Case Report

# *Candida parapsilosis* Cellulitis in a Patient with Systemic Lupus Erythematosus: A Case Report

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The authors described a case of *Candida parapsilosis* cellulitis in a systemic lupus erythematosus patient presented with a clinical of cellulitis unresponsive to antibiotic treatment. Yeast cells and pseudohyphae were seen in tissue biopsy and *C. parapsilosis* were identified by tissues culture. There was excellent outcome after surgical debridement and antifungal treatment.

***Keywords***: *Candida parapsilosis*, Cellulitis, Cellulitis in immunocompromised patients, Candidiasis, Fungus

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*Candida* cellulitis is an extremely rare skin and soft tissue infection most often caused by *Candida albicans*. However, a human commensal fungi, *Candida parapsilosis*, can be a pathogen especially in immunocompromised host[1]. Clinical symptoms of *Candida* cellulitis can mimic bacterial cellulitis and can progress slowly, which could lead to a delayed diagnosis. Currently, there have only been a few reported cases of primary *Candida* infection of skin and soft tissue[2-4]. Therefore, the authors reported a case of *C. parapsilosis* cellulitis in a systemic lupus erythematosus patient who presented with a clinical of cellulitis unresponsive to antibiotic treatment.

## Case Report

The medical records of the patient were retrospectively reviewed. Clinical information, including presenting symptoms, comorbidities, history of treatment were collected. For histopathology of lesional skin biopsy, the tissues were stained using hematoxylin and eosin (H&E), Gomori methenamine silver stain (GMS), and periodic acid-Schiff (PAS). Sabouraud dextrose agar was used for the isolation of fungi.

## Results

A 37 year-old-female with history of systemic lupus erythematosus (SLE) for six years, presented with six days of progressive pain, redness, and swelling of her left leg. Her past illnesses were Purtscher-like retinopathy, hemoglobin E trait, vasculitis of both legs for three years, and a history of non-tuberculous mycobacterial infection of the right leg diagnosed seven months prior to this presentation. At that time, she presented with right leg cellulitis. Acid-fast bacilli (AFB) stain of pus was positive and culture confirmed *Mycobacterium chelonae* and *Mycobacterium abscessus* infection. Her medications at the time of presentation were prednisolone five milligrams daily for SLE and 1,000 milligrams of ciprofloxacin and clarithromycin, which had been taken for four months for the treatment of NTM. She had no history of leg trauma or any injection of drug in her leg. The lesion was very painful, especially with movement. She was treated with oral clindamycin for four days with no improvement. Physical examination

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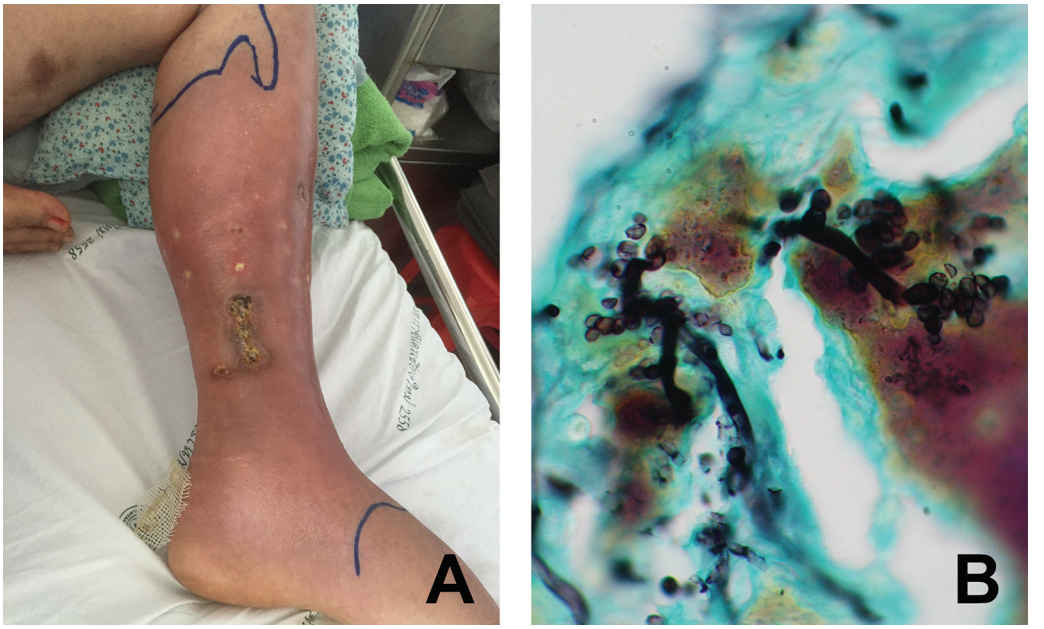


**Figure 1.** (A) Multiple ulcers on an ill-defined, erythematous, swollen plaque on patient's left leg. The blue line depicts the lesion's boundary before treatment. (B) Lesional biopsy specimen shows budding yeasts with pseudohyphae (Gomori methenamine silver stain, original magnification ×1,000).

showed body temperature of 38.3°C and an ill-defined, erythematous, swollen plaque with overlying warmth and tenderness on the left leg, measuring 30×25 cm. There were multiple ulcers with some necrotic tissue on the surface of the lesion on the medial aspect of the left leg (Figure 1A). Complete blood count revealed hemoglobin 12.9 g/dl, hematocrit 41.0%, white blood cell count of 13,200 per cu.mm with neutrophil 83.4%, and 175,000 per cu.mm. of platelet. Her fasting plasma glucose was 95 mg/dl. She was given intravenous imipenam/cislastatin and vancomycin for two weeks, but did not improved. Magnetic resonance imaging of her lower extremities demonstrated fluid signal intensity tracking in the left leg along the subcutaneous tissue, deep fascia and intermuscular planes with thickening of the deep fascia and skin, suggestive of non-necrotizing fasciitis, which in terms of radiology means soft tissue and fascial inflammation without evidence of necrosis along the left leg and foot.

Biopsy was performed of the plaque on the left leg and histopathology showed psoriasiform epidermal hyperplasia and granulation tissue in the dermis and necrotic areas in the deep dermis and subcutaneous tissue. The GMS and PAS stains were positive for pseudohyphae and yeast cells in the necrotic area (Figure 1B). As the lesion progressed, surgical debridement was performed. Multiple specimen of pus and tissues from the left leg were sent for culture and grew *C. parapsilosis*. Hemoculture was negative. After initiation of intravenous amphotericin B (1.5 milligrams/kilogram/day) for two weeks and wound care with vacuum-assisted closure dressing, the lesion markedly improved and the patient recovered without complication.

## Discussion

*Candida* cellulitis, an invasive infection of skin and soft tissue, is very rare and most often caused by *C. albicans*. However, *C. parapsilosis*, a non-*albicans Candida* species as found in the present case, has recently shown a marked increase in incidence[1]. Until now, there have been only a few reported cases of primary *Candida* infection of skin and soft tissue. All reported patients were immunocompromised, such as those with uncontrolled diabetes mellitus, those with hematologic malignancy undergoing myeloablative chemotherapy, and those receiving corticosteroid therapy[2-4]. Similar to what has been reported in the literature, the present patient was treated with long-term courses of systemic glucocorticoids.

There are many proposed mechanisms for possible pathways of *Candida* infection including direct inoculation, contiguous spread from nearby affected site, and hematogenous spreading[3,4]. These

pathways, combined with predisposing host factors, are believed to cause invasive *Candida* infection of skin and soft tissue. In the present patient, the prolonged use of antibiotics for NTM treatment may have been another risk factor. Many studies have also found that invasive *C. parapsilosis* infections were more prominent in the patients with central venous catheter insertion[1,5,6]. Colonization with *C. parapsilosis* were also believed to precede the invasive infection[1,5]. The present patient had negative blood cultures and there was no evidence of *Candida* infection at nearby sites. In general *C. parapsilosis* is a commensal of human integument system and the intact skins can protect humans from its pathogenicity. However, it is possible that the previously ulcerated lesions on her left leg acted as a site of direct inoculation of *C. parapsilosis*.

On this admission, the patient was previously prescribed some broad-spectrum antibiotics without any improvement. The clinical symptoms slowly progressed, which could have led to the delayed diagnosis of primary *Candida* infection of the skin and soft tissue. The MRI of lower extremities revealed fluid signal intensity tracking in the left leg along the subcutaneous tissue, deep fascia and intermuscular planes, and thickening of the deep fascia, which made it difficult to distinguish from necrotizing fasciitis[7]. Superficial skin and soft tissue infection such as cellulitis, if left untreated, can progress to necrotizing fasciitis. However, intraoperative findings from the present patient showed intact fascia, so necrotizing fasciitis was excluded.

Currently, there is no standardized therapeutic regimen and duration for the treatment of invasive *Candida* infection of skin and soft tissue[8]. The present patient was treated with intravenous amphotericin B and responded well. Fever subsided and the skin lesion gradually improved without complications.

## Conclusion

The authors presented a case of primary *C. parapsilosis* infection of the skin and soft tissue, a condition that is extremely rare and difficult to diagnose. It can be a clinical mimicker of necrotizing fasciitis. Although the clinical picture is less aggressive than the infection caused by bacteria, it should be considered in immunocompromised patients who present with skin and soft tissue infections unresponsive to antibiotic therapy.

## What is already known on this topic?

• Most of the causative pathogen for cellulitis is bacteria, such as *Staphylococcus aureus* and β-hemolytic streptococci.

• *Candida* cellulitis is extremely rare and most often caused by *C. albicans*.

## What this study adds?

• Cellulitis with systemic signs of infection that unresponsive to antibiotic therapy should be considered of uncommon pathogen such as fungal infection.

• Fungal cellulitis can be a clinical mimicker of necrotizing fasciitis

## Conflicts of interest

The authors declare no conflict of interest.

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