Case Report

Human papilloma virus infection and psoriasis: Did human papilloma virus infection trigger psoriasis?

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Abstract

Psoriasis is an autoimmune chronic inflammatory skin disease known to be triggered by streptococcal and HIV infections. However, human papilloma virus infection (HPV) as a triggering factor for the development of psoriasis has not been reported yet. We, hereby report a case of plaque type with inverse psoriasis which probably could have been triggered by genital warts (HPV infection) and discuss the possible pathomechanisms for their coexistence and its management.

Key words: Condyloma acuminate, human papilloma virus, inverse psoriasis, plaque psoriasis

INTRODUCTION

Psoriasis is an autoimmune chronic inflammatory skin disease. The etiological factors for psoriasis are a genetic predisposition, trauma, infections, drugs, sunlight, stress, alcohol, smoking, HIV, and AIDS.[1] Psoriasis preceded by streptococcal infection and herpes simplex virus (HSV) infection has been reported in past. Also, the lipophilic yeast *Malassezia* furfur has been implicated in the triggering of scalp lesions in psoriasis.[2] Association between psoriasis and diseases such as vitiligo, bullous pemphigoid, systemic diseases such as lymphoproliferative malignancy and metabolic syndrome have been established; but no such associations have been evaluated, yet between psoriasis and human papilloma virus (HPV) infection. Although, a case of Buschke-Löwenstein-type giant penile condyloma developed in a human immunodeficiency virus-negative, a 25-year-old man, after 4 years of intermittent cyclosporine therapy (5 mg/kg/day) for pustular psoriasis has been reported.[3] But here we report a case of psoriasis which probably could have been triggered by genital warts (HPV infection) and discuss the possible association between the two rare co-existing dermatological conditions.

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CASE REPORT

A 20-year-old unmarried, male patient, farmer by occupation presented with multiple scaly, erythematous plaques of various sizes ranging from 1 cm × 1 cm to 3 cm × 4 cm present over the both upper limbs, lower limbs, back, and scalp [Figure 1]; multiple scaly lesions over both palms and soles since 1½ months. He had erythematous plaques with mild scaling involving both axilla and groins, (inverse psoriasis) which developed before the appearance of similar lesions on the rest of the body. He also had proximal onycholysis involving right ring and middle and ring finger of left hand. Grattage test and Auspitz sign were positive. He also had multiple soft, pink colored, and papilliferous masses of condyloma

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acuminata over the coronal sulcus extending over glans since 2 months [Figure 2]. The lesions were nontender without any surrounding or urethral discharge or any adjacent lymphadenopathy. He gave a history of multiple unprotected sexual contacts with sex workers. History of sexual contact through oral or anal route was absent. No history of any other perianal or genital lesions in the past or any other concurrent infection was found. The patient had no history of smoking, alcoholism, fever, joint pain, burning micturition, or oral aphthae. KOH smear for skin lesion was negative. The skin biopsy report confirmed the diagnosis of psoriasis vulgaris. His investigations for complete blood count, liver function test, renal function test, and blood glucose level were normal. His HIV-I, II test by Coombs kit and VDRL were nonreactive. He was given potent topical steroids (0.05% clobetasol propionate ointment and lotion) and emollients for psoriasis which showed good response [Figure 3]. Podophyllin was applied once weekly and with the two applications there was an excellent response to the treatment with complete clearance [Figure 4]. He was advised to regular follow-up.

DISCUSSION

Psoriasis is an immunologically mediated chronic, inflammatory, and hyperproliferative skin disease with a genetic basis.[4] It affects approximately 2% of general population.[5] It can be developed at any age but it is more common in the age group of 15–30 years. Chronic plaque psoriasis manifesting after streptococcal infection[1] and after HSV infection have been reported in the past.[6] Association of psoriasis with *Helicobacter pylori* infection has also been described.[7] Increased prevalence of HPV has been demonstrated in a hair plucked from patients with psoriasis treated by psoralen with ultraviolet-A.[8] HPV-5 associated with epidermodysplasia verruciformis was also found to be more prevalent in the psoriatic patient.[9] Also, exacerbation of psoriasis has been linked with skin

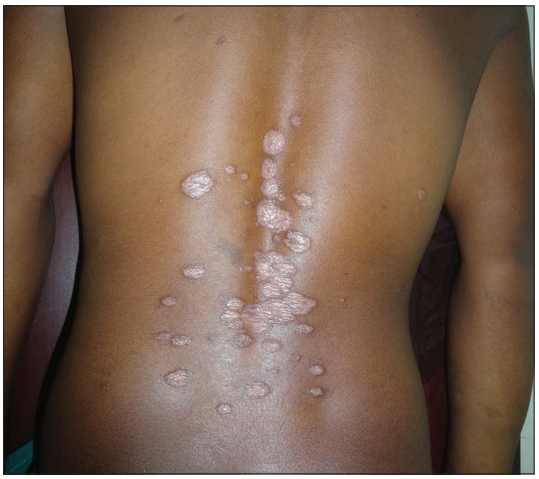

Figure 1: Multiple scaly plaques of the lower back

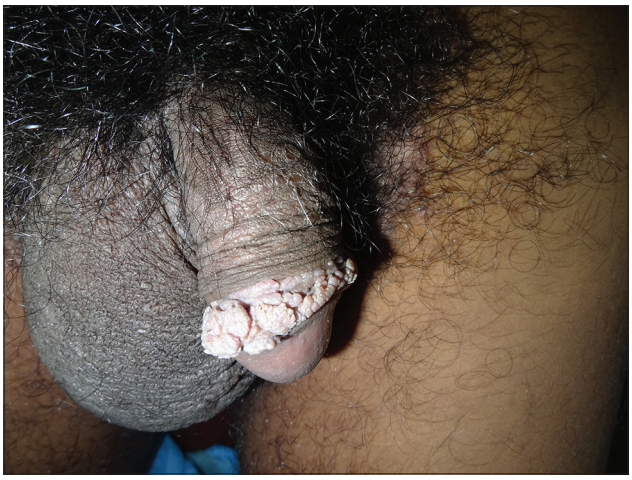

Figure 2: Verrucous growth over the coronal sulcus (condyloma acuminata) extending over glans penis

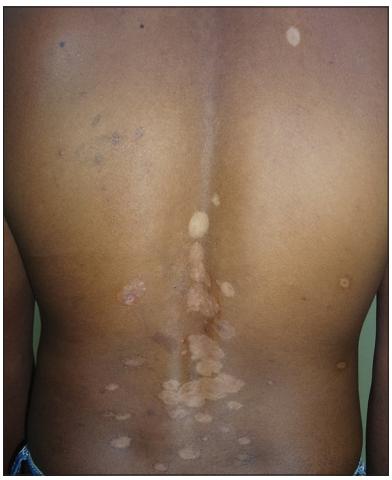

Figure 3: Complete clearance of the psoriatic lesions of the back

Figure 4: Complete clearance of genital warts after podophyllin application

and/or gut colonization by *Staphylococcus aureus*, *Malassezia*, and *Candida albicans*.[10] Also in systemic lupus erythematosus; a strong conjunction with HPV infection was found in a study in UK women with a recent systemic lupus erythematosus diagnosis wherein they had disturbingly elevated levels of HPV infections (particularly with European HPV-16 variants at a high viral load), abnormal cervical cytology, and squamous intraepithelial lesions.[11]

But psoriasis manifesting after HPV infection presenting as condyloma acuminata has not been reported till date. The patient developed condyloma acuminata of the external genitals prior to the development of psoriatic lesions. The patient developed lesions of psoriasis initially in the groin which then spreads to other body areas. The patient gave a history of multiple unprotected sexual contacts with multiple sex workers over last 3–4 years. Though his HIV testing is nonreactive the patient could be in the window period. Association period. Association of psoriasis and HIV is well established; through the CD_{4+} T-cells depleted in the HIV leading to exacerbation of psoriasis.

Inflammatory state after infection leads to the upregulation of nerve growth factor (NGF) synthesis, NGF influences the pathological features of psoriasis – keratinocyte proliferation, angiogenesis, T-cell activation. NGF production in fibroblast, endothelial cell, glial cell induce by cytokines like interleukin (IL)-1, tumor necrosis factor-alpha, and IL-6. Also, immune cells of the innate and acquired immunity shows enhanced NGF synthesis after stimulation with specific antigen and cytokines. The NGF plays a critical role in the initiation, maintenance, the perpetuation of the chronic inflammatory process. Both T-lymphocyte and keratinocyte plays an important role in the pathogenesis of psoriasis. The uniqueness of psoriatic keratinocytes is not well established, but a high level of NGF is reported in the psoriatic prone skin as compared to normal skin; psoriatic skin primed to make the increased level of NGF.[12] We, hereby conclude that this case may have developed due to primary HPV infection triggering psoriatic lesions, or the association could be a mere coincidence. The patient after clearance of skin and genital lesions was lost to follow-up. Long-term follow-up and number of cases are required to conclude that HPV could trigger psoriasis.

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Conflicts of interest
There are no conflicts of interest.

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