"Guttate Psoriasis: An Overview of Immunopathogenesis and Therapeutic Management"

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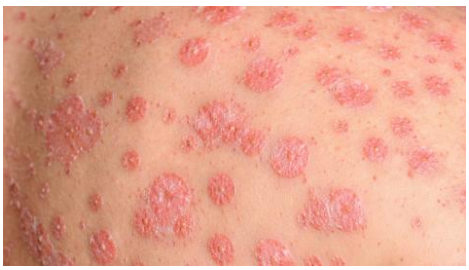
1.ABSTRACT:

Guttate psoriasis is an acute form of psoriasis that typically affects children and young adults. It is characterized by the abrupt emergence of numerous tiny, drop-shaped, erythematous, scaly lesions. It is an immune-mediated inflammatory skin condition that is closely linked to both environmental triggers, especially streptococcal upper respiratory tract infections, and genetic predisposition. Immune dysregulation involving T cells and pro-inflammatory cytokines, particularly the IL-23/Th17 pathway, is the disease's main cause, resulting in keratinocyte hyperproliferation and chronic inflammation. Clinically, guttate psoriasis manifests as pruritus and large, teardrop-shaped lesions; in certain cases, it may develop into persistent plaque psoriasis. The majority of the diagnosis is made clinically, with laboratory and histopathology results used as needed. In severe cases, management consists of systemic medications, phototherapy, topical medicines, and biologics. Results are improved by early diagnosis and tailored treatment.

KEY WORDS: Biologics, Koebner phenomenon, methotrexate, pruritus, tear-drop-shaped papules/plaques and topical corticosteroids.

2.INTRODUCTION:

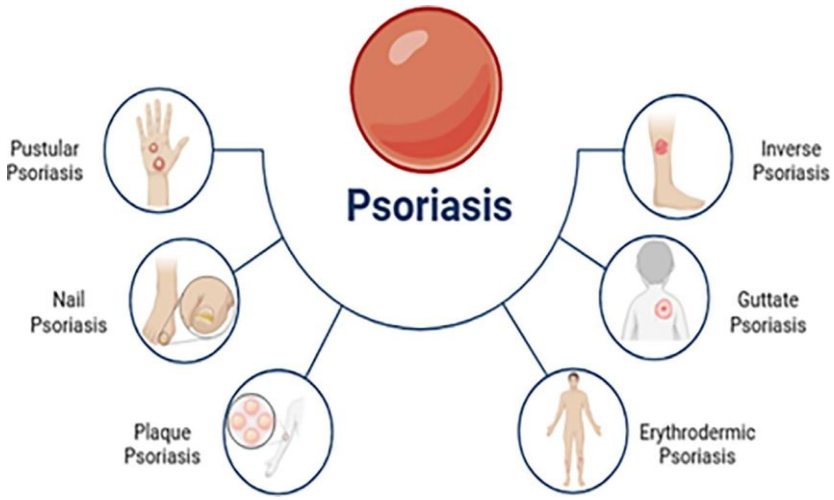
A chronic, immune-mediated inflammatory skin condition that affects roughly 2-3% of people worldwide, psoriasis has a strong hereditary component. Psoriasis is classified by the World Health Organization as a chronic, non-communicable, painful, deforming, and incapacitating disease due to its significant physical, emotional, social, and financial cost.



Psoriasis is becoming more widely acknowledged as a systemic disorder, despite the characteristic erythematous, scaly plaques. Comorbid conditions such obesity, insulin resistance, dyslipidemia, cardiovascular disease, psoriatic arthritis, hepatic involvement, and psychological disorders are common in patients. Excessive keratinocyte proliferation, vascular hyperplasia, and inflammatory cell infiltration in the skin are caused by immunological dysregulation, specifically improper T lymphocyte activation. A better knowledge of these pathways has made it possible to develop targeted biologic medicines that enhance patient quality of life and disease control.

Several types of psoriasis have been identified:

1. Plaque Psoriasis (characterized by dry scaly patches).
2. Pustular Psoriasis (contains pus like fluid infiltrated with white blood cells).
3. Erythrodermic Psoriasis (exfoliation of fine scaly skin with pain and itching).
4. Guttate psoriasis (characterized by drop like dots).
5. Inverse Psoriasis (affects the flexure surfaces and smooth inflamed lesions).
6. Others including scalp psoriasis and nail psoriasis.



3. LITERATURE REVIEW:

3.1 M Garritsen, D E Kraag et al reported that guttate psoriasis (GP) can be triggered not only by streptococcal pharyngitis but also by perianal streptococcal infection. They described a 19-month-old child who developed GP following perianal streptococcal dermatitis without throat infection. Treatment with oral antibiotics and topical corticosteroids led to marked improvement. Review of nine previous case reports showed similar outcomes. The authors emphasized the importance of examining the perianal region for streptococcal infection in children with GP.

3.2 Rahul Mahajan et al are studied about Psoriasis is a chronic inflammatory papulosquamous disease characterized by recurrent relapses and remissions. Although it was once thought to be primarily a disorder of keratinization, current evidence shows that psoriasis is mainly driven by immune dysregulation, especially involving Th1 and Th17 cells. Recent studies have also identified epidermal barrier dysfunction in psoriasis, similar to that seen in atopic dermatitis, contributing to disease development.

3.3 Adriana Rendon et al are studied about the Recent advances in psoriasis research have deepened knowledge of skin biology and immune responses. A better understanding of the IL-23/Th17–driven inflammatory pathway has transformed the management of psoriasis, resulting in targeted and highly effective treatments. These findings have also improved understanding of the mechanisms involved in chronic inflammatory diseases.

3.4 Barry Ladizinski et al Reported about the Inflammatory skin conditions like psoriasis are linked to a number of comorbidities. The Koebner phenomenon is significant to the wound care physician because psoriasis can result from any superficial damage. Particularly vulnerable to flare-ups of this syndrome are joints and wounds. The epidemiology and management of psoriasis are highlighted in this review.

3.5 Nanette B. Silverberg et al studied about the Paediatric psoriasis constitutes about one-third of all psoriasis cases and is increasingly recognized as a systemic inflammatory condition rather than a disease limited to the skin. Recent studies show its association with metabolic abnormalities such as obesity, elevated blood pressure, abnormal lipid levels, and insulin resistance.

This understanding emphasizes the need for early lifestyle modification, weight control, and appropriate therapeutic interventions in affected children.

3.6 Ted Zhou et al analysed the Guttate psoriasis (GP) is a distinct form of psoriasis characterized by multiple drop-like papules and plaques and accounts for up to one-quarter of psoriasis cases, with a significant proportion progressing to chronic plaque psoriasis. This systematic review analysed 75 studies and found limited high-quality evidence, as most were case reports or retrospective studies, with only a few randomized controlled trials. Among available treatments, topical corticosteroids, calcipotriol, and especially narrowband UVB phototherapy showed the strongest efficacy, while antibiotics demonstrated limited benefit. Based on current evidence, a stepwise treatment approach is suggested, though further randomized controlled trials are needed to establish standardized guidelines.

4. AIM AND OBJECTIVES:

AIM:

The aim of this study is to enhance understanding of guttate psoriasis by explaining its etiology, pathogenesis, clinical manifestations, diagnostic methods, treatment options, and management strategies in order to improve patient care, treatment outcomes, and quality of life.

OBJECTIVES:

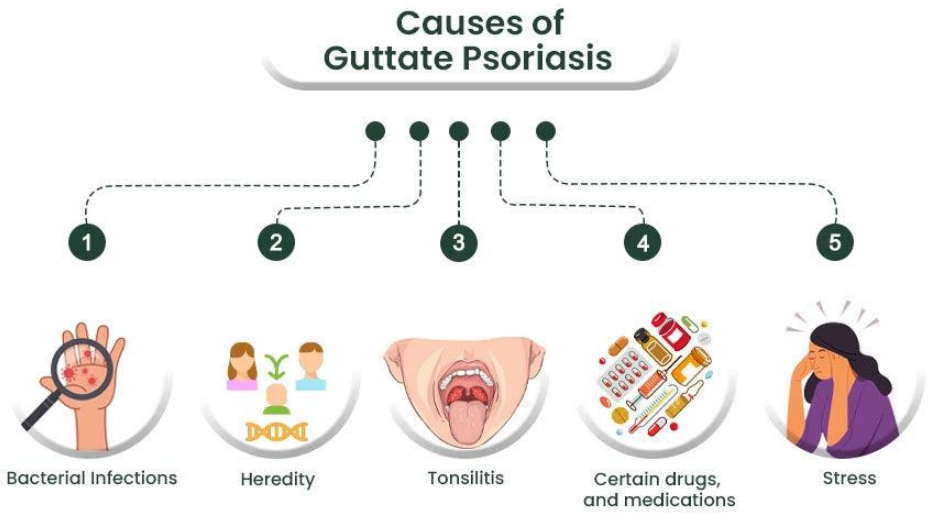
The primary objective of this study is to provide a comprehensive and systematic understanding of guttate psoriasis, a distinct and acute variant of psoriasis that commonly affects children and young adults. This study aims to explain the etiology and epidemiology of guttate psoriasis, with particular emphasis on genetic predisposition and environmental triggers such as streptococcal infections, tonsillitis, stress, cold weather, skin injury, smoking, alcohol consumption, and certain medications that are known to precipitate or exacerbate the condition. It seeks to describe the immunopathogenesis of the disease by outlining the role of immune cells, cytokines, and inflammatory pathways involved in the development of characteristic skin lesions. The study also aims to identify and explain the typical clinical manifestations of guttate psoriasis, including the sudden onset of multiple small, erythematous, drop-like scaly papules distributed mainly over the trunk and extremities. Furthermore, this work intends to discuss the diagnostic approaches used in clinical practice, including clinical evaluation, laboratory investigations, and differential diagnosis to distinguish guttate psoriasis from other papulosquamous disorders. Another important objective is to evaluate the available management strategies, including pharmacological treatments such as topical therapies, antibiotics, phototherapy, and systemic agents, as well as non-pharmacological measures like lifestyle modification and patient counselling. In addition, the study emphasizes the role of healthcare professionals particularly pharmacists, nurses, and physicians in optimizing treatment outcomes through appropriate drug selection, monitoring for adverse effects, improving patient adherence, and providing education on disease prevention and recurrence. Overall, this study aims to enhance clinical knowledge, promote early recognition, and support effective management of guttate psoriasis, thereby improving patient quality of life and long-term disease outcomes.

5. ETIOLOGY:

Guttate psoriasis lesions are broadly distributed, especially on the trunk and proximal extremities. The face may also have lesions. Patients with or without a history of plaque psoriasis may develop guttate psoriasis.

It is linked to group A beta-haemolytic streptococcal infections, which frequently appear two to three weeks before skin symptoms. This illness is frequently self-limiting and can either go away on its own or with therapy.

Chronic guttate psoriasis or chronic plaque psoriasis can occur in some guttate psoriasis patients. Guttate psoriasis could be an acute flare-up of chronic plaque-type psoriasis that already existed, or it could be the first manifestation of psoriasis. Patients often have a history of tonsillitis, laryngitis, or upper respiratory tract infections.



6.PATHOGENESIS:

Commonly caused by streptococcal upper respiratory tract infections, guttate psoriasis is an acute, immune-mediated form of psoriasis that primarily affects children and young people. Infectious agents, immunological dysregulation, genetic predisposition, environmental factors, and epigenetic pathways all interact intricately in this multifactorial etiology.

The PSORS1 locus on chromosome 6p21 and the HLA-Cw6 allele have been found to be strongly associated with genetic vulnerability. Variants in the genes that control the IL-23/Th17 pathway, such as IL-23R, STAT3, and CARD14, further put people at risk for aberrant keratinocyte proliferation and heightened immunological responses.

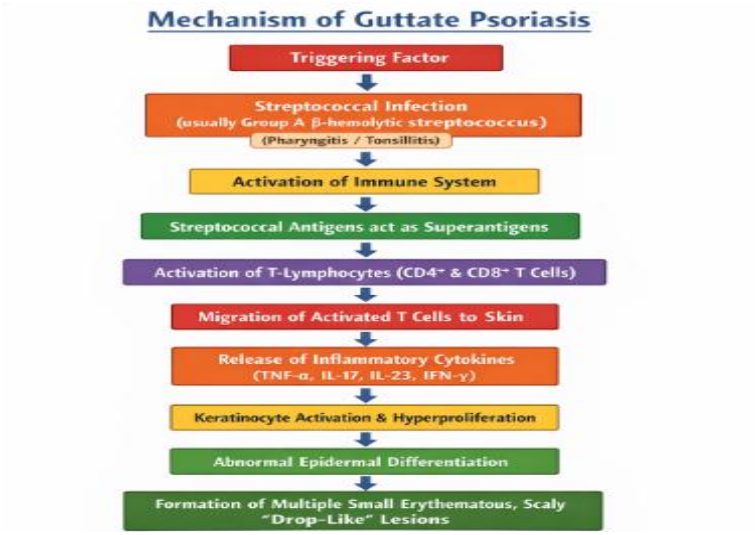
Via molecular mimicry, infectious triggers—specifically, Group A β-hemolytic streptococcicause illness by activating T cells that are cross-reactive because their antigens are similar to keratin proteins. When activated CD4+ and CD8+ T cells reach the skin, they release pro-inflammatory cytokines such IL-17, IL-22, TNF-α, and IFN-γ.

These cytokines promote inflammation, keratinocyte proliferation, and epidermal hyperplasia. In order to maintain Th1 and Th17 responses, dendritic cells are essential because they present antigens and generate cytokines. By secreting antimicrobial peptides and inflammatory mediators, keratinocytes actively contribute to the self-amplifying inflammatory cycle. Environmental elements that can worsen disease activity include stress, trauma, medicines, and weather.

Furthermore, epigenetic modifications, such as dysregulated microRNAs and DNA methylation, affect the expression of immunological and epidermal genes, which in turn affects the severity and course of disease.

7. PATHOPHYSIOLOGY:

There is strong evidence that psoriasis development is mostly influenced by genetic factors. 9.8% of children in the research conducted in northern India had a family history of psoriasis, the percentage reached 28%. The child has a 16% chance of getting psoriasis if only one parent has the condition. If both parents have psoriasis, the likelihood rises to 50%. According to twin pair study, there is 72% concordance between monozygotic twins and 22% concordance between dizygotic twins. Men are more likely than women to pass down psoriasis to their children because of genomic imprinting. Numerous HLA haplotypes have been linked to psoriasis (human leukocyte antigen).



8. EPIDEMIOLOGY:

The prevalence of psoriasis, a chronic inflammatory skin condition that affects people of all ages, is between 0.5 and 2% worldwide in the pediatric population. Due to increased clinical awareness and diagnostic precision, pediatric psoriasis which was previously thought to be uncommon in children is now more widely acknowledged. In both children and adults, plaque psoriasis (psoriasis vulgaris) is the most prevalent of the several clinical variations that have been found. The majority of cases in children are characterized by clearly defined erythematous plaques covered in silvery scales. In children and teenagers, guttate psoriasis is the second most prevalent type. It usually manifests as an abrupt eruption of several tiny, drop-like erythematous papules, frequently after streptococcal infections.

Although guttate psoriasis primarily affects children, teenagers, and young adults, it can develop at any age, suggesting that the diagnosis is not excluded by age alone. While inverse psoriasis, pustular psoriasis, and erythrodermic psoriasis are rare in children, their severity makes them clinically relevant. While pustular psoriasis manifests as sterile pustules, erythrodermic psoriasis is characterized by extensive erythema and scaling and can be fatal. In youngsters, inverse psoriasis may go undiagnosed because it affects intertriginous tissues.

Males and females in the pediatric age group have comparable prevalence, according to epidemiological studies. A significant part is played by genetic predisposition; many impacted children report a good family history. Disease development and progression are influenced by the interaction of genetic vulnerability and environmental factors, including infections, stress, trauma, and climate.

9.SIGNS AND SYMPTOMS:

The body, back, limbs, and occasionally the neck, head, and scalp are all covered with a rash of tiny patches (up to 1 cm in diameter) caused by guttate psoriasis. On fair skin types, these patches are typically bright pink or red, but those with darker skin tones may note that the patches are dark instead of red. Additionally, guttate areas could have some fine scaling. While some people find guttate psoriasis to be extremely itchy or painful.

- **Patches:** Darker skin tones may see purple or brown patches, while lighter skin tones will see round or teardrop-shaped pink or red patches. Typically, the patches range in width from 2-10 mm. Dry skin scales that separate from the red areas may be seen. The arms, legs, and chest are where the patches appear.
- **Itching**: The patches of skin will itch and irritated.
- **Teardrop-Shaped Spots:** Oval, round, or small red or pink pimples that frequently come out of nowhere.
- **Location:** Usually affects the arms, legs, and trunk, but it can also affect the face, scalp, and ears.
- **Scales:** Although they are occasionally absent, the spots typically contain a thin, silvery-white scale that can peel off.

Burning and Itching: Affected areas frequently experience burning or itching.

- **Dryness:** The skin may become flaky and dry.

- **Infection Triggered by:** Usually occurs after an upper respiratory infection or strep throat. Changes to the nails may include discolouration or pitting.
- **Temporary Nature:** Usually goes away in a few weeks or months, but it can develop into persistent plaque psoriasis.
- **Nail Changes:** Some persons have thickness, discolouration, or pitting of their nails.
- **Fatigue:** Prolonged exhaustion brought on by the body's inflammatory reaction.
- **Progression:** Chronic plaque psoriasis develops in about one-third of guttate psoriasis sufferers.

10. RISK FACTORS:

10.1 Genetic factor:

Inflammatory skin disease mediated by T cells, psoriasis is typified by the infiltration of CD8+ T cells in the epidermis and CD4+ and CD8+ T cells in the dermis. In contrast to Th1 and Th17 cytokines, which include IFN-γ, TNF-α, IL-1, IL-6, IL-17, and IL-22, Th2 cytokines, such IL-10, are downregulated. Keratinocyte damage brought on by environmental stimuli activates dendritic cells and releases IL-12 and IL-23. In response to these cytokines, Th1 and Th17 cells proliferate, angiogenesis occurs, neutrophils are drawn in, and distinctive erythematous, scaly plaques are formed.

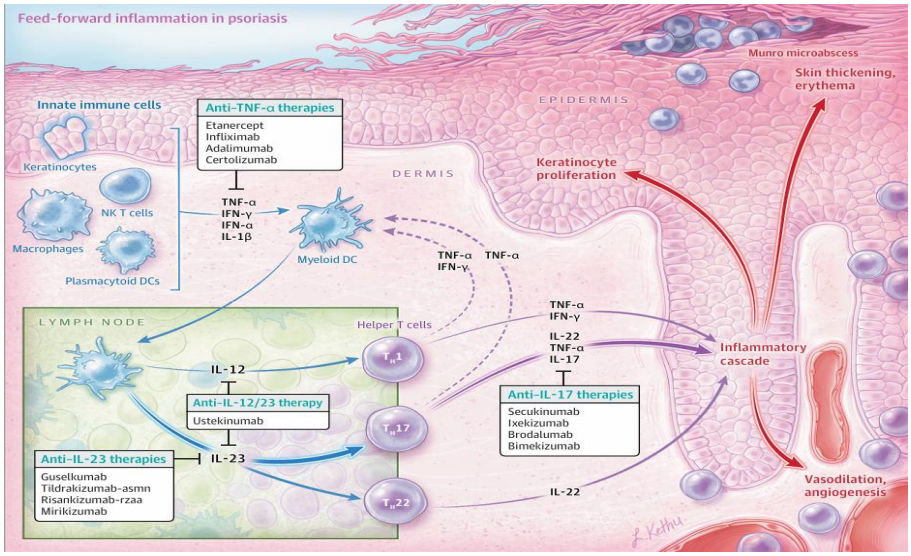
10.2 Based on environmental factor:

The environmental factors and immune responses interact to cause chronic skin inflammation. The process begins with genetic susceptibility combined with environmental triggers such as infections, trauma, stress, or drugs. These factors lead to keratinocyte injury in the epidermis and increased presentation of antigens. Injured keratinocytes release inflammatory signals that alert the immune system and initiate the disease process.

10.3 Based on impaired skin barrier:

In guttate psoriasis, an impaired skin barrier allows triggers (such as streptococcus bacteria) to penetrate, activating the immune system and causing inflammation, rapid skin cell turnover (hyperproliferation), and the characteristic small, red, scaly spots, creating a vicious cycle in which barrier damage fuels inflammation and inflammation further damages the barrier, with lipid changes (ceramides) and genetic factors playing important roles.

- Disruption of Physical Barriers:
- Keratinocyte Differentiation and Proliferation Disruption:
- Immune Barrier Dysregulation:



11. COMPLICATION:

Psychological distress and the possibility of developing chronic plaque psoriasis, which can result in major problems like psoriatic arthritis, nail issues, and elevated risks for metabolic conditions (obesity, diabetes, heart disease), eye inflammation, and other autoimmune disorders, are common complications of guttate psoriasis. Guttate psoriasis is a warning indication for the development of these long-term, systemic health issues, even though it is typically transient. Psychosocial effects and impaired quality of life. Complications of guttate psoriasis for ex: metabolic syndrome, non-alcoholic fatty liver disease [Liver problems and psoriasis and Dyspigmentation in skin of colour].

11.1 Acute complications:

- **Secondary Skin Infections**: Scratching due to severe itching (pruritus) can breach the skin's barrier and allow bacteria to enter, resulting in infections.
- **Psychological factors**: The abrupt, extensive rash can significantly lower quality of life and induce anxiety and stress.
- **Koebner Phenomenon**: New guttate lesions may develop at the areas of skin trauma (cuts, sunburns, etc.).
- **Transition to Chronic Psoriasis:** About 25-30% of individuals with guttate psoriasis may develop chronic plaque psoriasis.

11.2 Chronic or Systemic complication:

Although guttate psoriasis is essentially a skin disorder that is frequently brought on by infection, it can be a sign of more serious systemic consequences if it develops into a chronic form of psoriasis or if there is underlying systemic inflammation that affects several organ systems. Up to 30% of individuals with psoriasis (including the guttate variant) have psoriatic arthritis (PsA), a serious consequence that causes joint discomfort, stiffness, and swelling. If treatment is delayed and ineffective, it may become incapacitating.

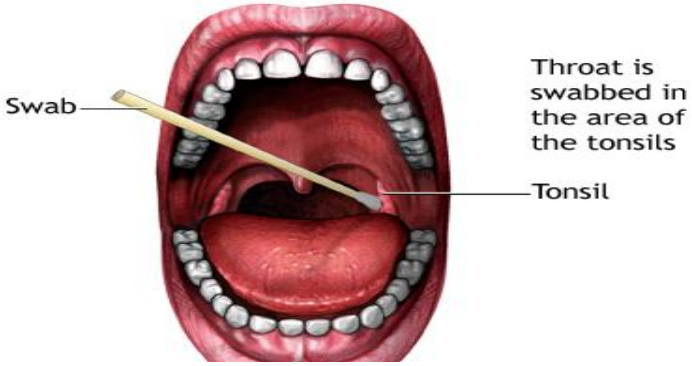
12. DIAGNOSIS:

Widespread, numerous, tiny, dispersed, scaly, erythematous, oval papules, and plaques on the trunk and limbs are characteristic physical characteristics that are the primary basis for the clinical diagnosis, particularly if there has been a prior history of streptococcal infection. Dermo copy, which usually displays a dull-red or bright-red backdrop with diffuse white scales

and dotted vessels dispersed in a pattern, can help with the diagnosis. 3 Patients who do not exhibit symptoms or indicators of a streptococcal infection typically do not require laboratory testing. Measurement of serum antistreptococcal antibody and culture from a suitable site (such as the throat and perianal area) can be beneficial in patients exhibiting signs of streptococcal infection. Antibody titres typically peak between three and six weeks later and stay high for several months. Although typically not required, a skin biopsy could be taken into consideration if the diagnosis is uncertain.

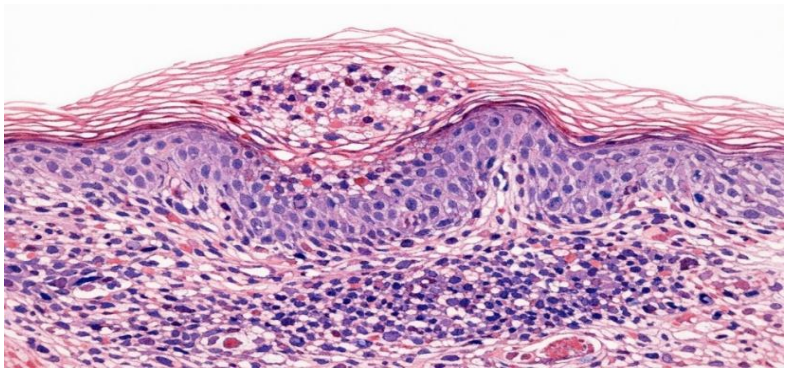
12.1 Throat swab culture:

Because guttate psoriasis is frequently brought on by a prior Streptococcus (strep) throat infection, a throat swab culture, which particularly looks for bacteria like Group A Streptococcus is a typical diagnostic for the illness. Although there are other factors, such as viruses, a positive swab establishes the infection, directing treatment and possibly ending the psoriasis flare. Another way to identify a recent strep infection is by blood testing (titres).



12.2 Skin biopsy:

In order to identify skin disorders including cancer, infections, rashes, or inflammation, a dermatologist may often perform a skin biopsy, which involves removing a little sample of skin tissue for microscopic analysis. Shave (superficial scraping), punch (circular core), and excisional (removing the entire lesion) are frequent types performed under local anaesthesia. Results are usually available in a matter of days to weeks after a pathologist analyses the sample, assisting medical professionals in identifying the precise source of skin problems and directing appropriate treatment. All corresponding patients' electronic medical records were examined by a dermatology resident physician who was blind to the histology results in order to identify evidence that either confirmed or denied a psoriasis diagnosis



13. MANAGEMENT:

Except for cosmetic reasons or the management of itching, active therapy might not be required due to the potential for spontaneous remission within a few months. However, other writers advocate for active treatment of guttate psoriasis since it can develop into chronic plaque psoriasis in 40–50% of patients. In this context, factors such as patient age, disease severity, quality of life effect, comorbidities, response to prior treatment, and patient preferences should be taken into account.

Although there isn't a cure for guttate psoriasis at currently, there are a number of treatment options that can help with the

condition's symptoms, signs, and skin lesions. Despite this, numerous therapy approaches have been thoroughly researched. A ten-year-old kid with streptococcal pharyngitis had an enlarged left tonsil with tonsillar exudate. Guttate psoriasis appeared two weeks after this A 4-year-old boy who has scalp dermatitis caused by streptococci. After three weeks, the youngster developed guttate psoriasis. These therapeutic approaches have not been formally tested for the treatment of guttate psoriasis, unlike the therapy of plaque psoriasis.

13.1 Treatment options for guttate psoriasis:

1. **Topical therapies**
   - Corticosteroids
   - Vitamin D analogues
   - Calcineurin inhibitors
   - Anthralin

2. Phototherapy

3. Systemic therapies
   - Methotrexate
   - Cyclosporine
   - Retinoids
   - Fumaric acid esters
   - Biologics

4.Antistreptococcal interventions
   - Systemic antibiotic therapy and Tonsillectomy.

14. CONCLUSION:

A unique and clinically significant form of psoriasis, guttate psoriasis emphasizes the intricate interactions among genetic predisposition, immunological dysregulation, environmental variables, and infectious triggers, especially streptococcal infections. Guttate psoriasis is not a merely cutaneous or benign disorder, despite the fact that it frequently manifests as an acute, self-limiting eruption of tiny, drop-like erythematous lesions. Rather, it is a systemic inflammatory disease driven by the immune system that may develop into chronic plaque psoriasis and result in long-term multisystem problems. Excessive cytokine release, keratinocyte hyperproliferation, angiogenesis, and chronic inflammation are all consequences of aberrant T-cell-mediated immunological responses, particularly stimulation of the IL-23/Th17 pathway. Disease onset and severity are further influenced by environmental triggers such infections, stress, and drugs, as well as genetic vulnerability, particularly HLA connections. The abrupt onset, widespread lesions, pruritus, cosmetic problems, and psychological burden of the disorder seriously impair quality of life, particularly in children and young people. Disease severity, patient age, comorbidities, and quality of life should all be taken into account when managing a patient. Preventing the course of the disease and its systemic involvement requires early detection and adequate treatment. Biologic therapy developments that target certain cytokines have greatly enhanced results, highlighting how crucial an understanding of immunopathogenesis is for directing efficient management approaches.

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REFERENCES:

1. Alexander KC Leung, Barankin B, Lam JM, Kin Fon Leong. Childhood guttate psoriasis: an updated review. Drugs in context. 2023 Oct 23; 12:1–20.

2. Handa S, Mahajan R. Pathophysiology of psoriasis. Indian Journal of Dermatology, Venereology, and Leprology. 2013;79(7):1.

3. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis. JAMA. 2020 May 19;323(19):1945–60.

4. Psoriasis: Pathophysiology and diagnosis. Clinical Pharmacist. 2013.

5. Alhammad IM, Aseri AM, Alqahtani SAM, Alshaebi MF, Alqahtani SA, AL Zahrani RA, et al. A review on updates in management and Treatment of Psoriasis. Archives Of Pharmacy Practice [Internet]. 2021;12(1):74–8.

6. Dhabale A, Nagpure S. Types of Psoriasis and Their Effects on the Immune System. Cureus [Internet]. 2022 Sep 1;14(9).

7. Garritsen FM, Kraag DE, de Graaf M. Guttate psoriasis triggered by perianal streptococcal infection. Clin Exp Dermatol. 2017;42(5):536–538. https://doi.org/10.1111/ced.13129

8. Duffin KC, Hwang ST, Krueger JG. Advances and Controversies in Our Understanding of Guttate and Plaque Psoriasis. The Journal of Rheumatology [Internet]. 2023 Nov 1 [cited 2024 Mar 26];50(Suppl 2):4–7. Available from: https://www.jrheum.org/content/50/Suppl_2/4.

9. Koca TT. A short summary of clinical types of psoriasis. Northern Clinics of Istanbul. 2016;3(1).

10. É. Sbidian, Chaimani A, Guelimi R, I. García-Doval, Hua C, Hughes C, et al. Systemic pharmacological treatments for chronic plaque psoriasis: a network meta-analysis. The Cochrane library. 2023 Jul 12;2023(7).

11. Kimmel GW, Lebwohl M. Psoriasis: Overview and Diagnosis. Updates in Clinical Dermatology [Internet]. 2018;1–16. Available from: https://link.springer.com/chapter/10.1007%2F978-3-319-90107-7_1

12.Singh S, Bhansali A. Randomized placebo control study of metformin in psoriasis patients with metabolic syndrome (systemic treatment cohort). Indian Journal of Endocrinology and Metabolism. 2017;21(4):581.

13. Ju HJ, Park HJ, Choi IH, Lee KH, Kwon MY, Park CJ. Comparison of Th1 and Th17 inflammatory cytokine profiles between chronic plaque and acute guttate psoriasis. Ann Dermatol. 2022;34(3):200–205. https://doi.org/10.5021/ad.2022.34.3.200.

14. Yan K, Han L, Deng H, et al. The distinct role and regulatory mechanism of IL-17 and IFN-γ in the initiation and development of plaque vs guttate psoriasis. J Dermatol Sci. 2018;92(1):106–113. https://doi.org/10.1016/j.jdermsci.2018.07.001.

15. Krueger G, Ellis CN. Psoriasis – recent advances in understanding its pathogenesis and treatment. J Am 16. Luba KM, Stulberg DL. Chronic plaque psoriasis. Am Fam Physician

16.Accad Dermatol. 2005;53(1 Suppl. 1): S94–S100. https://doi.org/10.1016/j.jaad.2005.04.035.

17. Fan X, Yang S, Sun LD, et al. Comparison of clinical features of HLA-Cw*0602-positive and -negative psoriasis patients in a Han Chinese population. Acta Derm Venereol. 2007;87(4):335–340. https://doi.org/10.2340/00015555-0253

18. Chen L, Tsai TF. HLA-Cw6 and psoriasis. Br J Dermatol. 2018;178(4):854–862. https://doi.org/10.1111/bjd.16083

19. Gudjonsson JE, Karason A, Runarsdottir EH, et al. Distinct clinical differences between HLA-Cw*0602 positive and negative psoriasis patients--an analysis of 1019 HLA-C- and HLA-B-typed patients. J Invest Dermatol. 2006;126(4):740–745. https://doi.org/10.1038/sj.jid.5700118

20. Mallon E, Bunce M, Savoie H, et al. HLA-C and guttate psoriasis. Br J Dermatol. 2000;143(6):1177–1182. https://doi.org/10.1046/j.1365-2133.2000.0388 Mallon E, Bunce M, Savoie H, et al. HLA-C and guttate psoriasis. Br J Dermatol. 2000;143(6):1177–1182. https://doi.org/10.1046/j.1365-2133.2000.03885.x5.x.

21. Telfer NR. The Role of Streptococcal Infection in the Initiation of Guttate Psoriasis. Archives of Dermatology. 1992 Jan 1;128(1):39.