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https://doi.org/10.5021/ad.2017.29.3.387

Fournier's Gangrene: A Rare Complication of Sweet's Syndrome

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Dear Editor:

A 31-year-old woman presented with 7 days history of pruritic multiple various sized erythematous plaques on whole body (Fig. 1A). Before the skin lesions appeared, she was prescribed some medicines at a private hospital. The patient had a fever (38.2°C) and the lab findings showed increased ESR (77, 0～20 mm/h) CRP (7.72, 0.0～0.3 mg/dl), and normal procalcitonin (0.125, 0～0.5 ng/ml). Based on her past history, clinical and lab findings, Sweet's syndrome (SS) was suspected, and she was treated with high dose steroids for 5 days (80 mg of intravenous methylprednisone every 12 hours, tapered to 20 mg). During hospitalization, edematous papillary demis and neutrophilic infiltrate with leukocytoclasis could be seen in the biopsy (Fig. 1B). Our patient showed 1) sudden onset of erythematous plaques, 2) neutrophilic dermal infiltrate (2 major criteria), 3) fever, 4) rapid response to steroid therapy (2 minor criteria)[1]. Consequently, on basis of clinical, histologic, lab findings, and criteria, we could diagnose as SS. Three days after discharge, she revisited us

Received June 9, 2016, Revised August 18, 2016, Accepted for publication August 19, 2016

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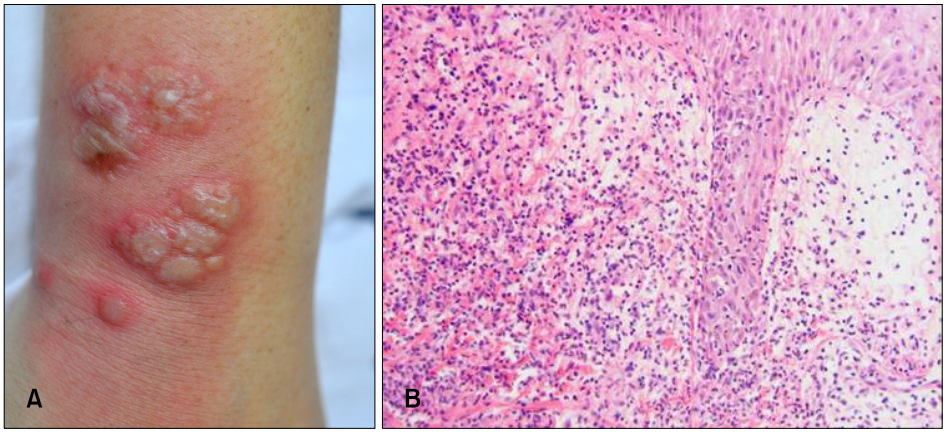

Fig. 1. (A) Clinical appearance and (B) skin biopsy from inflammatory infiltrate of neutrophil around blood vessels (H&E, ×200).

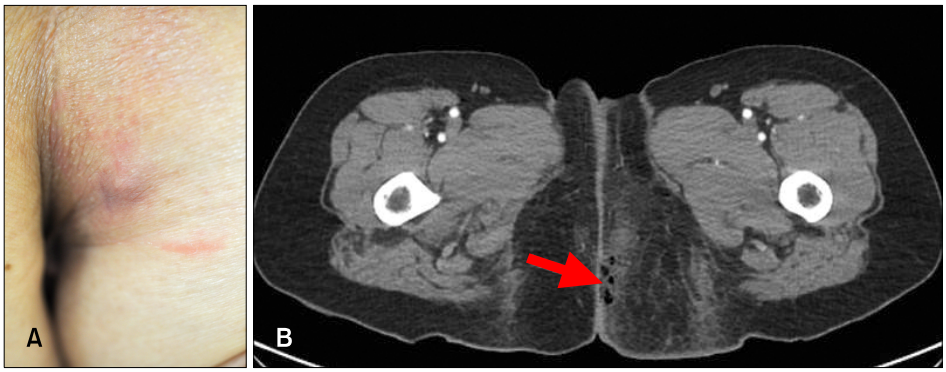

Fig. 2. (A) A 2 cm-sized erythematous plaque & swelling on buttock. (B) Computed tomography scan showed a free gas which invased to gluteus maximus muscle layer.

unexpectedly and presented painful 2 cm-sized erythematous plaque & swelling on buttock (Fig. 2A). In drainage, smell of rotten fish and greenish pus was discharged. Although X-ray findings did not seem unusual, but computed tomography (CT) scan showed a free gas which invased to gluteus maximus muscle layer and edematous change (Fig. 2B). Bacterial culture showed *Streptococcus anginosus*, *Pseudomonas*, *Clostridium* many. And more than synthesize history, culture, and CT results were diagnosed with Fournier's gangrene (FG). The patient was hospitalized for 17 days and was treated with wide debridement and broad-spectrum antibiotics. Two months later, the lesion was healed completely without severe sequelae.

SS is characterized by an abrupt onset of cutaneous lesions consisting of painful, erythematous plaques accompanied by fever, leukocytosis with neutrophilia[2]. Immune depressive agents such as systemic corticosteroid and cyclospoin are the mainstay of therapy for SS[2]. FG is a rapidly progressive necrotizing fasciitis of the perianal and perineal region that is often polymicrobial in nature, especially a host of microbes, including gram positive, gram negative and anaerobic species. Therefore, FG is an opportunistic infection most commonly affecting the immunosuppressed[3]. And, FG could be confused with erythema multiforme, erythema nodosum, leukocytoclastic vasculitis, cellulitis, and etc. The diagnosis of FG is primarily based on clinical findings, lab test, and radiography. CT can be used to detect the presence of soft tissue air in the lesion and plays an important role in the diagnosis of FG[4]. Early aggressive debridement is the cornerstone for treatment. In addition, good intensive care and microbial treatment (ampiciiln, clindamycin, metronidazole, and etc.) are key treatments[5]. We postulated that first, short course of high dose corticosteroid for SS could make her immune-deficient status. Second, SS itself can increase the incidence of opportunistic infection. To the best of our knowledge, this is the first report that FG and SS are occuring in conjunction. Dermatologists should consider the possibility of FG when a patient, who takes immunosuppressive agents for disease, presents painful nodules on perianal and perineal area and we suggest rapid evaluation and aggressive treatment for FG.

ACKNOWLEDGMENT

This study was supported by research fund from Chosun University Hospital, 2017.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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