

Symmetrical Keratoderma On The Palms, Soles And Elbows Associated With Acute Hepatitis B Viral Infection

S Funaki, D Tsuruta, M Ishii, K Nakagawa

Citation

S Funaki, D Tsuruta, M Ishii, K Nakagawa. *Symmetrical Keratoderma On The Palms, Soles And Elbows Associated With Acute Hepatitis B Viral Infection*. The Internet Journal of Dermatology. 2010 Volume 8 Number 2.

Abstract

HB virus is well-known to cause several skin manifestations. We present an unusual case of keratoderma associated with acute hepatitis B. Hepatitis B antigens were detected in a 24-year-old Japanese woman as part of a regular health examination. Three weeks later, she felt common cold-like symptoms, and noticed eruption of her palms and soles. Clinical examination showed well-circumscribed erythematous hyperkeratosis on the palms and soles, with partial extension into the dorsal aspect of her hands and feet. In addition, she had well-demarcated erythematous hyperkeratotic plaques on both elbows. Histopathology obtained from the right palm showed hyperkeratosis, acanthosis, and focal parakeratosis without granular degeneration. We consider that the keratoderma appeared in our patient must be related to acute hepatitis B virus infection. The differential diagnosis of our case was made for acquired PPK (APPK), hereditary PPK (HPPK), PSE, PRP, and psoriasis inverse. The most plausible diagnosis of keratoderma in our case is PRP, because PRP has reported to be precipitated with viral infections including hepatitis A and C. Another possibility is APPK associated with hepatitis B.

CASE REPORT

Hepatitis B antigens were detected in a 24-year-old Japanese woman in laboratory tests done as part of a regular health examination. Since she had no symptoms, she left untreated. Three weeks later, she felt common cold-like symptoms with general malaise, and noticed red spots appearing on the palms of her hands and soles of her feet. She visited a local dermatologist for an examination. The laboratory tests showed an abnormally high level of liver enzymes, so she was referred and admitted to our hospital. In a clinical examination upon admission, she had well-circumscribed, painful erythematous hyperkeratosis with partial fissure on the palms of her hands and soles of her feet, with partial extension into the dorsal aspect of her hands and feet (Fig. 1A, B). In addition, she had well-demarcated erythematous hyperkeratotic plaques on both elbows (Fig. 1C). Marked improvement of the lesion on the palm one week after the initial hospitalization (Fig. 1D).

Figure 1

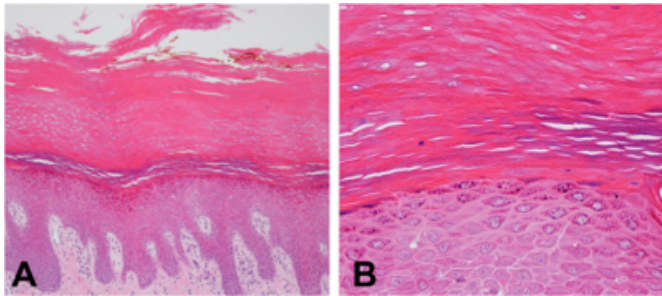
Figure 1 Clearly-demarcated erythema and severe hyperkeratosis with fissure were observed on both palms (A) and feet (B). Hyperkeratotic plaques were found on both elbows (C). Marked improvement of the lesion on the palm one week after the initial hospitalization (D).



Histopathology obtained from the right palm showed hyperkeratosis, acanthosis, and focal parakeratosis without granular degeneration (Fig. 2A, B).

Figure 2

Figure 2 Histopathology of the right palm showed hyperkeratosis, acanthosis, and focal parakeratosis (H&E stain magnification (A) $\times 100$, (B) $\times 400$).



Laboratory tests showed severe liver function damage (AST 1235 IU/l [normal, 10–40 IU/l], ALT 2256 IU/l [normal, 5–45 IU/l], g-GT 118 IU/l [normal, 10–40 IU/l], HB-antigen positive, Hepatitis C virus-antibody negative, IgM-HBc-antibody positive). Daily glycyrrhizin injections and rest markedly improved her general malaise and her hepatic enzyme abnormalities 2 months after the first admission (AST: 60 IU/l, ALT 47 IU/l). In addition, all of the skin symptoms diminished within 3 weeks with use of only urea cream (Keratinamin[®] ointment, Kowa Co. Ltd., Japan) on the affected region (Fig. 1D). The patient told us that she did not use any home remedies. There were no recurrences afterward, and she did not receive prophylactic dermatological treatments.

DISCUSSION

The lesion in our case appeared and remitted in relation with the onset of acute HB and corresponded with the titer of her liver enzymes. Moreover, after the liver symptoms improved, the lesion never relapsed. Therefore, we consider that the keratoderma appeared in our patient must be related to acute hepatitis B virus infection. The differential diagnosis of our case was made for acquired PPK (APPK), hereditary PPK (HPPK), PSE, PRP, and psoriasis inverse. We excluded HPPK and PSE because there was no family history, no early childhood onset and its transient nature. We excluded psoriasis inverse because there was no vasodilation or inflammatory cell infiltration in the histopathology [4]. Therefore, we focused on APPK or PRP as the differential diagnosis of our case. PRP is characterized by palmoplantar hyperkeratosis and erythematous perifollicular papules that progress into sheets of reddish-orange, scaly erythroderma [3]. Although we do not have biopsy specimen from the

elbow (the patient did not permit us to do biopsy from her elbow), the most plausible diagnosis of keratoderma in our case is PRP, because PRP has reported to be precipitated with viral infections, such as cytomegalovirus infection, herpes simplex infection, infectious mononucleosis, human immunodeficiency virus infection, hepatitis A and C [6-9]. If this is the case, this is the first report of PRP associated with hepatitis B. Another possibility is APPK associated with hepatitis B. APPK is a diverse entity of diseases that are clinically characterized by abnormal thickening of the skin on the palms of the hands and soles of the feet [10]. APPK is categorized into keratoderma climactericum, drug-related, malnutrition-associated, chemically-induced, systemic disease or infection-related, malignancy-associated, dermatoses-related, and idiopathic types [10]. If our patient is APPK, systemic disease or infection-related APPK caused by an acute HB viral infection is the diagnosis. However, there have been no reports of APPK associated with hyperkeratosis on the elbow, suggesting that this case is not APPK, but we still cannot exclude this possibility.

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Author Information

Satoko Funaki, MD, PhD

Department of Dermatology, Osaka City University Graduate School of Medicine

Daisuke Tsuruta, MD, PhD

Department of Dermatology, Osaka City University Graduate School of Medicine

Masamitsu Ishii, MD, PhD

Department of Dermatology, Osaka City University Graduate School of Medicine

Koichi Nakagawa, MD

Department of Dermatology, Saiseikai Tondabayashi Hospital