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Recognizing Guttate Psoriasis and Initiating Appropriate Treatment

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Abstract

Guttate psoriasis is a less common form of psoriasis. It manifests with numerous small, teardrop shaped, scaly plaques on the trunk and extremities. The etiology includes both environmental and genetic factors. It commonly arises 3-4 weeks following a beta hemolytic streptococcal infection. In some cases, it may be misdiagnosed as an allergy to the antibiotics being used to treat the streptococcal infection. The treatment of guttate psoriasis can vary by severity, but the mainstay treatment includes photo therapy and topical steroids. This case report presents the etiology, clinical findings and current treatment options of guttate psoriasis. It also discusses importance of differentiating guttate psoriasis from an antibiotic allergy. The confusion between the two can often delay and make treatment more difficult.

Introduction

Psoriasis is a common skin condition affecting 2% of the general population. It presents as erythematous plaques due to the infiltration of inflammatory cells and increased proliferation of keritinocytes.[1] It has multiple variants such as plaque, guttate, inverse, erythrodermic, and pustular. Plaque psoriasis is the most common and most chronic. Plaque psoriasis presents with red, scaly, circular- oval plaques located over the extensor surfaces and scalp. It is a chronic relapsing inflammatory condition with a strong familial predisposition.[2] Guttate psoriasis is a less common form of psoriasis, with a prevalence of less than 30% of patients with psoriasis. It presents with numerous, small teardrop shaped plaques on the trunk and extremities. Guttate psoriasis often follows a beta hemolytic streptococcal upper respiratory tract infection. The plaques usually develop 3-4 weeks after the infection.[3]

Post-streptococcal rashes can be seen and can be difficult to determine the etiology. Common rashes seen after beta-hemolytic streptococcal infections include scarlet fever, drug eruption, and guttate psoriasis.[4] It is important to differentiate between the rashes because treatment options as well as outcome vary widely. Symptoms, clinical findings and timing are important characteristics to consider when choosing from a differential diagnosis.

Case Report

A 53-year-old Caucasian female with no history of psoriasis presented with sore throat, fever and chills for 24 hours. On physical exam, she was febrile (101F) and in no acute distress. She had oropharyngeal erythema, exudate, and swollen tonsils. She had normal rate and rhythm, normal S1 and S2, without murmurs. No swollen lymph nodes were appreciated. A rapid strep antibody test was preformed and was positive for beta hemolytic streptococcus. She was started on amoxicillin 875MG oral twice daily for 7 days.

One week later, she was still complaining of sore throat and tender lymph nodes on the left side of her neck. Per the patient, her symptoms only improved to 90%. On physical exam, she was afebrile (98F), had oropharyngeal erythema, but no exudate and no swollen tonsils. She had anterior chain tenderness and enlarged anterior chain lymphadenopathy on the left side. Since her condition improved by 90% on amoxicillin, we extended the course of antibiotics for 5 more days.

Five days after finishing the antibiotics, she developed a rash on her legs that spread to her torso within one day. At this point, she phoned her primary care physician and explained her symptoms. It was non-puritic, erythematous and raised. At this point, “amoxicillin allergy” was added to the patient’s medical record and she was instructed to come in if her symptoms worsened.

The rash persisted for another 3.5 weeks on the trunk and legs, and she returned to her primary care physician. Some of the areas began to change and look scaly, “like they were drying up” but some had new erythematous areas, which were mildly puritic. On physical exam, she had multiple papulosquamous, 5mm lesions over torso and proximal extremities, sparing the hands and feet (Figure 1, 2). A diagnosis of Guttate Psoriasis was given and CBC with diff, CCP and RPR were ordered along with a dermatology referral. The CBC with diff was within normal limits and RPR was negative. CCP was within normal limits, except for a globulin level of 3.6gm/dl and a total protein of 8.4gm/dl. The dermatologist confirmed the diagnosis of guttate psoriasis.

The patient was treated with triamcinolone acetonide Cream 0.1% application twice daily for 3 weeks. She was also instructed to get as much natural sunlight as possible without burning. Treament with PUVA (psoralen + UVA treatment)

was considered, however, at a three-month follow-up with her primary care doctor, her psoriasis was resolving, and at one-year follow-up, it was completely resolved.

Discussion

Our patient presented with a rash 2.5 weeks after she was diagnosed with strep throat. Initially, the etiology of her rash was unclear. Due to the frequency of drug-induced allergic reactions and failure to visualize the rash, she was misdiagnosed with an allergy. Amoxicillin is a penicillin derivate and one of the most common causes of drug-induced allergic reaction. Antihistamines and systemic corticosteroids are recommended for the treatment of drug-induced allergic reactions; however, many times withdrawal of the drug will suffice.[5]

A drug eruption caused by amoxicillin commonly presents 8-10 days after initiating treatment. It manifests as a combination of macules and papules and usually become confluent affecting large areas of the body.[5] When the rash was visualized 3.5 weeks later, it displayed the classic clinical findings of guttate psoriasis. The rash presented approximately 2.5 weeks after the diagnosis of strep throat and had the characteristic distribution to the trunk and extremities and the classic tear drop appearance.

The etiology of guttate psoriasis includes both environmental and genetic components. The genetic component has been located in the MHC region of chromosome 6p21.3 where the psoriasis susceptibility locus (PSORS)1 resides.[6] This area is thought to house the psoriasis gene. HLA-Cw0602, an allele of the HLA-C gene has been found to have the strongest association with psoriasis phenotype. However, it is important to note that not all HLA-Cw0602 positive individuals develop psoriasis and more than 30% of psoriasis patients are HLA-Cw0602 negative.[6]

T cells are thought to play a critical role in the pathogenesis of guttate psoriasis. Proposed mechanisms for the relationship of post-streptococcal guttate psoriasis have been postulated. Examples include cross reactivity between streptococcal M proteins presented to immune cells in the tonsils and structurally similar type I keratins in the epidermis trigger a T cell mediated autoimmune reaction. In addition, recent studies have demonstrated that streptococcal M proteins and streptococcal pyrogenic exotoxins act as superantigens, which cause a marked expansion of both CD4+, and CD8+ T cells.[1] Evidence of the T cell involvement is seen in the effective treatment with immunosuppressive drugs which inhibit the T cell activation and cytokine secretion such as anti-CD3, corticosteroids, and cyclosporine A.[1]

Guttate psoriasis may spontaneously remit within several weeks to months, it may intermittently reoccur, or may persist and progress to chronic plaque

Figure 1. *Lesions covering patient's upper back*

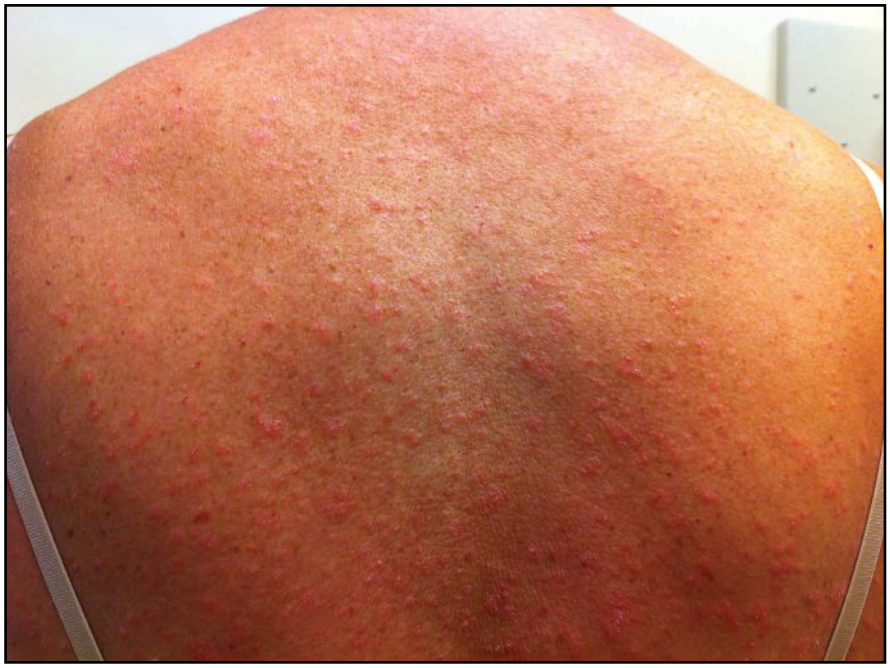
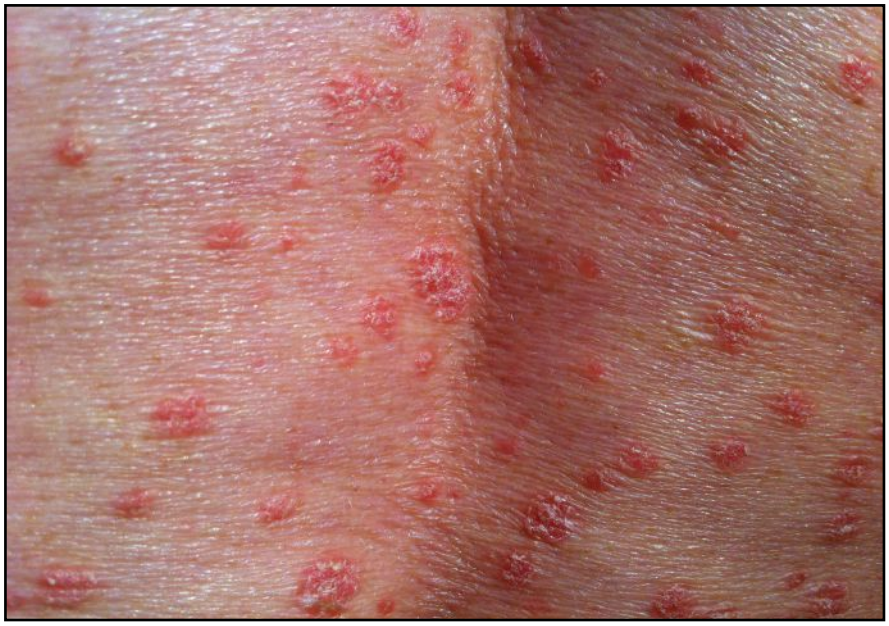


Figure 2. *Closer view of lesions on patient's upper back*



psoriasis. Due to the potential for guttate psoriasis to remit, foregoing treatment is an option.[7] First line therapy includes ultraviolet phototherapy. Options include narrow band UVB, broadband UVB and psoralen plus UVA (PUVA). Patients without access to phototherapy can see improvement in their guttate psoriasis with cautious outdoor sun exposure.[7]

Topical corticosteroids and vitamin D analogues can also be used as monotherapy or as an adjuvant to phototherapy. Systemic immunosuppressive therapies utilized for plaque psoriasis may be used for guttate psoriasis if first line therapy fails.[7] Tonsillectomy has been documented effective in some cases of recurrent post-streptococcal associated guttate psoriasis; however, data is insufficient to recommend routine tonsillectomy. Systemic antibiotic therapy should be used to treat active streptococcal infection; however, little evidence shows efficacy in using systemic antimicrobials for the skin involvement.[7]

It is important not to treat guttate psoriasis with systemic corticosteroids due to the rebound flare phenomenon. They may temporarily show signs of improvement; however, severe rebound attacks occur when the dose is removed, reduced, or even maintained.[8] The rebounds tend to be worse than the initial eruption, and can be life threatening. The rebound attack is often unresponsive to any form of treatment.[8]

Conclusion

Guttate psoriasis is important for primary care physicians to recognize and treat appropriately. Due to its common presentation after a streptococcal infection and probable completion of antibiotic therapy, it is important not to confuse guttate psoriasis with an antibiotic allergy due to the treatment of the allergy worsening the psoriasis.

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