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Psoriasiform rash possibly induced by oral propranolol in a 12-month-old girl with infantile hemangioma: a case report and literature review

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Abstract

Infantile hemangiomas are the most common soft-tissue tumors in children, with propranolol, a non-cardioselective β-blocker, considered the first-line treatment for complicated cases. β-blockers have been reported to be the most common causative agents for drug-induced psoriasis in adults. In the pediatric population, only one previous case exists. We report the case of a 12-month-old girl who developed a psoriasiform rash after starting oral propranolol for infantile hemangiomas on the scalp. The patient had no personal or family history of psoriasis, and the rash appeared one week post-initiation of propranolol, presenting as well-defined erythematous, scaly plaques over the body, including the scalp. Infectious causes were excluded, and the rash was diagnosed as a psoriasiform rash, possibly induced by oral propranolol. The patient was switched to atenolol, which resulted in improvement of the hemangioma and complete resolution of the skin lesions. This case highlights the rare but significant risk of psoriasiform eruptions associated with β-blocker therapy in infants, emphasizing the need for careful recognition and monitoring of this potential adverse effect in pediatric patients treated for infantile hemangiomas.

Introduction

Infantile hemangiomas are the most common soft-tissue tumors of childhood, with the incidence in the general population being approximately 5%. Propranolol is a non-cardioselective β-blocking drug that is widely considered the first-line treatment for complicated infantile hemangiomas, which are associated with functional impairment, pain, bleeding, or disfigurement. Potential adverse effects of propranolol include bronchospasm, bradycardia, hypotension, hypoglycemia, sleep disturbance, and discoloration with cooling of the hands and feet.[1] Dermatologic side effects include triggering and exacerbation of psoriasis, contact dermatitis, occupational contact dermatitis, Raynaud's disease, alopecia, and lichen planus-like drug eruption.[2] However, these dermatologic reactions have primarily been documented in the adult population, with limited reports in pediatric cases. Herein, we describe the case of a 12-month-old girl, with no personal or family history of psoriasis, who developed a generalized psoriasiform rash, possibly induced by oral propranolol therapy for infantile hemangioma of the scalp.

Case Report

A 12-month-old girl presented to the pediatric dermatology department for the evaluation of an infantile hemangioma over the scalp present since 3 months of age. The hemangioma was a 2.5 x 2 cm solitary, deep ulcerated red nodule with erosions over the frontal scalp area, associated with on and off bleeding (Figure 1). The parents reported that the infant was previously started on oral propranolol, and one week after starting the medication, she noticed a widespread rash eruption. The rash was an itchy, generalized, well-defined erythematous scaly patches and plaques over the body and skin folds with scalp involvement (Figure 2). These lesions were consistent with a psoriasiform eruption. Other potential causes of the rash, such as infectious triggers (*e.g.*, streptococcal infections), were considered but were denied by the parents, who reported no history of recent upper respiratory infections or other symptoms suggestive of an infectious etiology. The patient had no personal or family history of psoriasis. On examination, the patient had hypopigmented macules and patches over the trunk, likely representing post-inflammatory hypopigmentation. Therefore, propranolol-induced psoriasiform eruption, possibly induced by oral propranolol, was diagnosed. The patient was switched to oral atenolol as the hemangioma was still cosmetically concerning and associated with occasional bleeding. Atenolol was continued with the improvement of the hemangioma size and no recurrence of the psoriasiform eruption.

Discussion

Psoriasis is a chronic, immune-mediated, inflammatory skin disease with a well-known genetic predisposition. The prevalence of the disease in children varies among the medical literature depending on the study population and age, with 1% as a commonly quoted figure.[3,4] Although psoriasis can develop at any age, one-third of cases begin in childhood, with the median age of onset ranging from 7 to 10 years.[5,6] Precipitating factors such as trauma, infections (*e.g.*, streptococcal pharyngitis or perianal streptococcal dermatitis), stress, and drugs are more common in pediatric psoriasis than in adult-onset psoriasis.[7,8]

Beta-blockers are now considered to be the first-line systemic therapy for medically complex and cosmetically significant infantile hemangioma cases.[1] They are also widely used drugs that are strongly linked to the development of psoriasis. They are known to exacerbate pre-existing

psoriasis and precipitate it *de novo* in adults.[9] Several authors since the 1980s from various parts of the world have reported eruptions following cardioselective and non-cardioselective β-blockers such as propranolol, metoprolol, atenolol, cetamolol, nadolol, and even topically used timolol. The eruptions vary from pustular psoriasis all the way to erythroderma. The latency period between drug intake and onset of eruption may range from weeks to as long as 12 months.[9,10] The pathogenesis involves the cyclic adenosine monophosphate (cAMP) pathway. cAMP acts as a control signal for cellular differentiation, and a blockade of the intracellular beta 2 receptors by β-blockers causes a decrease in cAMP, leading to an increase in epidermal cell turnover as seen in psoriasis. Additionally, β-blockers lead to excessive release of cytokines from macrophages and neutrophils, further exacerbating the psoriatic condition.[11]

Few drugs have been associated with the development of psoriasis or psoriasiform eruptions in the pediatric population. These agents include tumor necrosis factor-alpha inhibitors,[12] imiquimod,[13] growth hormone therapy,[14] and rituximab.[15] In the literature, there has been only one case of a psoriasiform eruption due to β-blockers: an 18-month-old child developing psoriasiform diaper rash linked to the intake of oral propranolol for infantile hemangioma.[16] In contrast to our patient, who developed the rash one week after beginning propranolol therapy, this child developed it six weeks later. Both patients had no family history of psoriasis and were using oral propranolol for infantile hemangioma when they developed the psoriasiform diaper rash. Our patient additionally developed axillary, scalp, and truncal involvement.

In 1990, 3 cases of atenolol-induced pustular psoriasis were reported in adult patients.[17] Our patient, at the time of writing this article, reports no eruptions with atenolol. This finding could possibly be explained by the selectivity of atenolol compared to the non-cardioselective selective propranolol. However, it is important to consider the cross-reactivity between propranolol, oxprenolol, and atenolol. As a result, every possible β-blocker can probably provoke, aggravate, and induce psoriasiform eruptions.[10]

Conclusions

β-blockers are now considered to be the first-line systemic therapy for complicated cases of infantile hemangioma. Therefore, in recent years, its use has expanded in the pediatric population. We report here the first case of a generalized psoriasiform rash developing after propranolol

therapy in a child with infantile hemangioma. For physicians, awareness of this potential adverse event will facilitate its early recognition and prompt treatment.

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Figure 1. A 2.5 x 2 cm solitary deep ulcerated red nodule with erosions over the frontal scalp.

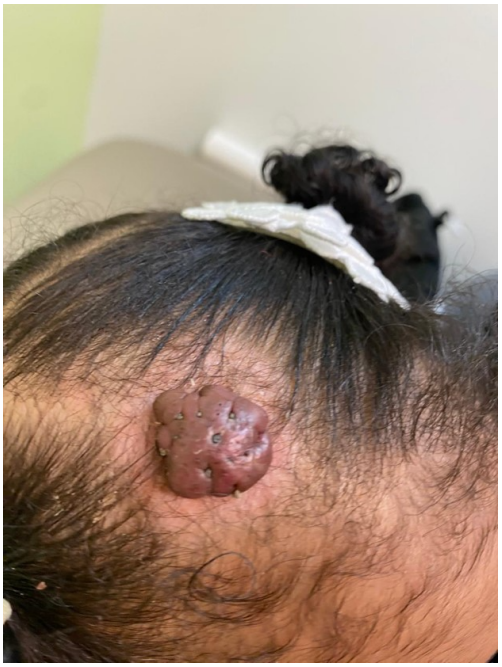

Figure 2. Psoriasiform rash with well-demarcated erythematous scaly patches and plaques over the trunk and axilla one week after beginning propranolol therapy.