**Case Report**

**Guttate Psoriasis Can Present With Psoriatic Arthritis after Urinary Tract Infection: Case Report**

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**ABSTRACT**

**Introduction:** Psoriasis and psoriatic arthritis (PsA) are serious, poorly understood, diseases. As many as 10-30% of psoriasis patients develop an inflammatory arthritis termed psoriatic arthritis which is progressive and leads to destruction of the joints if it is not treated assertively. Several triggering factors may elicit or aggravate the expression of psoriasis, of which focal infections is a well-established triggering factor, in particular infections of the upper respiratory tract. Guttate (eruptive) psoriasis, known to have a better prognosis than other types of psoriasis with rapid involution and longer remission, but its clinical course has barely been studied. We are presenting a case of guttate psoriasis with severe onset and association with nail psoriasis, scalp psoriasis and psoriatic arthritis, all which started unexpectedly after urinary tract infection.

**Case Presentation:** We are presenting a young woman of 32-years-old who presented to our service with a typical guttate psoriasis lesion after urinary tract infection. Her guttate psoriasis exhibited unusual severe course with associated onset of nails psoriasis, scalp psoriasis, conjunctivitis and psoriatic arthritis. All the symptoms started few days after urinary infection symptoms.

**Conclusion:** Guttate psoriasis that occurs after urinary tract infection, may exhibit severe onset and rapid progression to psoriatic arthritis.

**KEYWORDS:** Psoriasis; Psoriatic arthritis; Guttate; Urinary tract infection.

**ABBREVIATIONS:** PsA: Psoriatic arthritis; HIV: Human Immunodeficiency Virus; WBC: White Blood Cell; MCV: Mean Cell Volume; RBC: Red Blood Cells; ESR: Erythrocyte Sedimentation Rate; ASO: Anti-Streptolysin O; TPHA: Treponema pallidum hemagglutination.

**INTRODUCTION**

Psoriasis and psoriatic arthritis (PsA) are chronic inflammatory diseases that have a major impact on health.

As many as 10-30% of psoriasis patients develop an inflammatory arthritis termed psoriatic arthritis which is progressive and leads to destruction of the joints if it is not treated assertively.¹ Environmental risk factors including streptococcal pharyngitis, stressful life events, low humidity, drugs, human immunodeficiency virus (HIV) infection, trauma, smoking and obesity have been associated with psoriasis and psoriatic arthritis.²

Of psoriasis, guttate psoriasis is a distinct eruptive dermatosis that classically occurs in children and young adults following upper respiratory tract infection.³ It might present as either the initial manifestation of psoriasis in individuals previously unaffected by psoriasis or as an acute exacerbation in individuals with pre-existing chronic plaque psoriasis. Guttate psoriasis is strongly associated with antecedent or concomitant streptococcal infection and of-
ten occurs 1 to 2 weeks after streptococcal pharyngitis or a viral upper respiratory infection. Typically, they manifests as multiple scaly, well-demarcated, salmon-pink to erythematous, drop like round to oval papules ranging in size from 1 mm to 10 mm in diameter, appearing primarily on the trunk and extremities, sparing the palms and soles. Fine silvery scale is often present on more established lesions.

Although, limited information is available about the long-term prognosis of individuals with first-manifestation of guttate psoriasis. Ko et al reported that patients have two distinguishable clinical courses, a rapid involution course with long-term remission and a chronic course without remission. Others reported that approximately 33% of patients with guttate psoriasis might eventually develop chronic plaque psoriasis. The diagnosis of guttate psoriasis is essentially a clinical diagnosis, and a careful history regarding recent illness or medication use can help to clarify the condition from other differential diagnosis.

Here, we present unusual case of sudden onset of guttate psoriasis with nail psoriasis, scalp psoriasis, conjunctivitis and psoriatic arthritis, without preceding history of upper respiratory tract infection.

CASE HISTORY

Our case is a 32-year-old woman, who presented with typical skin lesions of guttate psoriasis after urinary tract infection. Her guttate psoriasis exhibited unusual scenario with concomitant development of nails psoriasis, scalp psoriasis, and psoriatic arthritis.

The patient presented with 2 weeks chief complains of abrupt onset of eruptive scaly rashes over her body, scaly lesion over the scalp, eyes soreness and a painful swollen right wrist and left knee. Patient reported a difficult and painful mouth opening that started with her right wrist and left knee swelling. Few days earlier to her symptoms development she noticed darkness of her urine with burning micturation. Ten days after the rashes inception she developed painful left knee swelling and sore redness of both eyes. It’s the first attack of its type with no significant previous medical history or similar familial history. There was no preceding upper respiratory tract infection or bowel motion disturbance.

Examination revealed a young woman in severs’ pain and inability to walk because of left knee pain. Temperature was 38 °C, arterial blood pressure 128/84 mmHg, heart rate 89 beats per minute and respiratory rate 22 per minute. Conjunctivitis was evident in both eyes (Figure 1). There was no peripheral lymphadenopathy. The scalp was full of dry scales (Figure 2). First and third right nails were yellow in color with onycholysis (Figures 3A and 3B). There was a scaly eruptive lesion over the trunk and all the four extremities with spare of palms and soles. Rashes were erythematous papules with fine silvery scales that can be seen over some of the lesions. Dermatological consultation confirmed the nature of the skin lesions as a guttate psoriasis (Figure 4). Left knee and right wrist were red and swollen. Both
temporo-mandibular joints were tender and very painful to mild touch. Right hand showed dactylitis of the little finger with sausage shape look (Figure 3B).

Investigations revealed high white blood cell (WBC) count of 14.3×10^3/ul, with neutrophilia of 10.6×10^3/ul, Hb 11.8 g/dl, mean cell volume (MCV) 95.3 fl and platelet 466. There were no particular alterations in the electrolytes, renal or liver functions. Urine showed a pus cells over the full field, 2-4/hpf red blood cells (RBC) and squamous epithelial cells (three plus). A high inflammatory marker with erythrocyte sedimentation rate (ESR) of 130 mm/hr and C-reactive protein of 175.4 mg/l. Negative anti-streptolysin O (ASO) titer, HIV I+II+0+p24 antigen and Mantoux test. Rapid plasma regain antibody and treponema pallidum hemagglutination (TPHA) were non-reactive. Brucella abortus and melitensis antibodies were 1:80. Aerobic and anaerobic blood cultures showed no growth. A chest, hands and knees X-rays were normal.

A diagnosis of psoriasis and psoriatic arthritis had been made based on the characteristic body lesion, scaly scalp, bilateral eye conjunctivitis, nail psoriasis, polyarthritis and dactylitis.

Two triamcinolone injections were given in the right wrist and in the left knee. Antibiotics eye ointment and drops were given for the conjunctivitis. Skin lesion treated with local application of coal tar preparations, topical corticosteroids and Fusidic acid cream. Urinary tract infection was covered with a course of antibiotics. Methotrexate of 15 mg/wk and folic acid 5 mg/wk were started after hepatitis screen came negative for both hepatitis B and hepatitis C.

On the 4th day of treatment the psoriatic lesion became clearer with more silvery scales that can be appreciated over more lesions (Figure 5).

**DISCUSSION**

Psoriasis is a chronic inflammatory disease affecting 1-3% of the world’s population. Joints can be affected in up to 30% of patients.8

As psoriasis has a large spectrum of clinical features and evolution, classification of its clinical features has been a controversial subject among investigators. Thereafter, no complete agreement on the classification of the clinical variants exists.8

It’s reported that psoriasis can be provoked or exacerbated by a variety of different environmental factors, particularly infections and drugs.7 Despite that it has been reported that various microorganisms are associated with the provocation and/or exacerbation of psoriasis, their roles in the disease pathogenesis are unknown.10-12

Knowledge of the factors that may trigger, psoriasis is of primary importance in clinical practice. Extensive evidence supports that the disease can be triggered by a variety of different environmental factors, particularly streptococcus pyogenes, which has been recognized for at least 50 years and implicated in both acute and chronic forms of the disease.9,13,14 The link between psoriasis and infections is probably explained by the “superantigen theory”, that superantigens are the products of bacteria, virus or fungi, which can bypass normal immunological pathway and cause powerful stimulation of the immune system.15 Wang et al13 suggested that cell-wall-deficient bacterial infection may be a virtual triggering factor in psoriasis by regulating T-cell activation. To the best of our knowledge, this is the 1st case report of guttate psoriasis after urinary tract infection. More, it’s the 1st case to have abrupt onset of guttate psoriasis, nail and scalp psoriasis and psoriatic arthritis.

**CONCLUSION**

There are conflicting views in the literature regarding the triggering infection factors and the efficacy of anti-streptococcus antibiotics on psoriasis. Hence, other organism and different kinds of infection factor could be implicated in psoriasis development. Organisms causing urinary tract infection could trigger
psoriasis through the same mechanism as streptococcus bacteria of upper respiratory infection do.

COMPETING INTERESTS

The authors declare that they have no competing interests.

AUTHORS CONTRIBUTIONS

HS wrote the manuscript and compiled the figures. AI edited the manuscript. Both authors analyzed and interpreted the patient data. Both authors read and approved the final manuscript.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

REFERENCES

1. Nograles KE, Brasington RD, Bowcock AM. New insights into the pathogenesis and genetics of psoriatic arthritis. Nat Clin Pract Rheumatol. 2009; 5(2): 83-91. doi: 10.1038/nccprheum0987

2. Chandran V, Raychaudhuri SP. Geoepidemiology and environmental factors of psoriasis and psoriatic arthritis. J Autoimmun. 2010; 34(3): 314-321. doi: 10.1016/j.jaut.2009.12.001

3. Ko HC, Jwa SW, Song M, Kim MB, Kwon KS. Clinical course of guttate psoriasis: Long-term follow-up study. J Dermatol. 2010. 37(10): 894-899. doi: 10.1111/j.1346-8138.2010.00871.x

4. Chalmers RJ, O’Sullivan T, Owen CM, Griffiths CE. A systematic review of treatments for guttate psoriasis. Br J Dermatol. 2001; 145(6): 891-894. doi: 10.1046/j.1365-2133.2001.04505.x

5. Potok O, Prajapati V, Barankin B. Can you identify this condition? Can Fam Physician. 2011; 57(1): 56-57.

6. Fitzpatrick TB, Johnson R, Wolf K, Suurmond D. Color Atlas and Synopsis of Clinical Dermatology: Common and Serious Diseases. 4th ed. New York, NY, USA: McGraw Hill; 2001.

7. Martin BA, Chalmers RJ, Telfer NR. How great is the risk of further psoriasis following a single episode of acute guttate psoriasis? Arch Dermatol. 1996; 132(6): 717-718. doi: 10.1001/archderm.1996.03890300147032

8. Ayala F. Clinical presentation of psoriasis. Reumatismo. 2007; 59(Suppl 1): 40-45. doi: 10.4081/reumatismo.2007.1s.40

9. Fry L, Baker BS. Triggering psoriasis: The role of infections and medications. Clin Dermatol. 2007; 25(6): 606-615.

10. Buslau M, Menzel I, Holzmann H. Fungal flora of human faeces in psoriasis and atopic dermatitis. Mycoses. 1990; 33(2): 90-94. Web site. http://europepmc.org/abstract/med/2191222. Accessed September 16, 2016.

11. Favre M, Orth G, Majewski S, Baloul S, Pura A, Jablonska S. Psoriasis: A possible reservoir for human papillomavirus type 5, the virus associated with skin carcinomas of epidermodysplasia verruciformis. J Invest Dermatol. 1998; 110(4): 311-317. 10.1046/j.1523-1747.1998.00164.x

12. Gudjonsson JE, Thorarinsson AM, Sigurgeirsson B, Kristinsson KG, Valdimarsson H. Streptococcal throat infections and exacerbation of chronic plaque psoriasis: A prospective study. Br J Dermatol. 2003; 149(3): 530-534. doi: 10.1046/j.1365-2133.2003.05552.x

13. Telfer NR, Chalmers RJ, Whale K, Colman G. The role of streptococcal infection in the initiation of guttate psoriasis. Arch Dermatol. 1992; 128(1): 39-42. doi: 10.1001/archderm.1992.01680110049004

14. Raza N, Usman M, Hameed A. Chronic plaque psoriasis: Streptococcus pyogenes throat carriage rate and the therapeutic response to oral antibiotics in comparison with oral methotrexate. J Coll Physicians Surg Pak. 2007; 17(12): 717-720. doi: 12.2007/JCPSP.717720

15. Wang GL, Li XY, Wang MY, et al. Cell-wall-deficient bacteria: A major etiological factor for psoriasis? Chin Med J (Engl). 2009. 122(24): 3011-3016. Web site. http://www.medicinabioeconomic.com.br/biblioteca/pdfs/Doencas/do-1704.pdf. Accessed September 16, 2016.