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Successful treatment of disseminated post-burn pyogenic granuloma with systemic corticosteroid: a case report

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Consent for publication: informed consent for publication of the images and all relevant information was obtained from the child's parents.

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Abstract

Pyogenic granuloma (PG), also known as lobular capillary hemangioma, is extremely rare following burn injuries and there is no conclusive theory about its pathogenesis and best treatment option. Here we report a brief case of a 2-year-old child with a third-degree burn who developed disseminated pyogenic granuloma and was successfully treated with systemic corticosteroid.

Introduction

Pyogenic granuloma (PG), also known as lobular capillary hemangioma, is a non-neoplastic, inflammatory hyperplasia of the skin and oral mucosa, often caused by local irritation, traumatic injury, or hormonal factors.[1] PGs are fragile and bleed easily. The most common sites of their occurrence are the face and distal extremities, possibly due to the higher incidence of minor trauma at these locations.[2]

Trauma is a well-established cause of PG's manifestation, However, the eruptive, disseminated post-burn form of PG, which is regarded as an abrupt development of angiomatous papules secondary to burn, is extremely rare, especially when the entire burn population is considered. In addition, the exact etiology remains ambiguous, and treatment is still controversial.[3]

Herein, we report a case of generalized eruptive PG in an infant following a third-degree burn with hot water that was treated with systemic corticosteroid.

Case Report

A 2-year-old girl was referred to our department (Imam Khomeini Hospital; Referral Center for the Treatment of Skin Diseases, Tehran, Iran) with multiple papillomatous and nodular lesions on her face and body. The patient had sustained a third-degree burn covering 40% of her body due to boiling water 2 months prior to her consultation. She had been followed at another facility, where daily dressings with silver sulfadiazine were applied, and the burned skin on her forearms and chest was successfully repaired with a full-thickness skin graft from her thigh. Three weeks after the scald injury, she gradually developed numerous reddish-brown bumps, which appeared not only on the burn site but also on intact skin and the donor site on her thigh (Figure 1).

During the visit, she was restless and febrile, prompting her admission for diagnostic and therapeutic measures. Laboratory investigations, including a complete blood count, comprehensive metabolic panel, and an HIV test, were unremarkable. Blood and tissue cultures were positive for Pseudomonas aeruginosa. The patient was treated with gentamicin (2.5 mg/kg/dose every 6 hours) and meropenem (20 mg/kg every 8 hours). Subsequent cultures became negative and fever subsided; however, the

lesions continued to progress. Therefore, corticosteroid therapy was initiated after culture turned negative and fever resolved, considering the inflammatory nature of the lesions.

A biopsy was performed, and histopathological examination revealed a lobular pattern of benign vascular proliferation with hyperkeratosis, dermal edema, and significant inflammatory cell infiltration (mostly lymphocytes), suggestive of PG (Figure 2). Immunohistochemistry was not performed due to the lack of facilities in the city where the patient was hospitalized and the family's inability to afford testing in another city. However, pathology samples were reviewed by three pathologists, including one dermatopathologist.

The lesions were unresponsive to conservative treatment, and their number increased daily. Due to the extent of the lesions, surgical and other ablative treatments were not feasible. Prednisolone was initiated at a dose of 1 mg/kg. There was a dramatic response following prednisolone administration, resulting in the cessation of new lesions and the healing of existing ones (Figure 3).

Prednisolone was gradually tapered over 2 months, and no recurrence was observed during a 6-month follow-up period.

Informed consent for publication of the images and all relevant information was obtained from the child's parents.

Discussion

PG is a common acquired vascular tumor, particularly in the pediatric population, characterized by an abnormal proliferation of capillaries at the site of trauma. While infections may coexist with PG, it is essential to recognize that PG itself is not an infectious process.[4]

Recent literature indicates a potential genetic predisposition linked to abnormalities in the *FLT4* gene, which encodes a tyrosine kinase receptor involved in pathological angiogenesis. This suggests that PG is a reactive lesion resulting from tissue injury and subsequent impaired wound healing.[5]

The development of PG following burn injuries is exceedingly rare, and the precise etiology remains inconclusive. Most reported cases have been in children, with a slight female predominance.[6,7] Notably, many cases originate from regions such as China, Iran, and Turkey, indicating a possible ethnic susceptibility.[8] The onset of PG typically occurs between 1 to 4 weeks post-burn injury, aligning with the timeline observed in our patient, whose lesions manifested approximately 3 weeks after her scald injury.[9]

Interestingly, while most reported cases of PG arise at the site of trauma, our patient exhibited lesions not only on the burned skin but also on unaffected areas and the donor site. Disseminated post-burn PG,

however, is extremely rare. Lesions at distant uninvolved sites and donor graft areas have only rarely reported in the literature. The progressive increase in the number of lesions without improvement raises the possibility of a systemic process contributing to their development.

Various treatment modalities for PG have been proposed, including topical and systemic therapies such as cryotherapy, curettage, cauterization, pulsed-dye laser therapy, and surgical excision.[7,9,10] Conservative and surgical approaches tend to yield favorable outcomes; however, in our case topical therapies were not used due to the high risk of systemic absorption in the setting of extensive lesions. Ablative therapies were not feasible because of lesion distribution (including the face and graft/donor sites), their painful nature, the risk of disfigurement, and the need for general anesthesia in a young child. Therefore, we initiated treatment with prednisolone at a dosage of 1 mg/kg. The patient was monitored by a pediatric specialist with respect to growth parameters and potential treatment-related adverse effects. This approach led to a remarkable improvement, with the cessation of new lesions and healing of existing ones. After cessation of new lesion formation and partial improvement (approximately 50% reduction in the number and size of existing lesions), corticosteroids were tapered over 2 months.

The initiation of corticosteroid therapy with prednisolone proved to be pivotal in managing the patient's condition. The dramatic response observed following treatment suggests that the underlying mechanism driving the lesion proliferation was likely inflammatory in nature. Corticosteroids are known to exert anti-inflammatory effects and can modulate the immune response, which may have contributed to halting the progression of new lesions and promoting healing of existing ones.[11]

This case also raises questions about the management of similar cases in pediatric populations. The challenges associated with ablative therapies in young children, particularly when lesions are widespread and involve sensitive areas like the face, necessitate a careful consideration of non-invasive treatments. This case report highlights systemic corticosteroids as an effective treatment option for managing severe cases; however, it also emphasizes the need for ongoing monitoring to ensure long-term stability.

Conclusions

In conclusion, this case underscores the need for further research into the mechanisms underlying post-burn PG development and emphasizes the importance of considering systemic inflammation in its management. Future studies could explore genetic factors and inflammatory pathways involved in PG formation, potentially leading to targeted therapies for affected individuals.

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Figure 1. Bright red papules mainly on face and extremities of the patient.

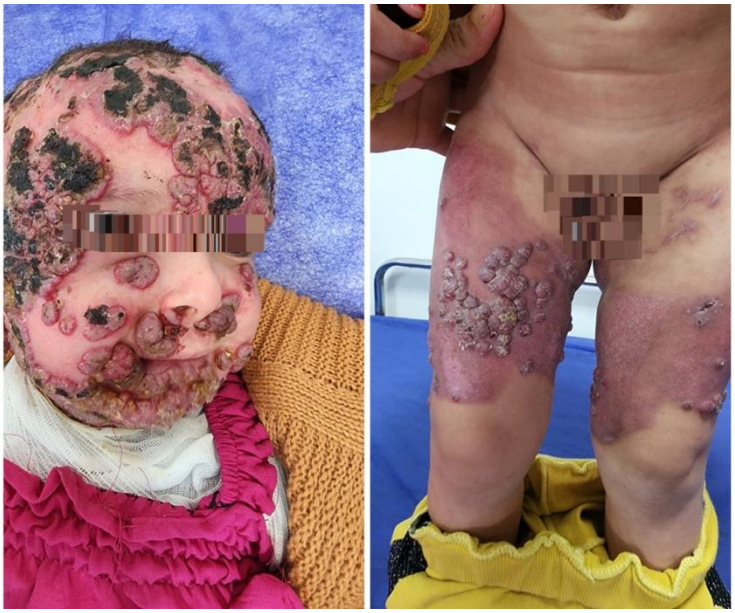

Figure 2. Low power showing **A**) epidermal hyperplasia with new vascularization and dermal infiltrate (hematoxylin and eosin ×4) and **B**) multiple branching blood vessels with dermal infiltrates consisting mainly lymphocytes (hematoxylin and eosin ×40).

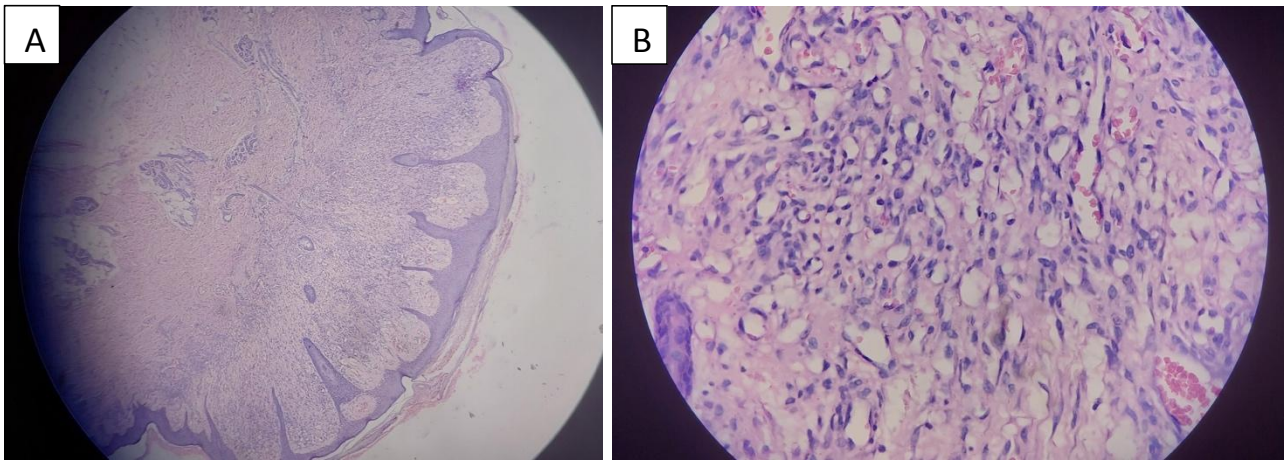

Figure 3. The patient after treatment with systemic corticosteroid. Healing of previous lesions is evident.