

Successful Treatment of Papuloerythroderma of Ofuji Associated with *Helicobacter pylori* Infection

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Papuloerythroderma of Ofuji (PEO) is a rare skin condition characterized by widespread pruritic eruptions of coalescing erythematous-brown papules sparing skin folds, known as the “deck-chair sign”. While most cases are idiopathic, PEO can be associated with atopic dermatitis (AD), malignancies such as gastric carcinoma, and infectious diseases (1).

Infections are implicated in a small percentage of PEO cases. Viral infections such as human immunodeficiency virus (HIV) (2) and hepatitis C virus (HCV) (3) have been associated with PEO, with reports of resolution following successful antiviral treatment. Other infections, including bacterial sepsis and parasitic infestations, have also been reported (4). However, the role of bacterial infections, particularly *Helicobacter pylori* (*H. pylori*), in PEO has not been well established. Recent reports have also suggested that PEO may rarely develop following BNT162b2 mRNA COVID-19 vaccination, possibly due to immune dysregulation or viral reactivation (5).

We report a case of PEO associated with *H. pylori* infection that achieved complete remission after eradication therapy. This case highlights the importance of considering *H. pylori* as a potential trigger in PEO and suggests that bacterial eradication may be a therapeutic option in select cases.

CASE REPORTS

A man in his 70s presented with a 1-year history of persistent erythema and severe pruritus affecting his entire body. Diffuse faint erythema was observed across the entire body, with sparing of skin folds on the abdomen (Fig. 1). Prior treatments with topical

corticosteroids and antihistamines were ineffective. His medical history included untreated *H. pylori* infection, and a gastric ulcer diagnosed 3 years earlier. He had no history of AD or other significant dermatological conditions. On physical examination, no superficial lymphadenopathy was noted. A biopsy was performed, including both lesional and non-lesional skin (Fig. 2A). In the lesional area, hyperkeratosis and epidermal thickening were observed compared with the non-lesional area (Fig. 2B and C). Additionally, minimal lymphocytic infiltration was noted around the dermal blood vessels. Laboratory tests showed negative antibody titres for HIV and HCV. Anti-*H. pylori* IgG antibody was positive at 12 before treatment. However, post-eradication antibody levels were not available. Serum IgE and TARC levels were not measured. No significant abnormalities, including eosinophilia, were found. Computed tomography and endoscopic examinations were unremarkable except for the untreated gastric ulcer noted previously. During the diagnostic period, the patient did not receive COVID-19 vaccination. Based on these findings, the patient was diagnosed with PEO.

Despite treatment with topical corticosteroids and oral antihistamines, symptoms failed to improve during the diagnostic period. The patient underwent *H. pylori* eradication therapy with a proton pump inhibitor, amoxicillin, and clarithromycin. One month after starting the eradication treatment, both the rash and pruritus significantly improved. By 3 months, the rash had resolved completely (Fig. 3), and no recurrence was observed during an 18-month follow-up.

DISCUSSION

A recent systematic review involving 135 patients with PEO reported a mean disease duration of 2.6 years, with only a portion achieving complete remission using therapies such as oral corticosteroids, PUVA, and retinoids (6). No standardized treatment approach has been established, and recurrence or refractory cases remain

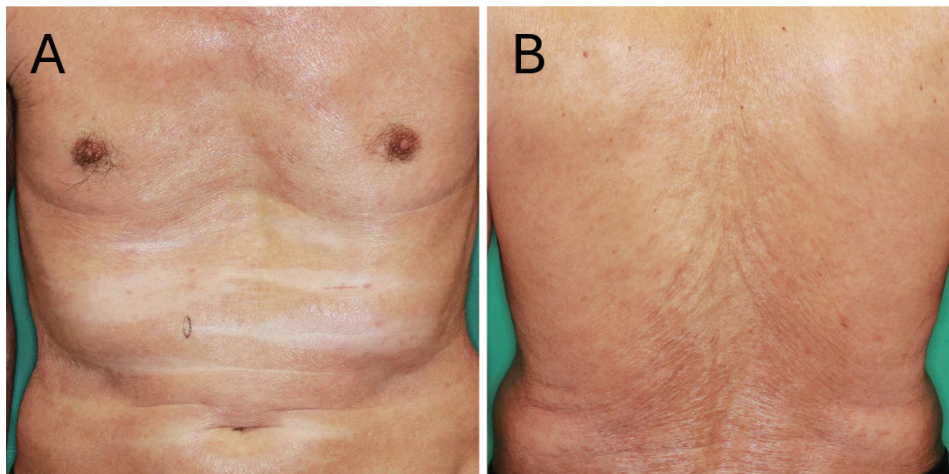


Fig. 1. Clinical pictures at the initial visit. The clinical presentation at the initial visit, with erythema observed on (A) the chest and abdominal region and (B) the back. The erythematous lesions spared the abdominal skin folds, presenting the characteristic “deck-chair sign”.

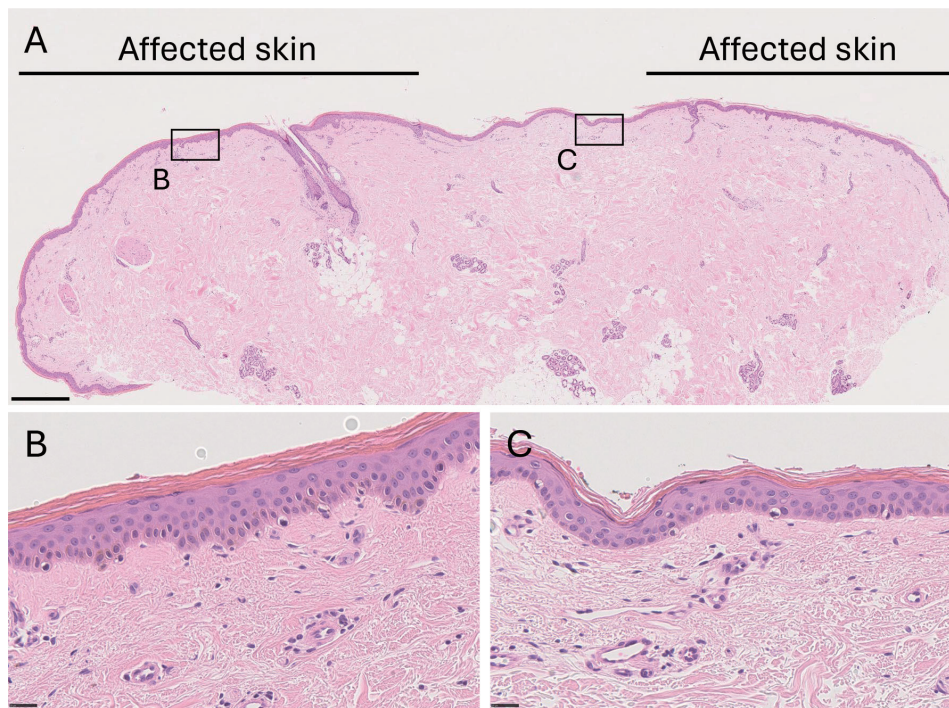


Fig. 2. Histopathological findings. (A) Low-power field of the areas marked in Fig. 1A. (B and C) High-power view of the area within the square in (A). (B) A magnified view of the lesional skin. (C) A magnified view of the non-lesional skin for comparison. The scale bars in Figs 2A, 2B, and 2C represent 500 μ m, 25 μ m, and 25 μ m, respectively.

common. Notably, the review also described 1 case of spontaneous remission without any therapeutic intervention, suggesting that while rare, spontaneous resolution may occur. While spontaneous remission may occur in rare cases, the majority of patients require active therapeutic intervention to achieve clinical resolution.

In this context, our case provides insight into a potentially treatable infectious trigger for PEO. This

case highlights the potential role of *H. pylori* infection in the pathogenesis of PEO, as complete remission was achieved following eradication therapy. While PEO has traditionally been associated with malignancies, AD, and immune dysregulation, infectious agents, including viruses and bacteria, have also been implicated in its pathogenesis. Our findings suggest that *H. pylori* may act as a triggering factor in certain cases of PEO.

H. pylori is a well-established aetiological agent of gastric diseases, but accumulating evidence indicates its involvement in various dermatological conditions, including chronic urticaria, rosacea, psoriasis, and vasculitis (7). In these disorders, eradication therapy has been associated with clinical improvement, suggesting a pathogenic role. The mechanisms linking *H. pylori* to skin diseases remain speculative but may involve systemic immune activation, molecular mimicry, and chronic inflammation (8, 9). Chronic *H. pylori* infection can lead to increased production of pro-inflammatory cytokines, such as IL-1, IL-6, and TNF- α , which may contribute to cutaneous inflammation (10). Additionally, molecular mimicry between *H. pylori* antigens and host proteins may promote autoantibody production, potentially leading to immune-mediated dermatological manifestations. The formation of circulating immune complexes in response to *H. pylori* infection has also been proposed as a mechanism contributing to vascular inflammation in the skin. Furthermore, alterations in gut barrier integrity caused by *H. pylori* may enhance antigen absorption and systemic immune activation



Fig. 3. Clinical appearance 3 months after eradication therapy. The eruptions were resolved.

(11), a phenomenon observed in other inflammatory skin disorders.

In our patient, the absence of other identifiable causes, the temporal association between *H. pylori* eradication and symptom resolution, and the sustained remission over 18 months strongly support a causal link between *H. pylori* infection and PEO. Although spontaneous remission cannot be entirely excluded, the rapid and complete resolution following eradication therapy suggests that bacterial infection played a significant role in disease activity.

These findings highlight the need to screen for *H. pylori* in cases of unexplained PEO, particularly when other triggers have been excluded. Non-invasive tests, such as the urea breath test or stool antigen test, can facilitate diagnosis, and eradication therapy may serve as a potential therapeutic approach in select cases. While further studies are needed to clarify the precise role of *H. pylori* in PEO and establish guidelines for screening and treatment, this case provides valuable insight into the possible contribution of bacterial infections to PEO pathogenesis.

The authors have no conflicts of interest to declare.

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