# Cutaneous Infection by *Fusarium solani* Associated with Arteriosclerosis Obliterans in an Immunocompetent Patient

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***Fusarium*** **species are a group of soil, saprobic and phytopathogic fungi. The pathogen generally affects immunocompromised individuals and infection of immunocompetent people is rarely reported. *Fusarium* species frequently have been found in immunocompetent people's burned tissue and skin ulcers. We describe a case of cutaneous infection by *Fusarium solani* in an immunocompetent 67-year-old patient associated with arteriosclerosis obliterans. To the best of our knowledge, this has not been previously reported in the Korean dermatological literature.**

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## INTRODUCTION

*Fusarium* infections do not play a prominent role in human mycoses because Fusarium species are weakly invasive and are considered opportunistic pathogens[1]. Thus, most infections are found in patients with neoplastic or other debilitating diseases maintained by immunosuppressive drugs, or in a severely burned or immunocompromised host[2]. The *Fusarium* infection's portals of entry include the respiratory and gastrointestinal tracts, catheter tips and indwelling central venous catheters, and the skin[1,3,4]. Infection occurs by direct contact with contaminated soil or plants, inhalation of airborne spores or ingestion of contaminated food.

Infections by *Fusarium* species in humans can result in localized, focally invasive or disseminated disease. Among immunocompetent individuals, skin lesions typically are localized and develop after skin breakdown at the site of infection. Cutaneous infections in these immunocompetent hosts present most commonly as necrotic lesions that complicate extensive wounds (burns and trauma) and foreign bodies, but to our knowledge no case of cutaneous infection by *Fusarium solani* in a patient with arteriosclerosis obliterans (ASO) exists in the Korean dermatological literature.

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## CASE REPORT

A 67-year-old man presented at our clinic with a two-month history of multiple cutaneous ulcers on his right foot. Physical examination revealed multiple areas of necrotic ulceration with eschar formation and purulent discharge on dorsal surface and the 4th toeweb of right foot (Fig. 1). At first, we did not examine the peripheral pulses because the patient denied claudication or right leg pain. We initially suspected vascular ulcer, but although we recommended visiting the vascular surgery clinic, the patient refused the consultation.

Histopathologic examination disclosed severe necrosis and ulceration of the epidermis extending to the dermis. It also revealed septate fungal hyphae

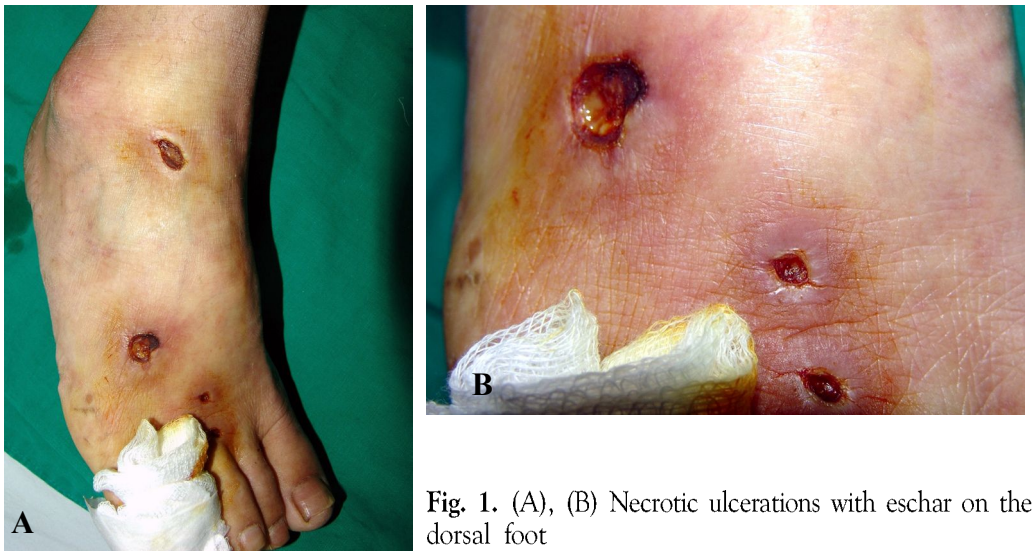


**Fig. 1.** (A), (B) Necrotic ulcerations with eschar on the right dorsal foot

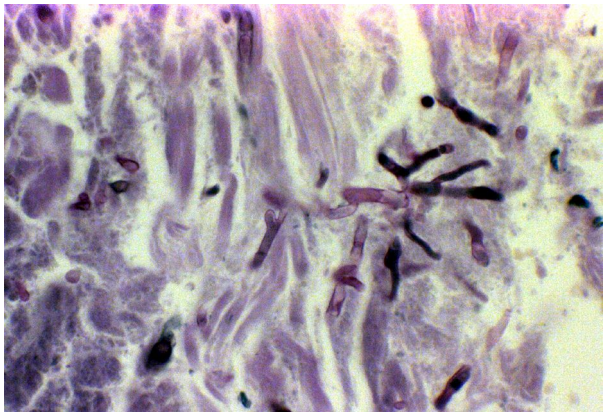

**Fig. 2.** Elongated and septate fungal hyphae and spores were found in the lesion (D-PAS, × 200)

and spores in the ulcer base (Fig. 2). We performed a fungus culture; seven consecutive fungal cultures grew pure and similar colonies on Sabouraud dextrose agar. This fine cottony aeromycelium with banana shaped multiseptate macroconidia and a pinkish red color on the back of the agar plate was consistent with the characteristic findings of a *Fusarium solani* colony (Fig. 3). There was no growth for bacteria including mycobacteria. Baseline investigations revealed elevated C-reactive protein (1.9 ㎎/㎗; normal range, 0-0.6 ㎎/㎗), whereas other routine laboratory analyses were within normal limits. Immunological studies including serum IgG, IgM, $C_3$ and $C_4$, WBC count, total number of T

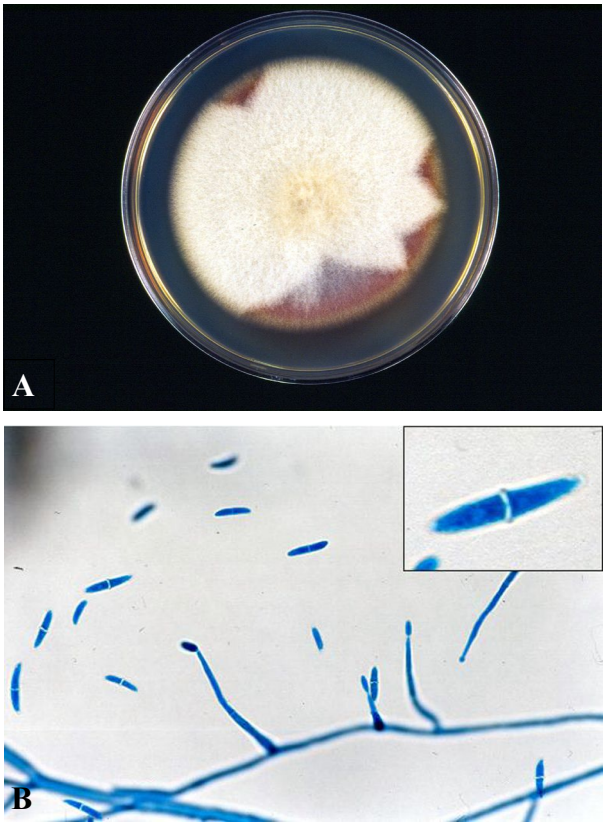


**Fig. 3.** (A) Fungus culture showing whitish cottony rapid-growing colonies on the 6th day and pinkish red color on the base (25℃, Sabouraud dextrose agar). (B) Banana shaped multiseptate macroconidia (lactophenol cotton blue stain, × 400)

and B cells, T cell subset, multiple cell mediated immunity (CMI) tests and diphenylcyclopropenone (DPCP) sensitization revealed no abnormal findings.

A diagnosis of *Fusarium solani* infection occurring in immunocompetent patient was made. The patient treated itraconazole 200 ㎎ once daily for 3 weeks, but the ulcers showed only slight improvement. The patient then failed to follow-up his treatment.

After one month, the patient revisited with aggravated ulcers and a cyanotic and edematous right foot. Physical examination revealed reduced palpable pulse on right dorsalis pedis and right popliteal arteries. These findings suggested ASO, so an arteriography was performed, which showed very poor circulation with severe obstruction of the common iliac and superficial femoral arteries (Fig. 4). A femoro-popliteal bypass with thrombolytic therapy corrected this vascular insufficiency. At that time, he had toxic hepatitis due to herbal medication, so we could not prescribe any antifungal medication. But after the surgery, ulcerative lesions improved rapidly with a simple wet dressing and topical antifungal agent.

## DISCUSSION

*Fusarium* species may cause a variety of skin infections ranging from onychomycosis in healthy hosts to widely disseminated lesions in immunocompromised patients with hematogenously disseminated multiple organic infections. A variety of skin infections accompany *Fusarium* infections including red or gray macules or papules, which progress to central ulceration or eschar formation, purpuric papules, pustules, and subcutaneous nodules[5]. The lesion most characteristic of *Fusarium* infection is a red or gray macule with a central ulceration of black eschar. Healing lesions may progress to an eschar surrounded by concentric scales. Patients with cutaneous disease related to *Fusarium* species can present with superficial and deep infections. In addition, *Fusarium* species may colonize wounds, burns, and chronic ulcers.

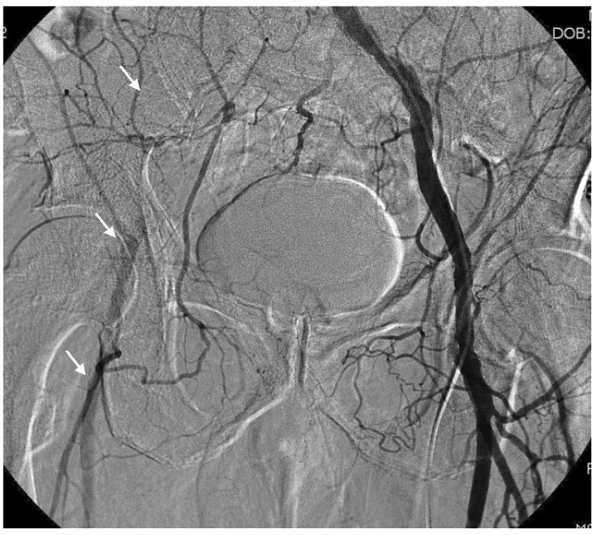


**Fig. 4.** The arteriogram demonstrates an occlusive right common iliac artery and superficial femoral artery (arrow).

As reported in a recent review of cutaneous infections by *Fusarium* species, immunocompetent patients more frequently had a history of skin breakdown than those who were immunocompromised[6]. In our patient, the predisposing cutaneous ulcer due to ASO seems to be a good condition for the growth of that fungus. Our patient had nothing predisposing him to opportunistic infection. In immunocompetent patients, *Fusarium* species should not be able to spread because they are usually well walled-off[6]. *Fusarium* species that directly invade through traumatized skin would usually spontaneously resolve, but in our patient the infection did not initially respond to the itraconazole pulse therapy. After correction of the vascular insufficiency, ulcerative lesions improved rapidly. It seems that predisposing skin breakdown as caused by trauma, severe burn, foreign body, or vascular insufficiency is the risk factor for *Fusarium* infection. Therefore, detection and correction of the underlying cause will be essential in *Fusarium* infection in immunocompetent patients.

The diagnosis of *Fusarium* infection is principally based on mycology and histopathology. Recently, a PCR technique has also been developed for specific detection of *Fusarium* species from both culture and clinical samples[7]. Cultures require incubation at 25℃ on a Sabouraud dextrose agar without cycloheximide. The most important microscopic features for species identification on culture are the conidia: the presence of fusoid macroconidia, which have foot cells with some type of heel, is accepted as the most definitive characteristic of the genus *Fusarium*[1]. Histologically, diagnostic clues include the presence of adventitious sporulation consisting of phialides

and phialoconidia and the presence of irregular hyphae with both 45- and 90-degree branching in a closed lesion[8,9].

*Fusarium* species are resistant to several chemotherapeutic agents, and treatment using these drugs has frequently failed in immunocompromised patients. But *Fusarium* infections in immunocompetent patients generally exhibit a good response to therapy[6]. Superficial *Fusarium* infections usually respond to local treatment, systemic ketoconazole and/or debridement. Localized deep infection may respond to surgical resection of infected tissue, amphotericin B alone or combined with surgical and medical treatments[6].

In summary, we presented a case of infection by *Fusarium solani* associated with ASO in an otherwise healthy man. This case may contribute to the recognition of *Fusarium* infection in immunocompetent patients. It also showed that a *Fusarium* infection could occur in a cutaneous ulcer of the ASO. Since the predisposing skin breakdown will be the risk factor for *Fusarium* infection, dermatologists must consider the possibility of underlying causes such as vascular insufficiency when *Fusarium* infection is unresponsive to conventional treatments, especially in immunocompetent patients.

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