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CASE REPORT

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Herpes zoster occurring over the site of tinea cruris—A case report based on the model of T-cell exhaustion in the Ruocco's immunocompromised cutaneous district

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Key Clinical Message

Immunocompromised district refers to the area of the skin with altered immune response predisposing secondary diseases to develop in an immunocompetent individual. This might be explained by the theory of T-cell exhaustion which is characterized by the impairment of the effector function of antigen-specific T cells due to chronic persistence of the primary antigen. T-cell exhaustion model is not well known; however, it serves as a newer concept in the pathogenesis of diseases occurring simultaneously over the same site. Thus, it is not surprising to have two different infectious or non-infectious dermatoses over the same site one preceding the other as observed in our patient. The concept of immunocompromised district and T-cell exhaustion is a rare phenomenon; however, it should be identified by the treating physicians/dermatologists for the optimum management of the atypical presentation of the diseases.

KEYWORDS

epitope sharing, herpes zoster, immunocompromised cutaneous district, T-cell exhaustion, tinea cruris

1 | INTRODUCTION

T-cell exhaustion is defined as the change in the phenotype observed in T cells under antigen persistence forming Ruocco's immunocompromised cutaneous district which is a localized area of skin with altered immunity over which secondary diseases develop in an immunocompetent individual.[1]

Immunocompromised district is also called locus minoris resistentiae which is actually a site of the body offering lesser resistance and is susceptible to opportunistic infections, tumors, and immune reactions.[2] Impairment of effector function and proliferative capacity of responding antigen-specific T cells ultimately affect the ability to confer host protection. We hereby present a rare case of herpes zoster concurrently occurring at the site of tinea cruris.

2 | CASE PRESENTATION

A 40-year-old female with no known comorbidities presented to our OPD with itchy erythematous annular

scaly plaques over the bilateral buttocks involving the natal cleft for 3 months. Potassium hydroxide mount was prepared from the scales obtained from the active border which showed long-branching septate fungal hyphae (Figure 1A). The patient was started on oral itraconazole 100 mg twice daily, topical sertraconazole twice daily local application, and oral levocetirizine 5 mg twice daily. The patient was advised to maintain proper hygiene and was planned for follow-up after 3 weeks.

However, patient re-visited our OPD after 7 days with painful grouped erythematous papulovesicular lesions on her left buttock for last 3 days within an area of tinea (Figure 1B). Tzanck smear was prepared from the floor after deroofing of the vesicle which showed multinucleated giant cells and acantholytic cells (Figure 1C). She denied the use of oral or topical immunosuppressive agents, trauma, and irradiation at the involved site. Polymerase chain reaction for varicella zoster virus DNA was not done as it was not available at our center. The serology for HIV was negative. The patient was diagnosed to have herpes zoster involving the S1-S2 dermatome on the left on the background of tinea cruris and the patient was treated with acyclovir 800 mg five times a day for 7 days.

After 7 days of treatment with acyclovir, the papulovesicular lesions healed completely with areas of hypopigmentation as depicted in Figure 2, and oral itraconazole, topical sertraconazole, and levocetirizine were continued for the next 2 weeks for tinea cruris. The patient was advised to follow-up after 2 weeks of antifungal therapy; however, she lost follow-up in between.

3 | DISCUSSION

After the resolution of primary infection, VZV-specific $CD4^+$ memory T-cells circulate through the skin via skin-homing proteins, cutaneous leukocyte antigen (CLA), and CC-chemokine receptor 4 (CCR4) which is host protective.[3] Reactivation of varicella zoster virus in the dorsal root ganglion causes herpes zoster which is due to the alteration in the memory T-cell triggered by increasing age, stress, immunocompromised status, and the use of immunosuppressive drugs.[4]

Herpes zoster appearing at the site of dermatophytosis can be explained by the phenomenon of T-cell exhaustion due to persistent dermatophytic antigens expressed at these sites known as immunocompromised sites as observed in our patient.[1] The proposed mechanism of T-cell exhaustion includes up-regulation of intrinsic inhibitory T-cell receptors (PD-1, TIM-3, CTLA-4, LAG-3, 2B4, and CD160), downregulation of cytokine receptors (IL-2Rβ, IL-4Rα, IL-7Rα, and IL-15Rα), increase in regulatory T cells, and suppressive cytokines (IL-10, TGF-B). In addition, exhausted T cells undergo impaired function including effector cytokines production, proliferation, cytokine-mediated self-renewal, differential expression of transcription factors, and altered regulation of signaling molecules.[5,6]

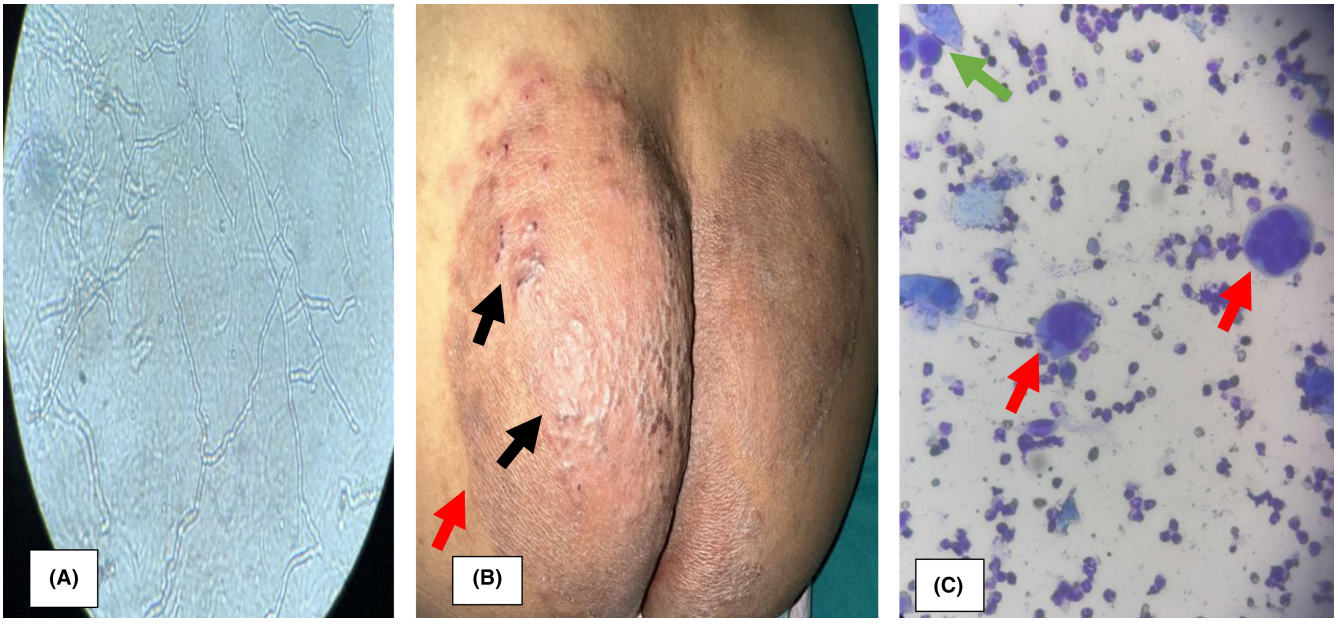

FIGURE 1 (A) Potassium Hydroxide Mount shows long branching septate hyphae from skin scrappings. (B) shows grouped papulovesicular lesions with crust (black arrows) on the S1-S2 dermatome, left side on the background of Tinea cruris (red arrow). (C) Tzanck smear (40×) of vesicular floor shows multinucleated giant cells (red arrows) and scattered acantholytic cells (green arrow).

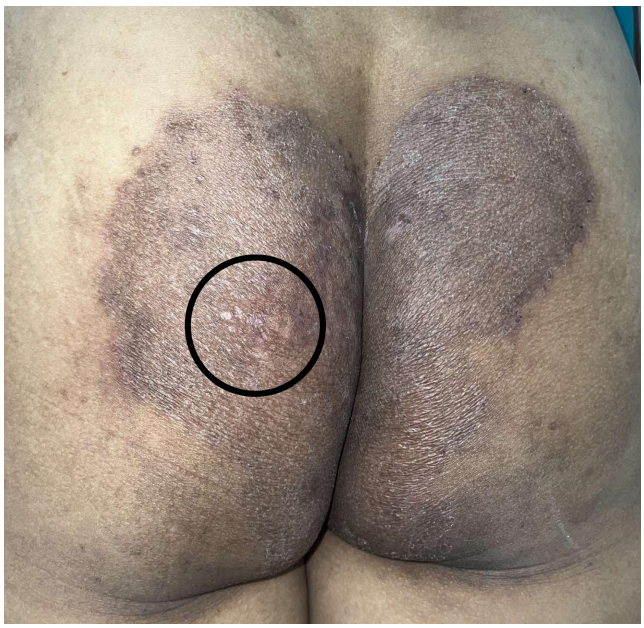

FIGURE 2 Complete healing of Lesions of Herpes Zoster after treatment with acyclovir with areas of hypopigmentation (black circle).

Similarly, the function of the dendritic cell is modulated by down-regulation of MHC and co-stimulatory molecules, differentiation into a tolerogenic subset, and inhibition of dendritic cell development by altered production of IFN. Regulatory T cells decrease the capacity of dendritic cells to prime T cells and alter T-cell activation signaling to diminish the proliferation and function of antigen-specific T cells.[5,6]

In our patient, chronic persistence of dermatophytic antigen might have caused exh-austion of dermatophytic specific T cells which via epitope sharing (e.g glycoproteins) between the dermatophytes and varicella zoster virus might have altered the varicella zoster specific $CD4^+$ memory T cells leading to reactivation of va-ricella zoster virus over the site of tinea cruris.[7,8]

There is a paucity of literature showing dermatoses of immunocompromised cutaneous district. Verma et al. in 2020 reported herpes zoster in three patients and Varicella in one patient over sites of healed and active superficial dermatophytosis respectively explained by the principle of T-cell exhaustion.[1] Similarly, Kapoor P et al. and Ghosh et al. reported molluscum contagiosum and lichen planus limited to the sites of dermatophytosis, respectively.[9,10]

4 | CONCLUSION

Although rare, it is not surprising to have two different infectious dermatoses over the same site one preceding the other as observed in our patient. Thus, it is important for all practicing dermatologists to identify an immunocompromised cutaneous district that can result in such a rare presentation of the disease.

AUTHOR CONTRIBUTIONS

Mahesh Mathur: Conceptualization; formal analysis; supervision; visualization; writing – original draft; writing – review and editing. **Neha Thakur:** Conceptualization; investigation; methodology; supervision. **Sunil Jaiswal:** Data curation; formal analysis; visualization; writing – original draft; writing – review and editing. **Srijana Maharjan:** Conceptualization; investigation; methodology; visualization. **Supriya Paudel:** Investigation; methodology; supervision; visualization. **Anjali Shrestha:** Data curation; formal analysis; methodology; supervision.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in repository name, [doi: 10.1002/ccr3.8067], [reference number: CCR38067].

CONSENT STATEMENT

The patient in this manuscript has given written informed consent for the use of their case details (including photographs) for publication.

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REFERENCES

1. Verma SB, Gupta S, Yadav A, Ruocco E. Varicella and herpes zoster appearing over sites of tinea corporis, hitherto unreported examples of immunocompromised districts: a case series. *Int J Dermatol*. 2020;59:758-759.
2. Caccavale S, Kannangara AP, Ruocco E. The immunocompromised cutaneous district and the necessity of a new classification of its disparate causes. *Indian J Dermatol Venereol Leprol*. 2016;82:227-229.
3. Zerboni L, Sen N, Oliver SL, Arvin AM. Molecular mechanisms of varicella zoster virus pathogenesis. *Nat Rev Microbiol*. 2014;12(3):197-210.
4. Koshy E, Mengting L, Kumar H, Jianbo W. Epidemiology, treatment and prevention of herpes zoster. A comprehensive review. *Indian J Dermatol Venereol Leprol*. 2018;84:251-262.
5. Jin HT, Jeong YH, Park HJ, Ha SJ. Mechanism of T cell exhaustion in a chronic environment. *BMB Rep*. 2011;44(4):217-231.
6. Yi JS, Cox MA, Zajac AJ. T-cell exhaustion: characteristics, causes and conversion: T-cell exhaustion. *Immunology*. 2010;129:474-481.
7. Blake JS, Dahl MV, Herron MJ, Nelson RD. An immunoinhibitory cell wall glycoprotein (mannan) from Trichophyton rubrum. *J Invest Dermatol*. 1991;96(5):657-661.

8. Oliver SL, Yang E, Arvin AM. Varicella Zoster Virus glycoproteins: entry, replication, and pathogenesis. *Curr Clin Microbiol Rep*. 2016;3(4):204-215.
9. Ghosh SK, Bandyopadhyay D, Chatterjee G, Saha D. Wolf's isotopic response: large annular polycyclic lichen planus occurring on healed lesions of dermatophytosis. *J Eur Acad Dermatol Venereol*. 2009;23(3):355-356.
10. Kapoor P, Bansal S, Boparai AS, Kaur N, Brar AK, Khera U. Molluscum contagiosum localised to sites of superficial dermatophytosis. *Indian J Dermatol Venereol Leprol*. 2021;87(5):748.

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