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# Inverse Psoriasis with Autoimmune Hepatitis

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Dear Editor:

Inverse psoriasis is characterized by a lack of or lesser degree of scaling at the intertriginous areas, which is the most prominent dissimilarity between classical plaque-type psoriasis and inverse psoriasis. Psoriasis is widely associated with comorbidities including but not limited to autoimmune diseases, neurological disorders, cardiometabolic diseases, and inflammatory bowel disorders[1].

A 49-year-old woman visited Chung-Ang University Hospital with erythematous to brownish scaly patches on both inguinal and axillary areas that had persisted for 4 years (Fig. 1A). Skin biopsies of the patient's right inguinal area demonstrated parakeratosis, psoriasiform hyperplasia, mild spongiosis, dilated blood vessels at the tip of the dermal papillae, and perivascular lymphocytic infiltration (Fig. 1B). The patient was treated for inverse psoriasis with a topical calcipotriol monohydrate and betamethasone dipropionate ointment (Xamiol gel; LEO Pharma A/S, Ballerup, Denmark) twice daily. This treatment resulted in improvement of the lesions.

Our patient subsequently visited the gastroenterology department with severe symptoms accompanied by myalgia, nausea, and profound jaundice 2 weeks after being diagnosed with inverse psoriasis (Fig. 1C). Laboratory findings revealed abnormal liver function test results (total bilirubin, 5.1 mg/dl; direct bilirubin, 4.0 mg/dl; aspartate aminotransferase (AST), 749 IU/L; alkaline phosphatase (ALP), 911 IU/L; lactate dehydrogenase, 355 IU/L; gamma-glutamyl transpeptidase, 68 IU/L), negative hepatic viral marker and prolonged prothrombin time (14.4 sec; normal range, 10.4～12.5). The patient's antinuclear antibody (Ab) level was also elevated (1:80). And other autoantibodies titers (anti-smooth muscle Ab [IgG, IgM], anti-mitochondrial Ab, anti-liver-kidney microsomal-1 Ab) were normal. A liver biopsy showed severe hepatic lobular and portoperiportal necrosis and inflammation with mixed lymphoplasma cells and eosinophils accompanied by periportal fibrosis (Fig. 1D). The patient was diagnosed with type 1 autoimmune hepatitis (AIH) and treated via systemic steroid administration, after which her symptoms and laboratory findings improved.

AIH is diagnosed by scoring system of the international AIH group in 1999[2]. In this system, the score of 15 points is indicative of 'definite AIH'. This patient's score was 18 points (female: +2, ALP/AST ratio＜1.5: +2, ANA 1:80: +2, viral marker negative: +3, drug history[−]: +1, alcohol[−]: +2, histological features +4 [interface hepatitis: +3, plasmacytic: +1], treatment response complete: +2). The diagnosis of affiliated skin lesions is varied, including urticarial, vitiligo, psoriasis, impetigo, erythema nodosum, prurigo nodularis, lichen planus, vasculitis, and pyoderma gangrenosum[3].

Recurrence of psoriasis in the same lesion suggests that tissue resident memory T ($T_{RM}$) plays a role in psoriasis pathogenesis. According to a previous study, in resolved psoriatic lesions, CD8+ T cells expressing the $T_{RM}$ marker, CD103 produce interleukin (IL)-17 and CD4+ T cells produce IL-22, providing evidence of the role of $T_{RM}$ cells in psoriasis[4]. The dysregulation of $T_{RM}$ cells may explain the connection between psoriasis and autoimmune diseases. A Danish study speculated that AIH could escalate in patients with psoriasis because the clinical characteristics of

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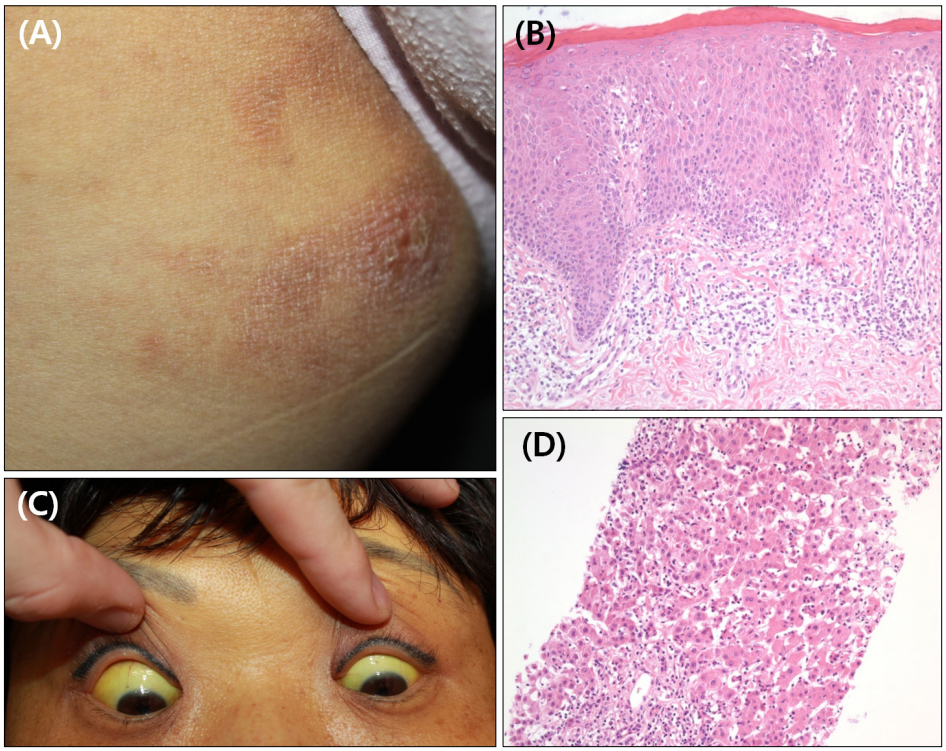


**Fig. 1.** (A) Erythematous to brownish scaly patches on the right inguinal area. (B) Parakeratosis, psoriasiform hyperplasia, mild spongiosis, dilated blood vessels at the tip of the dermal papillae, and perivascular lymphocytic infiltration (H&E, ×100). (C) Profound jaundice with icteric sclera. (D) Severe hepatic lobular and portoperiportal necrosis and inflammation with mixed lymphoplasma cells and eosinophils accompanied by periportal fibrosis (H&E, ×200).

human auto-inflammatory diseases suggest that $T_{RM}$ cells play a role in their etiology[5]. However, because distinct pathogenic mechanisms underlying classical-type psoriasis and inverse psoriasis have not been identified, $T_{RM}$ cells might perform an integral function in the mechanistic relationship between inverse psoriasis and AIH. This case has a limitation that inverse psoriasis and AIH could happen possibly coincidentally. However, our case contributes to the increasing body of evidence that points to inverse psoriasis-associated comorbidities, including autoimmune inflammatory disorder.

## CONFLICTS OF INTEREST

The authors have nothing to disclose.

## REFERENCES

1. Omland SH, Gniadecki R. Psoriasis inversa: a separate identity or a variant of psoriasis vulgaris? Clin Dermatol 2015;33:456-461.
2. Alvarez F, Berg PA, Bianchi FB, Bianchi L, Burroughs AK, Cancado EL, et al. International Autoimmune Hepatitis Group Report: review of criteria for diagnosis of autoimmune hepatitis. J Hepatol 1999;31:929-938.
3. Wong GW, Heneghan MA. Association of extrahepatic manifestations with autoimmune hepatitis. Dig Dis 2015;33 Suppl 2:25-35.
4. Cheuk S, Wikén M, Blomqvist L, Nylén S, Talme T, Ståhle M, et al. Epidermal Th22 and Tc17 cells form a localized disease memory in clinically healed psoriasis. J Immunol 2014;192:3111-3120.
5. Jensen P, Egeberg A, Gislason G, Hansen PR, Thyssen JP, Skov L. Increased risk of autoimmune hepatitis in patients with psoriasis: a danish nationwide cohort study. J Invest Dermatol 2016;136:1515-1517.