Chronic cutaneous lesions in immunocompromised patients

Wei-Chieh Lee, Wei-Sin Li, Chih-Hsiung Lee

ABSTRACT

Introduction: Chronic cutaneous lesions are common in immunocompromised patients and are attributable to a wide range of potential microbial pathogens. Common infections may have a variety of unusual manifestations, and unusual pathogens can also play an important role in these infections. Therefore, "how to approach" these lesions becomes a difficult problem.

Case Report: A 42-year-old male had medical history of immune thrombocytopenic purpura (ITP), chronic hepatitis C with liver cirrhosis, Child's-Pugh-Turcotte score A, and end stage renal disease requiring maintenance hemodialysis. The patient also suffered from multiple ecchymoses and tender plaques on all four limbs for one month. A pathology of skin biopsy showed lobular panniculitis and a strong positive finding of acid-fast bacilli (AFB). A bone marrow biopsy also showed a strong positive finding of AFB but no granulomatous inflammation. The patient was treated as disseminated tuberculosis infection and experienced anti-tuberculosis (TB) drug-related adverse effects. Finally, non-tuberculous mycobacteria (NTM) was diagnosed. There are difficulties regarding how to survey and treat immunocompromised patients due to multiple side effects and comorbidities. In recent years, NTM has been characterized as an emerging pathogen.

Conclusion: Cutaneous lesions in immunocompromised patients are complex because of a wide range of potential microbial pathogens. Common infections may have unusual manifestations in immunocompromised patients. The NTM is an emerging opportunistic pathogen in severely immunocompromised patients with acquired immunodeficiency syndrome or a transplantation in recent years. The incidence of pulmonary or extrapulmonary NTM infection has increased dramatically in recent years. We should consider NTM infection if a clinical condition does not improve after several months of anti-TB therapy.

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CASE REPORT

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adverse effects. Finally, non-tuberculous mycobacteria (NTM) was diagnosed. There are difficulties regarding how to survey and treat immunocompromised patients due to multiple side effects and comorbidities. In recent years, NTM has been characterized as an emerging pathogen. **Conclusion:** Cutaneous lesions in immunocompromised patients are complex because of a wide range of potential microbial pathogens. Common infections may have unusual manifestations in immunocompromised patients. The NTM is an emerging opportunistic pathogen in severely immunocompromised patients with acquired immunodeficiency syndrome or a transplantation in recent years. The incidence of pulmonary or extrapulmonary NTM infection has increased dramatically in recent years. We should consider NTM infection if a clinical condition does not improve after several months of anti-TB therapy.

Keywords: Chronic Cutaneous Lesion, End stage renal disease, Immune thrombocytopenic purpura, Nontuberculous mycobacteria

Wei-Chieh Lee¹, Wei-Sin Li², Chih-Hsiung Lee³

Affiliations: ¹MD, Division of Cardiology, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Taiwan; ²MD, Division of Infection, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Taiwan; ³MD, Division of General Medicine, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, Chang Gung University College of Medicine, Taiwan.

Corresponding Author: Chih-Hsiung Lee, MD 123, Ta Pei Road, Niao Sung District, Kaohsiung City, 83301, Taiwan, Republic of China; Email: leeweichieh@yahoo.com.tw

Received: 18 November 2014
Accepted: 04 December 2014
Published: 01 February 2015

How to cite this article

Lee Wei-Chieh, Li Wei-Sin, Lee Chih-Hsiung. Chronic cutaneous lesions in immunocompromised patients. Int J Case Rep Images 2015;6(2):90–94.

doi:10.5348/ijcri-201517-CR-10478

INTRODUCTION

Chronic cutaneous lesions are common in immunocompromised patients and are attributable to a wide range of potential microbial pathogens. Common infections may have a variety of unusual manifestations,

and unusual pathogens can also play an important role in these infections. Therefore, “how to approach” these lesions becomes a difficult problem.

CASE REPORT

A 42-year-old male had medical history of immune thrombocytopenic purpura (ITP), chronic hepatitis C with liver cirrhosis, Child’s-Pugh-Turcotte score A, and end stage renal disease requiring maintenance hemodialysis. The patient also suffered from multiple ecchymoses and tender plaques on all four limbs for one month. The lesions secreted bloody discharge (Figure 1A–D). Cellulitis was initially treated at our emergency department. The dermatologist suspected erythema induratum and suggested a skin biopsy. However, the procedure was postponed due to severe thrombocytopenia. Fever, chills, recent trauma, insect bites, pet contact, and travel history were all ruled-out.

A high fever with chills started three days after treatment for cellulitis with empirical antibiotics (intravenous oxacillin: one gram every four hours). Ulcerative plaques with bloody discharge on all four limbs became gradually more severe. New ecchymotic nodules with pus occurred. The patient’s vital signs were: blood pressure 166/108 mmHg, heart rate 106 beats per minute, respiratory rate 18 breaths per minute, temperature 39.3°C.

The patient’s blood workup revealed hemoglobin concentration of 7.7 g/dL, a leukocyte count of 13.3×10^3 cells/mm³ with an absolute neutrophil count of 87%, and a platelet count of 20×10^3 cells/mm³. C-reactive protein (CRP) was 69 mg/L. Chest radiography showed multiple nodules over the right upper lung and left upper lung, compared to previous radiographs (Figure 2). The patient did not have respiratory symptoms but was isolated because pulmonary tuberculosis could not be excluded. High resolution computed tomography scan showed a diffuse ground-glass picture over the central and upper lung, two cavitary lesions in the right middle lung (1.6 cm) and right lower lung (1.6 cm), and minimal pleural effusion (Figure 3A–D).

The high fever and chills persisted after antibiotics were modified several times. A skin biopsy was performed, and pathology showed lobular panniculitis and a strong positive finding of acid-fast bacilli (Figure 4). A bone marrow biopsy also showed a strong positive finding of acid-fast bacilli but no granulomatous inflammation. Both biopsies had negative gram stains and periodic acid-schiff stain. Disseminated tuberculosis infection involving skin and bone marrow was suspected. Anti-tuberculosis (TB) medication, including rifampin 360 mg, isoniazid 240 mg, and pyrazinamide 150 mg per day, was started. The high spiking fever persisted and new plaques with discharge continued to occur.

After a four-day course of anti-TB medication, hyperbilirubinemia (total bilirubin: 2.5 mg/dL and direct

bilirubin: 1.64 mg/dL) and hyperuricemia occurred. The anti-TB medication protocol was modified as follows: rifampin 600 mg, INH 300 mg and EMB 800 mg per day. Anti-TB treatment was stopped due to progressive jaundice. After 8 days of observation, hepatic function improved, and rifampin 450 mg, INH 200 mg per day and EMB 800 mg per two days was started. It was frustrating that the intermittent fever persisted and the skin lesions healed poorly after anti-TB treatment continued for two months. Unfortunately, the dose could not be increased due to hepatic function.

A culture of bone marrow and sputum showed the presence of *Mycobacterium haemophilum* approximately 82 and 98 days later. Therefore, the final diagnosis was disseminated *Mycobacterium haemophilum*, which

Figure 1: (A, B, C, D) Tender ecchymoses and purpuric plaques over the lower limbs and, elbows with bloody discharge or pus.

Figure 2: Multiple nodules over the lung (green arrows).

Figure 3: (A, B, C, D) Diffuse ground-glass picture over of the central and upper lung, two cavitary lesions in the right middle lung (1.6 cm) and right lower lung (1.6 cm) and minimal pleural effusion.

Figure 4: Lobular panniculitis and strong positive finding of acid-fast bacilli.

involved the skin, bone marrow, joints and lung. The antibiotics were changed to rifabutin 300 mg per day and clarithromycin 500 mg every twelve hours. After that, the fever subsided and treatment was continued for one year. The patient received regular outpatient department follow-up every three months and no pus or ulceration was noted.

DISCUSSION

Cutaneous lesions in immunocompromised patients are complex because of a wide range of potential microbial pathogens. Common infections may have unusual manifestations in immunocompromised patients. We could list several differential diagnosis as formulas VINDICATE for chronic cutaneous lesion with ulceration (Table 1) [1]. Figure 5 showed an algorithm to approach chronic cutaneous lesions in immunocompromised patients.

Lobular panniculitis is a group of diseases whose hallmark is inflammation of subcutaneous adipose tissue. Lobular panniculitis can be related to trauma, connective tissue disease (systemic lupus erythematosus or scleroderma), lymphoproliferative disease (lymphoma or histiocytosis), pancreatitis or pancreatic cancer, and other causes [2]. Positive acid-fast staining pathogens include all *Mycobacteria* (TB, NTM), *Actinomyces* (*Nocardia*, *Rhodococcus*, *Gordonia*, *Tsukamurella*, *Dietzia*, nuclear inclusion bodies (Lead poisoning, Bismuth poisoning), and cysts of some coccidian parasites (*Cryptosporidium parvum*, *Isospora belli*, and *Cyclospora cayetanensis*).

The skin biopsy showed positive acid-fast staining and favored cutaneous tuberculosis infection causing

Figure 5: An algorithm to approach chronic cutaneous infections in immunocompromised patients.

Table 1: Differential diagnosis as formulas VINDICATE for chronic cutaneous lesion

V	Vascular	Peripheral arteriosclerosis, venous ulcer, and varicose ulcers
I	Infections	Viruses, Bacteria, Spirochetes, Parasites, Fungus
N	Neoplasm	Sarcomas, mycosis fungoides
D	Degenerative	Peripheral neuropathy, syringomyelia, and peroneal muscular atrophy
I	Intoxication	Ulcer of chronic dermatitis
C	Congenital	Ulcers of sickle cell anemia
A	Autoimmune	Periarthritis nodosa, pyoderma gangrenosum
T	Trauma	Burns and radiation and secondary to unhealed lacerations and decubitus ulcers
E	Endocrine	Diabetic ulcers

ulceration and lobular panniculitis. Anti-TB treatment was started, but the fever and cutaneous lesions persisted even after two months of anti-TB treatment. NTM infection was considered. The cultures of bone marrow and skin showed *Mycobacterium haemophilum*. Disseminated *M. haemophilum* infection, involving the skin, lung and bone marrow, was diagnosed.

The patient often had hematomas on the lower limbs due to ITP. Our hypothesis was that hematomas were a good media for *M. haemophilum* growth. However, no obvious connection between immune thrombocytopenic purpura and *M. haemophilum* has been reported previously. Cell-mediated immunity is a significant factor in disease caused by *M. haemophilum* [3, 4].

M. haemophilum is sensitive to amikacin, clarithromycin, ciprofloxacin, rifampin and rifabutin, but it is usually resistant to other first-line antimycobacterial medications [3, 5]. Currently, according to guidelines from the 2007 American Thoracic Society/Infectious Diseases Society of America, patients with irreversible immunosuppression probably require maintenance therapy for months to years. Duration of therapy should be guided by the patient's underlying condition and clinical response. Severe disease (disseminated disease, osteomyelitis, and pulmonary disease) that responds well to treatment should be treated for a minimum of several months (e.g., 6–12 months) [6, 7].

In our case, we switched medication to rifabutin 300 mg once per day and clarithromycin 500 mg twice per day after confirmation of *M. haemophilum* infection. The fever subsided rapidly and no new nodules were noted on the lower extremities. After one week of therapy, the patient was discharged but continued follow-ups and medical therapy at our outpatient department. Treatment continued for one year. The skin lesions healed and no pus or ulcerations were noted after one year. The patient's hepatic function remained within the normal range [8].

CONCLUSION

A number of learning points are illustrated by this case. Firstly, careful examination of the skin may provide the earliest clues to the accurate diagnosis of systemic infections. Generalized skin lesions are often diagnosed as cellulitis. We cannot forget that cutaneous lesions in immunocompromised patients are complex because of a wide range of potential microbial pathogens. Common infections may have unusual manifestations in immunocompromised patients. Skin biopsy is usually necessary to confirm the diagnosis. Secondly, we also need to realize that nontuberculous mycobacteria (NTM) is an emerging opportunistic pathogen in severely immunocompromised patients with acquired immunodeficiency syndrome or a transplantation in recent years. The incidence of pulmonary or extrapulmonary NTM infection has increased dramatically in recent years. We should consider NTM

infection if a clinical condition does not improve after several months of anti-TB therapy.

Author Contributions

Wei-Chieh Lee – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Wei-Sin Li – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Chih-Hsiung Lee – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor

The corresponding author is the guarantor of submission.

Conflict of Interest

Authors declare no conflict of interest.

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ABOUT THE AUTHORS

Article citation: Lee Wei-Chieh, Li Wei-Sin, Lee Chih-Hsiung. Chronic cutaneous lesions in immunocompromised patients. Int J Case Rep Images 2015;6(2):90–94.

Wei-Chieh Lee is Attending Doctor at division of Cardiology, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, Kaohsiung, Taiwan. He has published three research papers in national and international academic journals. His research interests include general cardiology, interventional cardiology and electrophysiology study. He intends to pursue PhD's in future.

Wei-Sin Li is A Resident at Division of Infection, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, Kaohsiung, Taiwan. He has published only one research papers in reputed academic journals. His research interests include infection, microbiology and epidemiological.

Chih-Hsiung Lee is Chief at Division of General Medicine, Department of Internal Medicine, Kaohsiung Chang Gung Memorial Hospital, Kaohsiung, Taiwan. He has published several research papers in national and international academic journals. His major research interests include general nephrology, peritoneal dialysis and hemodialysis.

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