-excision, recognizing evidence that RT can reduce recurrence. Laser therapy and corticosteroid intralesional injections were also reviewed as adjuncts. A personalized multimodal plan is in development.

Discussion

Keloid formation represents a fundamental failure of the physiological wound-healing "offswitch," characterized by a dysregulated cytokine profile involving transforming growth factorbeta (TGF-β) and matrix metalloproteinases. While mechanical tension and physical trauma are established precursors to keloidogenesis, this case underscores the potential for the profound systemic inflammatory response associated with a measles exanthem to serve as a sufficient stimulus in genetically predisposed individuals. We propose that the cutaneous sequelae of measles may provide the requisite inflammatory nidus to trigger aberrant wound-healing cascades and subsequent keloid development, thereby expanding the understood etiology of pathological scarring beyond localized cutaneous injury.

The observed multi-year latency period in this patient aligns with the typical progression of keloid formation, wherein chronic, subclinical inflammation gradually manifests as palpable dermal hypertrophy [3]. This temporal progression is particularly significant, as it suggests that diffuse inflammatory processes associated with viral exanthems may initiate keloidogenesis in a manner analogous to focal trauma. Consequently, this case highlights the clinical importance of monitoring patients with systemic inflammatory dermatoses for long-term fibroproliferative complications.

Management of extensive keloids remains a formidable clinical challenge, with current evidencebased protocols indicating that monotherapy yields recurrence rates as high as 45–100%. To address this, a multimodal approach consisting of surgical resection followed by adjuvant radiotherapy—as planned for this patient—is utilized to reduce recurrence rates to below 20% while providing durable symptomatic

relief. The efficacy of radiotherapy in this context is driven by DNA-induced inhibition of fibroblast proliferation, attenuation of angiogenesis via endothelial radiosensitivity, and the modulation of gene expression pathways related to extracellular matrix organization. These collective biological responses suppress cell viability, induce apoptosis, and reduce collagen production; however, it should be noted that optimal radiation modalities, dosages, and fractionation schedules continue to vary across institutions [4].

Conclusion

This report documents the first known case of keloids originating from measles rash scars. It underscores the necessity of considering severe viral exanthems as potential triggers for keloid formation in high-risk populations. Clinicians should remain vigilant in monitoring patients with darker skin types following severe inflammatory cutaneous events to facilitate early therapeutic intervention.

Author Responsible for Statistical Analysis: No Statistics involved

Conflict of Interest: None

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