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SGLT2 Inhibitor-Induced Onychomycosis in a Patient with IgA Nephropathy: A Case Report

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Arjun Dang[1], Mankul Goyal[2], Ashima Jain Vidyarthi[1] and Binish Jawed[1]*

[1]*Department of Microbiology, Dr Dangs Lab LLP, India*

[2]*Department of Dermatology, Sampurnam Holistic Clinic, India*

***Corresponding Author:** Binish Jawed, Department of Microbiology, Dr Dangs Lab LLP, C-2/1 SDA, Delhi, India.

Abstract

We report the case of a patient with a 2-year history of IgA nephropathy who was prescribed an SGLT-2 inhibitor. Within a month of initiating therapy, the patient developed onychomycosis, which was confirmed by fungal culture. Treatment with antifungal agents resulted in significant improvement. This case underscores the potential for fungal infections to occur in areas beyond the genito-urinary region in patients taking SGLT-2 inhibitors. Although the link between SGLT-2 inhibitors and onychomycosis is not well documented, our findings suggest a need for further investigation into this association and the risk factors involved.

Keywords: nail diseases; anti-fungal treatment; fungal infection; medication-induced nephropathy; sglt-2 inhibitor; onychomycosis

Abbreviations

IgA: Immunoglobulin A.
SGLT: Sodium Glucose Transport Protein.
KOH: Potassium Hydroxide.

Introduction

SGLT2 inhibitors, which are primarily prescribed for diabetes, function by inhibiting glucose reabsorption in the kidneys, leading to increased glucose excretion in the urine [1]. Recently, these inhibitors have been approved for the treatment of IgA nephropathy because of their anti-inflammatory and renoprotective effects [2, 3]. A known side effect of SGLT2 inhibitors is an increased risk of mycotic infections, particularly in the genitourinary tract, where elevated glucose levels in bodily fluids create an environment conducive for fungal growth [4, 5].

This case, however, presents an uncommon instance of onychomycosis, raising questions about the broader potential of SGLT2 inhibitors to induce fungal infections beyond the genitourinary area.

Case Presentation

Clinical history: A 55-year-old female, known case of IgA nephropathy for 2 years (on treatment), presented to our outpatient department (OPD) with complaints of discoloration and thickening of nails after one month of initiating SGLT-2 inhibitor therapy (dapaglifozin 5 mg once daily). She also had a history of scalp lichen planopilaris and suffering from obsessive-compulsive disorder, particularly related to compulsive washing. Physical examination revealed subungual hyperkeratosis and whitish discoloration of the nails.

Diagnosis: Upon clinical suspicion of onychomycosis, a potassium hydroxide (KOH) microscopic examination and fungal culture were conducted on samples from various toenails. Multiple clippings and scrapings were collected and processed accordingly. The nail clips were dissolved in 20% KOH and examined microscopically the following day, revealing no fungal elements. In adherence to the laboratory protocol, four culture tubes were inoculated: one set contained plain Sabouraud's dextrose agar (SDA) and SDA with chloramphenicol and was incubated at 37°C, and the other set was incubated at 25°C. All four fungal culture tubes yielded yeast-like colonies, which were identified as *Candida tropicalis* via CHROMagar Candida Plus medium (Figure 1) and the Vitek-2 Compact system (Biomerieux).

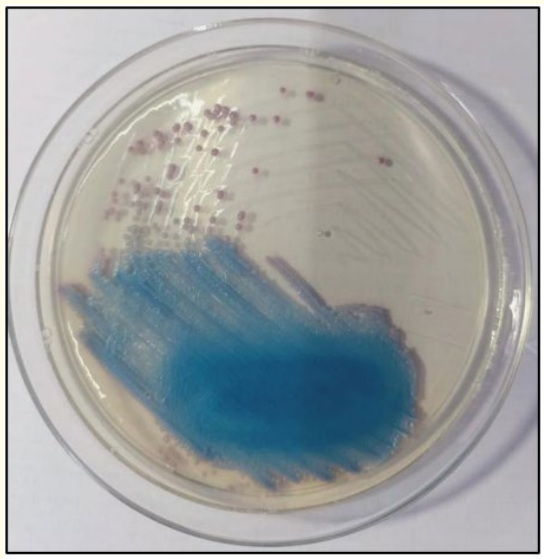

Figure 1: Growth of Candida tropicalis on CHROMagar Candida Plus medium.

Treatment: Consequently, the patient was prescribed an itraconazole soft gel formulation at a dosage of 130 mg once daily for six weeks, in addition to topical treatments with luliconazole 3% and tazarotene ointment. She has been steadily improving with this regimen (Figure 2).

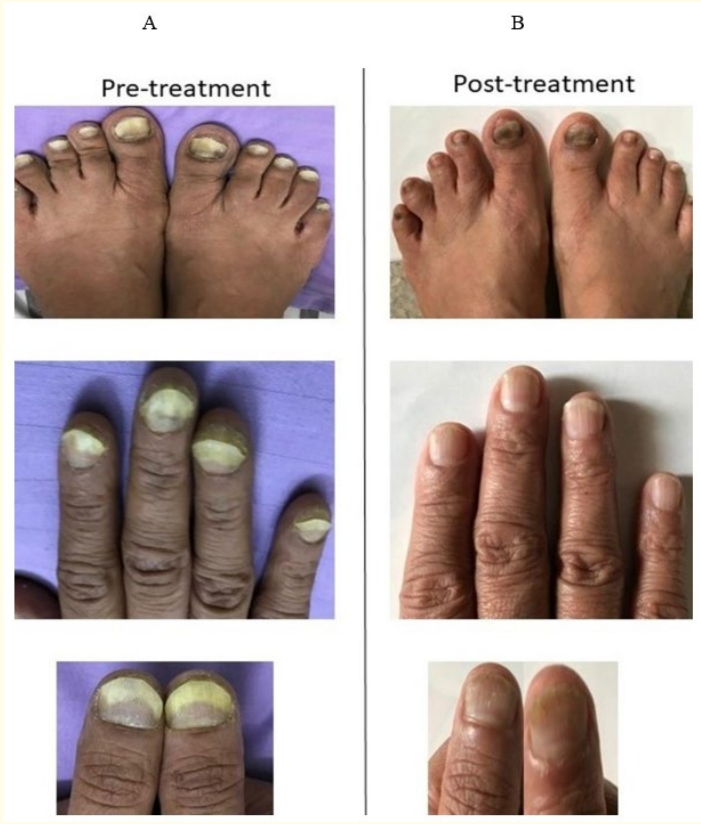

Figure 2: Pre- and post-treatment images demonstrate a potential case of SGLT2 inhibitor-induced onychomycosis in a patient with IgA nephropathy. In the left panel (A), the patient's nails show subungual hyperkeratosis and whitish discoloration prior to the initiation of antifungal therapy. The right panel (B) shows marked improvement in nail appearance following six weeks of treatment with oral itraconazole and topical antifungal agents. The resolution of discoloration and reduction in nail thickening suggest effective management of fungal infection.

Discussion

The occurrence of onychomycosis in our patient following the initiation of SGLT2 inhibitor therapy highlights important considerations regarding the broader spectrum of fungal infections associated with these medications, particularly in vulnerable populations. Although mycotic infections involving the genitourinary tract [6] are well documented with SGLT2 inhibitors, this case underscores the potential for fungal infections at extra-genital sites. The patient's clinical background includes IgA nephropathy, which may contribute to a degree of immune compromise, and obsessive–compulsive washing behavior, likely causing disruption of the skin and nail barrier. Both factors can predispose individuals to fungal colonization and infection. In addition to these underlying vulnerabilities, the use of dapagliflozin introduces another layer of complexity.

Dapagliflozin primarily targets SGLT2 to reduce glucose reabsorption in the kidneys, but its impact on other sodium–glucose transporters, such as SGLT3, is less explored [7, 8]. SGLT3 channels are expressed in sweat glands, and their inhibition may alter local glucose levels in sweat. Hyperhidrosis of the palms and soles, coupled with footwear that traps moisture, could lead to elevated glucose concentrations around the nail beds. This glucose-enriched microenvironment may promote fungal growth, as observed in this case

with the growth of *Candida tropicalis*.

The role of SGLT3 and sweat gland glucose transport is an emerging area of interest that may help explain localized fungal infections such as onychomycosis. Additionally, prolonged exposure to moisture, which is common in closed footwear, may exacerbate fungal proliferation by creating a conducive environment for infection.

This case reinforces the need for heightened vigilance in monitoring patients on SGLT2 inhibitors for fungal infections, including those at sites beyond the genitourinary tract. Patients with additional risk factors—such as immune compromise, excessive sweating, or habits that compromise skin integrity—may be particularly susceptible. Clinicians should consider regular dermatologic and nail examinations for these patients, particularly those presenting with signs of fungal infections.

Conclusion

Further studies are needed to clarify the mechanisms by which SGLT2 inhibitors, particularly their effects on extrarenal SGLT channels, might predispose patients to fungal infections. Prospective studies could explore whether patients receiving SGLT2 inhibitors are at greater risk for onychomycosis and assess preventive measures, such as optimizing hygiene practices, reducing moisture exposure, and early antifungal interventions. Additionally, understanding the specific conditions that enable fungal colonization—such as localized glucose dynamics—could inform strategies to mitigate this risk.

While SGLT2 inhibitors offer significant therapeutic benefits for managing diabetes and IgA nephropathy, their potential side effects, including fungal infections, warrant careful consideration. This case highlights the importance of recognizing fungal infections such as onychomycosis as possible adverse effects of SGLT2 inhibitor therapy, underscoring the need for a comprehensive approach to patient care that includes prevention, early detection, and timely management of such infections.

Conflict of interest

The author(s) declare that there are no conflicts of interest.

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