**Brief Report** 

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# Abrupt Development of Esophageal Candidiasis after Secukinumab Treatment in a Psoriatic Patient

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Dear Editor:

A 61-year-old male presented with a 40-year history of psoriasis on the whole body. He had previously been treated with methotrexate and topical betamethasone dipropionate/calcipotriol gel for two years in the local clinic. His medical history included diabetes mellitus (DM) and hypertension. Physical examination revealed multiple, erythematous scaly papules and plaques on the trunk and extremities (Fig. 1). Histopathologic findings showed parakeratosis, hypogranulosis and regular acanthosis with rete ridge elongation in the epidermis. Dilated capillaries and mild perivascular lymphohistiocytic infiltration were observed in the upper dermis (Fig. 2A). Routine laboratory examination showed no specific findings. He was treated with secukinumab 300 mg injected subcutaneously according



Fig. 1. Multiple, erythematous papules and plaques with scales on the trunk and extremities. We received the patient's consent form about publishing all photographic materials.

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Fig. 2. (A) Histopathologic findings showed parakeratosis, hypogranulosis and regular acanthosis with elongation of rete ridges in the epidermis. Dilated capillaries and mild perivascular lymphohistiocytic infiltration were observed in the upper dermis (H&E,  $\times$ 100). (B) Esophagogastroduodenoscopy revealed diffuse whitish to yellowish patches on the mucosal surface of esophagus.

to the standard schedule (weeks 0, 1, 2, 3, and 4, and every 4 weeks thereafter). After the fourth injection, dysphagia, odynophagia and abdominal discomfort developed. Esophagogastroduodenoscopy (EGD) demonstrated multiple whitish to yellowish plaques on the esophageal mucosal surface (Fig. 2B). Fungus culture and identification revealed *Candida albicans*. Under the diagnosis of esophageal candidiasis, he was treated with fluconazole 200 mg/day for seven days. Afterwards, the symptoms improved, and subsequent EGD showed resolution of candidiasis. Currently, the patient has been treated with guselkumab 100 mg for three months, and no recurrence of candidiasis has been observed.

Secukinumab is an anti-interleukin (IL) 17A monoclonal antibody used for the treatment of moderate-to-severe plaque psoriasis and psoriatic arthritis<sup>1,2</sup>. However, since IL-17A plays an essential role in immunological protection against various infections, inhibition of IL-17A can increase the susceptibility of patients to *Candida* infections<sup>2</sup>. According to the systematic review performed by Saunte et al.<sup>2</sup>, there was an increased incidence of *Candida* infections in patients treated with anti-IL-17A agents compared to placebo. Most cases involved orogenital candidiasis (26.5%), and esophageal involvement was relatively uncommon (2.4%). During the average follow-up period of 12 to 52 weeks, esophageal candidiasis occurred at least after eight weeks of injection, and there was no reported case that occurred in less than a month<sup>2</sup>.

Herein, we report an unusual case of a patient with psoriasis who developed esophageal candidiasis three weeks after treatment with secukinumab. He denied any history of candidiasis prior to the treatment. Since patients with underlying diseases such as DM, AIDS, or hematologic malignancies are more susceptible to infections<sup>3</sup>, our patient's underlying DM may have acted as a predisposing factor to such an early infection.

Thus, this case is worth reporting in three points. Firstly, it suggests that certain patients with underlying immune dysregulation can have *Candida* infections more abruptly than healthy individuals. Especially, since IL-17A plays a key role in host defense, clinicians should keep vigilance to the potential mucocutaneous infections when prescribing secukinumab<sup>4</sup>. Secondly, EGD must be used to evaluate esophageal candidiasis when the characteristic symptoms such as dysphagia, odynophagia, and abdominal discomfort occur. Lastly, not only clinicians, but also patients should be educated on the symptoms of *Candida* infections and advised to seek care when necessary<sup>3</sup>. Upon early diagnosis and appropriate antifungal treatments, most *Candida* infections are resolved, and biologic treatments can be continued without serious complications.

# **CONFLICTS OF INTEREST**

The authors have nothing to disclose.

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# Monoclonal Gammopathy-Associated Scleredema Adultorum of Buschke in a Patient with Diabetes Mellitus Successfully Treated with Intravenous Immunoglobulin and Narrow-Band Ultraviolet B Phototherapy: A Case Report

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## Dear Editor:

Scleredema is a rare connective tissue disease characterized by the deposition of collagen and mucin, mainly involving upper back and posterior neck. Skin findings include non-pitting edema, skin hardening, and movement limitation<sup>1</sup>. It is categorized based on underlying diseases, such as streptococcal infec-

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Jung Eun Kim Department of Dermatology, Soonchunhyang University Cheonan Hospital, Soonchunhyang University College of Medicine, 31 Suncheonhyang 6-gil, Dongnam-gu, Cheonan 31151, Korea Tel: +82-41-570-2270 Fax: +82-41-570-2271 E-mail: freesia0210@naver.com https://orcid.org/0000-0002-8399-8456 tion (type 1), monoclonal gammopathy (type 2), and diabetes mellitus (DM; type 3). Other diseases including primary hyperparathyroidism, ankylosing spondylitis, Sjögren syndrome, and dermatomyositis have also been described to be associated<sup>2</sup>. Treatment options including immunosuppressive agents, antibiotics, systemic glucocorticoids, and phototherapy have been tried, but no standard treatment protocol has yet been established<sup>1</sup>.

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A 53-year-old male presented with a 1-year history of progressive skin hardening on the posterior neck and upper back. He also reported decreased range of motion. Physical examination revealed non-pitting induration and erythema on the posterior neck and back (Fig. 1A, B). He had hypertension, obesity, and poorly controlled type 1 DM. There was no history of a preceding infection. A skin biopsy performed on his back showed thickened collagen bundles separated by clear spaces in the reticular dermis (Fig. 2). Laboratory tests including a com-