Persistent papular varicella in an immunocompetent male

Sir,

Varicella caused by the varicella zoster virus (VZV) is characterized by fever concurrent with a self-limiting rash on the skin and sometimes the mucosa. The rash typically begins as macules, rapidly progresses to papules, followed by a vesicular stage and crusting of lesions. However, many atypical variants such as chronic, verrucous, hemorrhagic, persistent, abortive and varioliform are known, especially in immunocompromised individuals.¹ Varicella zoster virus has also been reported to cause granulomatous, folliculitis like eruptions and lichenoid reactions.² Here, we report a case of varicella with papular lesions of more than 1 month duration in the absence of immunosuppression.

A 25-year-old healthy male was admitted to the Infectious Diseases Ward at Government Medical College, Kozhikode, India with fever and rash of 7 days duration. There was no history of any skin disease prior to the onset of lesions. He gave a history of contact with an individual suffering from varicella a few days prior to the eruption and was treated with acyclovir 800 mg five times daily. After 2 days of starting treatment with acyclovir, he developed erythematous lesions on his face that later progressed to involve the whole body. He also complained oforal erosions and a burning sensation in the eyes and was transferred to the Dermatology Ward with a clinical suspicion of drug-induced rash due to acyclovir or a possibility of Kaposi's varicelliform eruption. Dermatological examination revealed multiple, erythematous spiny papules, some showing crusting, present on the chest, abdomen and back; hyperkeratotic papules on dorsa of hands and feet; erythematous and purpuric papules on legs, palms and soles and multiple papules on the scalp [Figure 1a-c]. Oral mucosa examination revealed multiple pin-point vesicles on the soft palate and sides of tongue. Blood examination was normal except for elevated C-reactive protein levels. The serum was negative for human immunodeficiency virus, hepatitis B, C virus antibodies and CD4 count was normal. Tzanck smear revealed multinucleated giant cells. Fluid from an intact vesicle tested positive for varicella zoster virus DNA by real-time polymerase chain reaction and negative for enterovirus (Fast Track Diagnostics Inc., Luxembourg; performed at Manipal

Centre for Virus Research). Skin biopsy taken from the papular lesions revealed hyperkeratosis, hypergranulosis, acanthosis, absence of cytolysis and the presence of multinucleated giant cells in the lower layers of epidermis, dermis, within the follicles and sweat glands [Figure 2a–d]. Indirect immunofluorescence assay from papules confirmed varicella zoster virus infection (Chemicon Inc., USA) and demonstrated green fluorescence of multinucleated giant cells in the lower layers of the epidermis [Figure 3]. On the basis of these findings, the patient was diagnosed as atypical varicella and was continued on parenteral acyclovir. However, skin lesions showed no tendency for regression even though there were no new lesions. Subsequently, patient developed varicella pneumonia and succumbed to illness 2 months after the onset of eruption.

Varicella is characterized by polymorphous lesions in a centripetal distribution with minimal involvement of distal extremities. Unusual presentations such as verrucous or hemorrhagic and atypical durations such as chronic or persistent varicella of more than 1 month duration is well known in immunocompromised patients, such as post-renal transplant and HIV infection. Altered host response due to decreased cell-mediated immunity and altered varicella viral gene expression may precipitate the development of hyperplastic and hyperkeratotic lesions.¹ Verrucous lesions of varicella in HIV infected individuals are frequently associated with resistance to thymidine kinase-dependent antiviral agents.

Baccaredda in 1947 reported varioliform varicella with generalized pustules in adults. Ronaldson and Kelleher described an abortive form of varicella characterized by an arrest of the normal development of lesions that would not progress beyond the papular or vesicular stage.³

Atypical varicella with involvement of palms and soles has been reported in children though precipitating factors are unknown. Few reports of atypical varicella with palm and sole involvement showed antibody positivity against Coxsackie virus, suggesting subclinical hand, foot and mouth disease.^{4,5} Our patient had papular lesions on both palms and soles, but the possibility of a co-infection with Coxsackie virus was ruled out.

Histopathology of our patient correlated with the clinical appearance of papules as it depicted hyperkeratosis, acanthosis and hypergranulosis without cytolysis. There was no papillomatosis or pseudoepitheliomatous hyperplasia characteristic of verrucous varicella. The presence of multinucleated giant cells in the dermis, sweat ducts and hair follicles, which are rarely reported, might also have contributed to the papular appearance and persistent nature of the lesions.^{6,7}

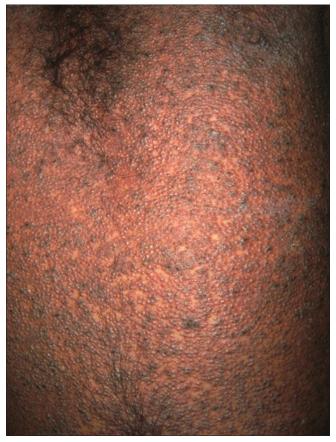


Figure 1a: Multiple erythematous spiny papules, some showing crusting on chest and abdomen

Our case depicts the co-existence of spiny, hyperkeratotic and purpuric papules of persistent nature in a generalized distribution, including palms and soles in an immunocompetent individual that preceded a dreadful complication. However, acyclovir resistance could not be demonstrated in our case due to non-availability of the test. Dermatologists should be aware of the fact that an innocuous disease can have an unusual presentation of unusual duration even in the absence of immunosuppression; which might predict a poor prognostic outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that the names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Figure 1b: Hyperkeratotic papules on dorsa of hands

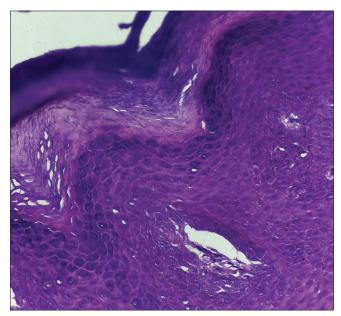


Figure 2a: Hyperkeratosis, hypergranulosis, acanthosis (H and E, $\times 200$)



Figure 1c: Purpuric papules on lower legs and soles

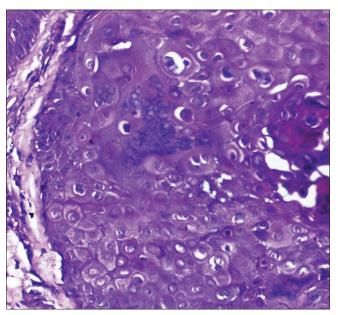


Figure 2b: Multinucleated giant cells in the lower layers of epidermis (H and E, $\times 400)$

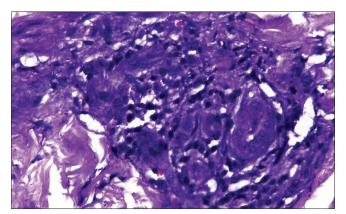


Figure 2c: Multinucleated giant cells in sweat ducts (H and E, ×400)

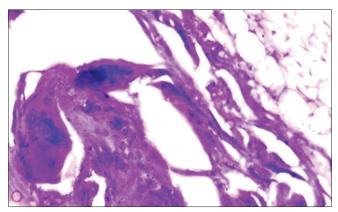


Figure 2d: Multinucleated giant cells within hair follicle (H and E, ×400)

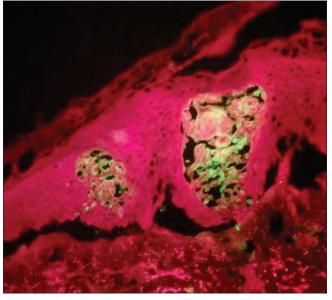


Figure 3: Green fluorescence of multinucleated giant cells in the lower layers of epidermis (×200)

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Anza Khader, Kunnummel Muhammed, Sarita Sasidharan Pillai, Chettithodi Sivasankaran Bindhu¹, Reena Mariyath, Pardeep Mann

Departments of Dermatology and Venereology and ¹Pathology, Government Medical College, Kozhikode, Kerala, India

Correspondence: Dr. Anza Khader, 5/1986 B, "Maskan" Rajiv Nagar Colony, PO Puthiyara, Kozhikode - 673 004, Kerala, India. E-mail: anzashaan@gmail.com

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