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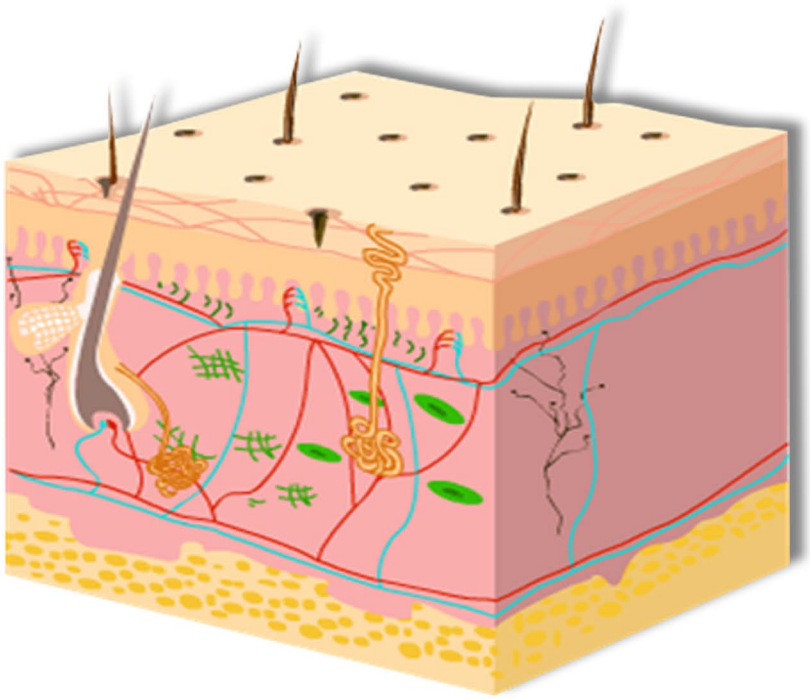

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Short Communication

*Corresponding author
Khalid Al Hawsawi, MD
Dermatology Consultant
King Abdul Aziz Hospital
House#4148, Al-Takassosi District
Branch#6134, Unit#1
Makkah 24323, Saudi Arabia
Tel. 00966-555756499
Fax: 00966-25424449
E-mail: hawsawik2002@hotmail.com

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Papulonecrotic Tuberculid: A Rare Case Report

Khalid Al Hawsawi, MD[1*]; Dania Amassi, MD[2]; Dalal Alesa, MD[3]; Faisal Alraddadi, MD[2]; Ghassan Niaz, MD[3]; Waseem Alhawsawi, MD[4]

[1]Dermatology Consultant, King Abdul Aziz Hospital, Makkah, Saudi Arabia
[2]Dermatology Resident, King Abdul Aziz Hospital, Makkah, Saudi Arabia
[3]Medical Intern, Umm Alqura University, Makkah, Saudi Arabia
[4]Medical Student, King Saud bin Abdulaziz University for Health Sciences, Jeddah, Saudi Arabia

ABSTRACT

Tuberculids were originally felt to be related to an allergic response to tubercle bacilli in a patient with tuberculosis at a remote site. They are currently believed to be the result of hematogenous dissemination of organisms from an internal focus to the skin, where they incite a cutaneous inflammatory response. Papulonecrotic tuberculid (PNT) is a form of tuberculids that as the name implies presents clinically as necrotic papules. Herein, we report a case of 59-year-old man who presented with recurrent asymptomatic symmetrical necrotizing papules scattered on his trunk for 9 months. The patient has also crusted plaque on his right forearm. Two skin biopsies were made, one from papulonecrotic lesion on his trunk and the other one from the crusted plaque on his right forearm. The crusted plaque on the forearm showed granulomatous cellular infiltrates and caseation necrosis in the dermis, whereas the papulonecrotic lesions showed patchy perivascular mononuclear cellular infiltrates as well as granulomatous cellular infiltrates in the dermis. Tuberculin test was positive. A diagnosis of lupus vulgaris on the forearm and PNT on the trunk were made based on clinicopathological findings. The patient was seen by chest physician where there was no systemic involvement. Patient was treated successfully with anti-tuberculosis drugs for 9 months with complete resolution of all skin lesions.

KEY WORDS: Papulonecrotic tuberculid; Anti-tuberculosis; Lymphadenopathy.

INTRODUCTION

Tuberculids were originally felt to be related to an allergic response to tubercle bacilli in a patient with TB at a remote site. They are currently believed to be the result of hematogenous dissemination of organisms from an internal focus to the skin, where they incite a cutaneous inflammatory response.[1] Tuberculids are uncommon manifestation even in a high prevalence TB areas. Once diagnosis of a tuberculid has been made, a thorough evaluation for active tuberculosis should be initiated. Mycobacterium tuberculosis culture from tuberculid is of low yield. Papulonecrotic tuberculid (PNT) is a form of tuberculids that as the name implies presents clinically as necrotic papules. Tuberculid was first described by Darrier in 1896. It represents an Arthus reaction (type III hypersensitivity reaction) accompanied by delayed-type hypersensitivity reaction (type IV).[2-5] Papulonecrotic tuberculid presents clinically as a chronic recurrent asymptomatic symmetrical necrotizing skin papules arising in crops and heal with atrophic varioliform scarring. It's primarily involving the extensor surfaces of extremities, trunk, and buttocks.[2-5] Treatment of PNT is like the treatment of tuberculosis by antituberculous treatment.

CASE REPORT

A 59-year-old man presented with 9 months history of recurrent asymptomatic skin lesions. The lesions last for 1-2 months then disappear spontaneously without treatment but recur again.

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Figure 1: (A) Forearm of the Patient showing Crusted Erythematous Plaque. (B) Trunk of the Patient Showing Multiple Discrete Non Scaly Erythmatous Papules with Necrotic Centers.

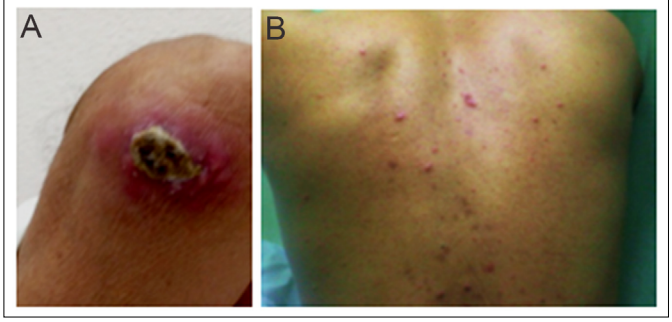
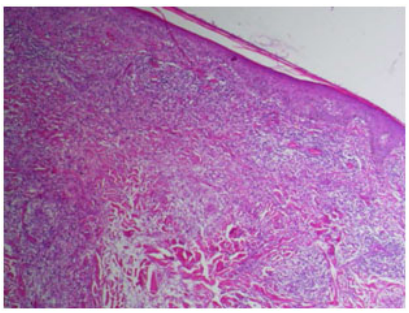


Figure 2: Histopathological Features of the Crusted Plaque on the Forearm of the Patient Showing Granulomatous Infiltration with Caseation Necrosis in the Dermis.



The patient has past medical history of cervical lymphadenopathy 6 years ago where it was excised surgically with unknown diagnosis. He did not receive any treatment at that time. No history of similar condition in his family. Reviews of systems were unremarkable. Skin examinations revealed two different types of skin lesions. The first one was crusted plaque measuring 5×5 cm on his right forearm. The second one was multiple discrete non scaly erythmatous papules with necrotic centers scattered on his trunk (Figure 1). There was no lymphadenopathy. Two skin biopsies were made, one from crusted plaque on his right forearm and the other one from the papulonecrotic lesions on his trunk. The crusted plaque on his forearm showed granulomatous cellular infiltrates and caseation necrosis in the dermis with positive acid-fast bacilli stain (Figure 2), whereas the papulonecrotic lesions showed patchy perivascular mononuclear cellular infiltrates as well as granulomatous cellular infiltrates in the dermis. Tuberculin test was positive. Sputum sample for TB staining and culture were negative. Chest X-Ray was normal. A diagnosis of lupus vulgaris on the arm and PNT on the trunk were made based on clinicopathological findings. The patient was seen by chest physician. There was no systemic involvement. Patient was treated successfully with anti-tuberculosis drugs for 9 months with complete resolution of all skin lesions.

DISCUSSION

Cutaneous tuberculosis (TB) is either "true" cutaneous TB (lupus vulgaris , TB verrucosa cutis , scrofuloderma , orificial TB , military TB) or tuberculids (Papulonecrotic tuberculid, nodular vasculitis, lichen scrofulosorum, and erythema nodusum).[1-6]

In cutaneous TB, the extracutaneous focus is found in only 30-40% of cases, with cervical lymph nodes being the most common site, as in our patient.[7]

Some authors proposed diagnostic criteria for PNT as the following: A strongly positive Mantoux test; chronic recurrent papular eruptions occurring in crops with necrosis, ulceration, and scarring; a tuberculoid histology with endarteritis and thrombosis of the dermal vessels; and regression in response to antituberculous treatment. Our patient fulfilled all diagnostic criteria of PNT.

Although PNT is a very rare, its association with lupus vulgaris is rarer.

Polymerase chain reaction (PCR) is a very sensitive tool to demonstrate organisms and the first instance of PNT yielding mycobacterium tuberculosis DNA was reported by Victor et al.[8]

CONSENT

Consent has been taken from the patient for purpose of using patient's photographs for publication in print or on the internet.

CONFLICTS OF INTEREST

The authors have no conflicts of interest that are directly relevant to the content of this review.

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REFERENCES

1. Paller AS, Mancini AJ. Bacterial, mycobacterial, and protozoal infections of the skin. *Hurwitz Clinical Pediatric Dermatology: Textbook of Skin Disorders of Childhood and Adolescence*. 5th ed. New York, USA: Elsevier; 2016: 334-359.

2. Jordaan HF, Van Niekerk DJ, Louw M. Papulonecrotic tuberculid, a clinical, histopathological, and immunohistochemical study of 15 patients. *Am J Dermaopathology*. 1994; 16(5): 474-485. Web site. http://journals.lww.com/amjdermatopathology/abstract/1994/10000/papulonecrotic_tuberculid__a_clinical,.2.aspx. Accessed January 6, 2017.

3. Gupta V. Papulonecrotic tuberculid with scrofuloderma: An uncommon association. *J Clin Diagn Res*. 2015; 9(2): WD03-

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WD04. doi: 10.7860/JCDR/2015/10751.5524

4. Oon HH, Chong WS, Oh CC, et al. Simultaneous occurrence of papulonecrotic tuberculid and erythema induratum in an Asian woman. *Skinmed*. 2016; 14(6): 457-459. doi: 10.1111/j.1525-1470.2012.01744.x

5. Wong S, Rizvi H, Cerio R, O'Toole EA. An unusual case of vulval papulonecrotic tuberculid. *Clin Exp Dermatol*. 2011; 36(3): 277-280. doi: 10.1111/j.1365-2230.2010.03925.x

6. Bae SH, Yun SJ, Lee JB, Kim SJ, Lee SC, Won YH. Papulonecrotic tuberculid: A rare skin manifestation in a child with mesenteric tuberculous lymphadenopathy. *Acta Derm Venereol*. 2017; 96(7): 137-138. doi: 10.2340/00015555-2486

7. Morrison JGL, Fourie ED. The papulonecrotic tuberculid. from arthus reaction to lupus vulgaris. *Br J Dermatol*. 1974; 91(3): 263-270. doi: 10.1111/j.1365-2133.1974.tb12894.x

8. Victor T, Jordaan HF, Van Niekerk DJT, Louw M. Papulonecrotic tuberculid. Identification of mycobacterium tuberculosis DNA by polymerase chain reaction. *Am J Dermatopathol*. 1992; 14(6): 491-495. Web site. http://journals.lww.com/amjdermatopathology/Abstract/1992/12000/Papulonecrotic_Tuberculid__Identification_of.1.aspx. Accessed January 6, 2017.

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Case Report

*Corresponding author
Khalid Al Hawsawi, MD
Dermatology Consultant
King Abdul Aziz Hospital
House#4148, Al-Takassosi District
Branch#6134, Unit#1
Makkah 24323, Saudi Arabia
Tel. 00966-555756499
Fax: 00966-25424449
E-mail: hawsawik2002@hotmail.com

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Erythema Annulare Centrifugum (Deep Type): A Rare Case Report

Khalid Al Hawsawi, MD[1*]; Hanadi Alzanbagi, MD[2]; Sara Almatrafi, MD[3]; Samaher Refae, MD[2]; Fatmah Al-Shahrani, MD[4]; Hatem Alsulimani, MD[3]; Randa Almatrafi, MD[5]

[1]Dermatology Consultant, Head of Dermatology Department, King Abdul Aziz Hospital, Makkah, Saudi Arabia
[2]Dermatology Resident, King Abdul Aziz Hospital, Makkah, Saudi Arabia
[3]Medical Intern, Umm Al-Qura University, King Abdul Aziz Hospital, Makkah, Saudi Arabia
[4]Medical Intern, King Khalid University, Abha, Saudi Arabia; King Abdul Aziz Hospital, Makkah, Saudi Arabia
[5]General Practitioner, Alnoor Hospital, Makkah, Saudi Arabia

ABSTRACT

Erythema annulare centrifugum (EAC) is one of the figurate erythemas. It is uncommon inflammatory condition characterized by annular or arcuate erythematous eruptions that slowly enlarge centrifugally. It persists from few days to several months. It can recurrent. Here in we present a 40-year-old-male otherwise healthy, is working as a nurse in leprosy hospital. He was concerned from having leprosy. He developed recurrent asymptomatic skin lesions on his face for the last 5 months. The lesions start as small pimples that are getting bigger every day. The lesions last for few weeks and then disappear but recur again after weeks to months. Sensation examination of the facial skin was normal. On palpation of periauricular nerves, there was nerve hypertrophy. His older brother had similar skin lesions 10 years ago that lasted for few months and then healed without treatment. Skin examination revealed multiple non-scaly annular erythematous plaques, with variable sizes ranging from 2 cm to 4 cm on his face. Skin biopsy showed normal epidermis. The dermis showed moderately dense perivascular and periadnexal mononuclear cellular infiltrate in coat sleeve pattern in both upper and lower dermis. Fite stain was negative. The patient was reassured.

KEYWORDS: Erythema annulare centrifugum; Figurate erythema; Erythema gyratum perstans.

ABBREVIATIONS: EAC: Erythema Annulare Centrifugum; GA: Granuloma Annulare.

INTRODUCTION

Erythema annulare centrifugum (EAC) is uncommon inflammatory skin disorder characterized by figurate erythematous eruptions that slowly enlarge centrifugally.[1-3] EAC is self-limited disease with variable course that lasts as little as few weeks to as long as three decades. The exact cause of EAC is not known. However, various agents have been implicated including hypersensitivity reaction to drugs (penicillin, salicylates, hydrochlorothiazide), arthropod bites, infections (bacterial, mycobacterial, viral, fungal, filarial), food allergy (blue cheese Penicillium), malignancy as (lymphoma, multiple myeloma, breast cancer), autoimmune and endocrine disease (Hashimoto Thyroiditis , Sjogren syndrome).[4-8]

EAC is classified into two types. The superficial type is characterized clinically by presence of fine collarette of scales on the trailing edge of the annular plaques which histopathologically show pronounced epidermal changes as well as perivascular cellular infiltrate in superficial dermis. The deep type is characterized clinically by non-scaly annular plaques with infiltrated borders which histopathologically show perivascular cellular infiltrate in both

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superficial and deep dermis with minimal epidermal changes.[3-8] A "Coat sleeve" pattern of the perivascular cellular infiltrate in the dermis is the classical histopathological feature of EAC.

A "Coat sleeve" pattern is a tight (sharply demarcated) mononuclear cellular infiltrate around blood vessels of the dermis. EAC occurs at any age but more commonly in fifth decade of life. Male to female ratio are equal. It can present in any part of the body but more commonly on trunk, the thigh, the legs and buttocks.[8]

CASE REPORT

A 40-year-old-male who is working as a nurse in leprosy hospital for the last 20 years, presented with 6-month-history of asymptomatic recurrent migratory skin lesions on his face. He was concerned from having leprosy. Over the last 6 months, he started to develop skin lesions that start as small pimples and then expand slowly forming large rings which then disappear gradually without treatment but reappear again in another site on his face. The lesions are not associated with loss of sensation or numbness. Review of systems and past medical history were unremarkable. Family history revealed that his younger brother developed similar condition 10 years ago that last for few months and then disappeared spontaneously without any recurrence until now. Skin examination revealed multiple annular non-scaly erythematous indurated plaques of variable sizes ranging from 2 cm to 5 cm on his face (Figure 1). Sensation examination of the facial skin was normal. On palpation of periauricular nerves, there were nerve hypertrophy. Skin biopsy taken from the edge of the lesion showed dense very tight perivascular mononuclear cellular infiltrate both in upper and lower dermis with a "coat sleeve" pattern (Figure 2). Stain for acid fast bacilli were negative. On the basis of the above clinicopathological findings, the diagnosis of deep type of erythema annulare centrifugum was made. The patient was reassured.

DISCUSSION

EAC is uncommon inflammatory condition characterized by annular or arcuate erythematous eruptions that slowly enlarge centrifugally.[3-8] The main differential diagnosis in our patient include leprosy especially that our patient has history of contact with leprosy patients. Other differential diagnosis include granuloma annulare (GA), annular elastolytic giant cell granuloma, and secondary syphilis.[3-8] However, the histopathology of the skin lesions was classical for EAC. The migratory feature and the spontaneous resolution of the skin lesions are not features of leprosy. GA is not migratory in nature. Familial EAC, as in our patient is rare. However, it has been reported before as "familial annular erythema".[5]

EAC resolves either spontaneously or once the underlying disease has been successfully treated. Topical medications like corticosteroids, tacrolimus, calcipotriene, oral metronidazole, subcutaneous etanercept and subcutaneous interferon-α have been all used with some benefit.[8] These have not been tried in our patient because the patient refused the treatment. He just wants to be reassured that he is not having leprosy.

Figure 1: Multiple Non-scaly Annular Erythematus Plaques with Infiltrated Border on Peri-auricular Area.

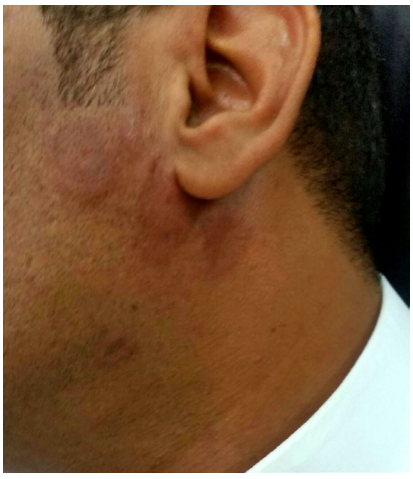
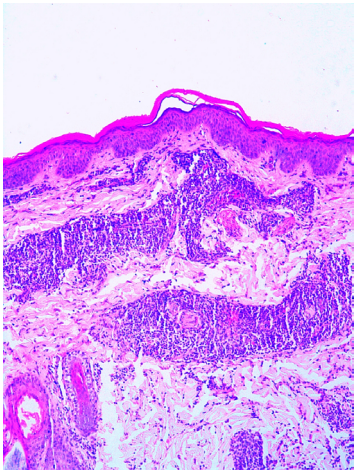


Figure 2: Histopathological Features of the Skin Lesions Showing Dense Very Tight Perivascular Mononuclear Cellular Infiltrate Both in Upper and Lower Dermis with A "Coat Sleeve" Pattern.



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CONFLICTS OF INTEREST

The authors have no conflicts of interest that are directly relevant to the content of this review.

CONSENT STATEMENT

Consent has been taken from the patient for purpose of using patient's photographs for publication in print or on the internet.

REFERENCES

1. Kavurt S, Aydemir O, Celik U, et al. Erythema annulare centrifugum as the presenting sign of pseudomonas aeruginosa sepsis in a newborn. *Eur J Pediatr.* 2013; 172(6): 847-849. doi: 10.1007/s00431-012-1848-8

2. Kim DH, Lee JH, Lee JY, Park YM. Erythema annulare centrifugum: Analysis of associated diseases and clinical outcomes according to histopathologic classification. *Ann Dermatol*. 2016; 28(2): 257-259. doi: 10.5021/ad.2016.28.2.257

3. Weyers W, Diaz-Cascajo C, Weyers I. Erythema annulare centrifugum: Results of a clinicopathologic study of 73 patients. *Am Dermatol*. 2003; 25(6): 451-462. Web site. http://journals.lww.com/amjdermatopathology/pages/articleviewer.aspx?year=2003&issue=12000&article=00001&type=abstract. Accessed February 4, 2017.

4. Ohmori S, Sugita K, Ikenouchi-Sugita A, et al. Erythema annulare centrifugum associated with herpes zoster. *J UOEH*. 2012; 34(3): 225-229. doi: 10.7888/juoeh.34.225

5. Bressler GS, Jones RE Jr. Erythema annulare centrifugum. *J Am Acad Dermatol*. 1981; 4(5): 597-602. doi: 10.1016/S0190-9622(81)70063-X

6. Ziemer M, Eisendle K, Zelger B. New concepts on erythema annulare centrifugum: A clinical reaction pattern that does not represent a specific clinicopathological entity. *Br J Dermatol.* 2009; 160(1): 119-126. doi: 10.1111/j.1365-2133.2008.08803.x

7. Watsky KL, Hansen T. Annular erythema in identical twins. *Cutis*. 1989; 44(2): 139-140. Web site. http://europepmc.org/abstract/med/2758863. Accessed February 4, 2017.

8. Mshrai H, Fallatah B, Alwafi D, Babkoor D, Al Sufyani H, Al Hawsawi K. Erythema annulare centrifugum (EAC): A case report of annually recurring EAC. *J Health Sci*. 2016; 6(5): 74-76. doi: 10.5923/j.health.20160605.02

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Case Report

*Corresponding author
Khalid Al Hawsawi, MD
Dermatology Consultant
King Abdul Aziz Hospital
House#4148, Al-Takassosi District
Branch#6134, Unit#1
Makkah 24323, Saudi Arabia
Tel. 00966-555756499
Fax: 00966-25424449
E-mail: hawsawik2002@hotmail.com

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Tinea Incognito: Case Report

Khalid Al Hawsawi, MD[1*]; Sumayah Alshehri, MD[2]; Nouf Al Muawad, MD[3]; Rwan Gaafar[4]; Khloud Alsadi[4]; Maather Alhajaji[4]; Samar Alwafi, MD[5]

[1]Dermatology Consultant, Head of Dermatology Department, King Abdul Aziz Hospital, Makkah, Saudi Arabia
[2]Dermatology Resident, Alnoor Specialist Hospital, Makkah, Saudi Arabia
[3]Medical Intern, Umm Alqura University, College of Medicine, Makkah, Saudi Arabia
[4]Medical Student, Umm Alqura University, College of Medicine, Makkah, Saudi Arabia
[5]Dermatology Resident, King Abdul Aziz Hospital, Makkah, Saudi Arabia

ABSTRACT

Tinea incognito (TI) is defined as absence of the classic annular configuration of tinea infection. It is caused by misuse of topical or systemic corticosteroids and less frequently by calcineurin inhibitors. Herein we present a 15-year-old boy presented with 8 months history of persistent mildly itchy skin lesions on his face. Patient used many topical treatments including steroid, but no improvement. Skin examination showed multiple well defined scaly patches and plaques on his face. Potassium hydroxide (KOH) microscopic examination and fungal culture revealed dermatophytes fungi. Itraconazole 200 mg capsules once daily for 2 weeks was prescribed. The skin lesions disappeared completely.

KEYWORDS: Tinea incognito (TI); Tinea atypica; Dermatophytoses.

INTRODUCTION

Tinea incognito (TI) is the term given to a dermatophyte infection with atypical appearance due to improper use of steroids or calcineurin inhibitors.[1,2] It was first coined by *Ive and Marks*.[3,4] The typical dermatophyte infection presents as annular lesions with erythematous scaly border and central clearing. In TI, this later feature is not seen. Rosacea-like, psoriasiform and erythroderma-like presentation of TI have been described in the literature.[5,6] The diagnosis is confirmed by isolation of dermatophytes by microscopic examination with potassium hydroxide (KOH) and fungal cultures. Systemic antifungal therapy is preferred over the topical antifungals.[5,7]

CASE REPORT

A 15-year-old boy presented with 8 months history of persistent itchy skin lesion on his face. Patient used many topical treatments including steroid, but no improvement. Past medical history and systemic review were all unremarkable. Family history revealed history of atopic dermatitis in one of his siblings. Skin examination showed multiple well defined scaly patches and plaques on the right side of his face (Figure 1). Differential diagnosis included psoriasis, atopic eczema, contact dermatitis, and subacute lupus erythematosus. KOH microscopic examination and fungal culture revealed dermatophytes fungi. On the base of the above clinical and laboratory findings, a diagnosis of TI was made. Itraconazole 200 mg capsules once daily for 2 weeks was prescribed. The skin lesions disappeared completely (Figure 2).

DISCUSSION

Topical application of steroids may modify the presentation of the dermatophyte infection. TI on the face may mimic lupus erythematosus, rosacea, and contact dermatitis.[5-8] The pathogenesis of TI is mostly due to a steroid-modified response of the host immunity to fungal infec-

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Figure 1: Multiple Well Defined Scaly Erythematous Patches on the Right Side of the Face.

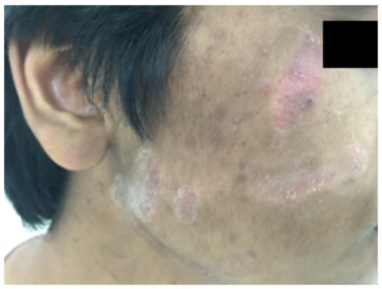
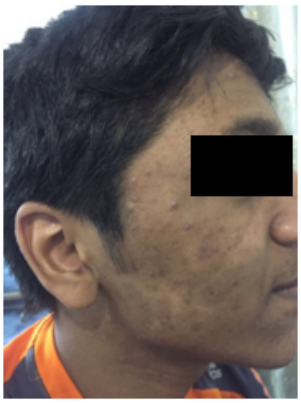


Figure 2: Face of the Patient Showing Post-inflammatory Hyperpigmentation after using Oral Itraconazole Capsules for 2 Weeks. Note the Acne Lesions that are Unrelated to this Case Report.



tion and not to a direct pharmacological effect on the fungus.[5-9] Both Potent fluorinated and non-fluorinated topical steroids may produce TI.[2-5] Arise of TI infection in recent years is partly due to an increasing number of patients who self-treat themselves with topical steroids that are obtained over the counter. More recently, a few cases of TI due to use of topical tacrolimus and pimecrolimus have been reported.[2-5] *Trichophyton rubrum* is one of the most common anthropophilic dermatophyte throughout the world and the most frequently isolated dermatophyte in TI.[3-6] Although localized dermatophyte infections respond well to topical antifungals agents, TI should be treated with oral antifungals. Terbinafin as well as the azoles like itraconazole and fluconazole are preferred over griseofulvin in treating TI.[3-7]

CONCLUSION

TI is a rare skin disease that presents as atypical dermatophytosis. The typical dermatophyte infection presents as annular lesions with active scaly borders and central clearing. It is a diagnostic challenge for dermatologist because it may mimic a variety of different dermatosis. A high index of suspension is required for dermatosis that are unresponsive to topical immunosuppressants. TI should be confirmed by KOH microscopic examination and fungal culture to isolate dermatophytes. It is better to be treated by treated oral antifungals.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest that are directly relevant to the content of this review.

CONSENT STATEMENT

Consent has been taken from the patient for purpose of using patient's photographs for publication in print or on the internet.

REFERENCES

1. Romano C, Maritati E, Gianni C. Tinea incognito in Italy: A 15-year survey. *Mycoses*. 2006; 49: 383-387. doi: 10.1111/j.1439-0507.2006.01251.x

2. Del Boz J, Crespo V, Rivas-Ruiz F, de Troya M. Tinea incognito in children: 54 cases. *Mycoses*. 2011; 54: 254-258. doi: 10.1111/j.1439-0507.2009.01810.x

3. Rallis E, Koumantaki-Mathioudaki E. Pimecrolimus induced tinea incognito masquerading as intertriginous psoriasis. *Mycoses*. 2008; 51: 71-73. doi: 10.1111/j.1439-0507.2007.01436.x

4. Rajpar SF, Abdullah A. Management of onychomycosis and awareness of guidelines among dermatologists. *Br J Dermatol*. 2006; 155: 1080-1082. doi: 10.1111/j.1365-2133.2006.07493.x

5. Al Aboud K, Al Hawsawi K, Alfadley A. Tinea incognito on the hand causing a facial dermatophytid reaction. *Acta Derm Venereol*. 2003; 83(1): 59.

6. Crawford KM, Bostrom P, Russ B, Boyd J. Pimecrolimus-induced tinea incognito. *Skinmed*. 2004; 3: 352-353. doi: 10.1111/j.1540-9740.2004.03796.x

7. Siddaiah N, Erickson Q, Miller G, Elston DM. Tacrolimus-induced tinea incognito. *Cutis*. 2004; 73(4): 237-238. Web site. http://europepmc.org/abstract/med/15134322. Accessed February 26, 2017.

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8. Pustisek N, Skerlev M, Basta-Juzbasić A, Lipozencić J, Marinović B, Bukvić-Mokos Z. Tinea incognito caused by trichophyton mentagrophytes -- A case report. *Acta Dermato-venerol*. 2001; 9: 283.

9. Gupta AK, Prussick R, Sibbald RG, Knowles SR. Terbinafine in the treatment of majocchi's granuloma. *Int J Dermatol.* 1995; 34: 489. doi: 10.1111/j.1365-4362.1995.tb00619.x

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Review

Corresponding author
Badriya Al-Lenjawi, PhD
Senior Assistant
Executive Director of Nursing
Hamad Medical Corporation
P.O.Box. 3050
20 Sahar Bin Ayash Street
Old Airport Area, Doha, Qatar
Tel. 00974-55559584
E-mail: blenjawi@hamad.qa

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Is It Time to Reconsider the 60 Seconds-Diabetic Foot Screen Reorganizing the 60 Second Foot Exam for People with Diabetes?

Badriya Al-Lenjawi, PhD[1*]; Hashim Mohamed, MD[2]; Azzam Azmy, MD[3]

[1]Senior Assistant Director of Nursing, Hamad Medical Corporation, Doha, Qatar
[2]Associate Professor, Weill Cornell Medical College, Doha, Qatar; Senior Consultant Family Medicine, Doha, Qatar
[3]General Practitioner, Doha, Qatar

ABSTRACT

Diabetic patients have 25% lifetime risk of developing foot ulceration. More than half of these ulcers may eventually become infected, which greatly increases the likelihood of subsequent amputations. Although a multidisciplinary approach is the standard management for treating diabetic foot ulcers (DFUs), screening of diabetic foot ulcers is an integral part of that process. This review highlights the importance of the Inow's 60 seconds screening tool but at the same time highlights serious gaps in the screening tool which is supposed to be a comprehensive screening tool for use in diabetic patients. The authors have addressed these gaps in a constructive and scientific way thereby solidifying the screening tool further in order to capture all the features and complications of the diabetic foot.

KEY WORDS: Diabetic foot; Screening tool; Diabetes.

ABBREVIATIONS: DFUs: Diabetic Foot Ulcers; IWGDF: International Working Group on Diabetic Foot; ABI: Ankle-Brachial Index.

INTRODUCTION

We read with interest the Inlow's 60 second foot exam for people with diabetes.[1] Comprehensive and easy to apply wound screening tools are a vital pre-requisite in formulating a management plan to achieve optimal wound healing and patient well-being. The authors of the 60 second foot exam for people with diabetes made a considerable effort in devising a simple and practical screening tool for health care professionals worldwide. Since 2004, the 60 seconds-diabetic foot screen remained unscrutinized and therefore remained unchanged. Although comprehensive history is a vital element of risk assessment, clinicians cannot fully assess patients with diabetes for risk factors for foot ulceration based on history alone; a comprehensive foot exam remains the essential element of this process. Essential elements of the history include previous foot ulceration and or amputation, Charcot foot, angioplasty, cigarette smoking (number of packets per year), electrical sensation, rest pain, claudication, history of microvascular and macrovascular complications neuropathic or peripheral vascular symptoms,[2,3] retinopathy, or chronic renal impairment renal replacement therapy.

A comprehensive examination of the feet and footwear in a well- lit clinic should routinely be done after the patient has taken off his shoes and socks. Although improper footwear is a common contributory element in the development of foot ulceration[4,5] most health care professionals do not pay attention to them, the footwear must be examined at every visit and

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also the patient usually comes to the clinic with a different footwear to the one(s) he or she is used to. So the health care professional must ask about home worn foot wear as well as outdoor foot wear. Both the health care professional and the patient must ask the question "*Are these footwear adequate for these feet?*", since improper footwear will result in friction, erythematic, blister, corn and callus formation and ultimately ulceration. The discipline of wound care and wound assessment are continuously evolving. However, few attempts have been carried out to revalidate screening tools[6-9] and advanced wound products wound products.[10,11]

Dermatological Problems in People with Diabetes

Skin disorders occur in 79.2% of people with diabetes.[12] A recent study of 750 patients with diabetes showed that the most prevalent skin conditions were coetaneous infections (47.5%), xerosis (26.4%), and inflammatory skin diseases (20.7%).[12] The majority of patients will eventually develop skin complications due to the long-term consequences of diabetes on the microcirculation and on skin collagen. Autoimmune-related cutaneous lesions are more prevalent in patients with type 1 diabetes whereas cutaneous infections are more common in type 2 diabetes.[13]

Skin manifestations of diabetes can be divided into those related to insulin resistance, type 1 diabetes and type 2 diabetes.

Skin conditions related to insulin resistance include, acanthosis nigricans, acrochordrons, diabetic dermopathy, eruptive xanthoma, rubeosis facei and epidermal necrolysis/Stevens-Johnson syndrome. Some of the most common cutaneous manifestations in patients with type 1 DM include, periungual telangiectasia, necrobiosis lipoidica, bullosis diabeticorum, vitiligo and lichen ruberplanus.

On the other hand patients with type 2 diabetes mellitus (T2DM) complain of the following problems yellow nails or onychomycosis, diabetic thick skin presenting as asymptomatic thick skin involving the fingers and hands ranging from pebbling over the knuckles to diabetic hand syndrome. The skin of the neck and back may also be involved leading to diabetic scleredema, with "peaud'orange" appearance and reduced sensitivity to touch and pain in the affected areas.[13] Other skin conditions linked to T2DM include diabetic dermopathy, acquired perforating dermatosis. Cutaneous infections associated with diabetes include, candidiasis, dermatophytosis, and bacterial infections.

Diabetic Foot (Epidemiology and Etiopathogenesis)

It is well established that a number of contributory elements working in a synergistic fashion eventually result in the final pathway to foot ulceration among patients with diabetes. The commonest element is peripheral neuropathy, external trauma, foot deformity, peripheral vascular disease, peripheral oedema[14] and improper foot care practices. This makes it vital to have a comprehensive annual screening of patients with diabetes especially the foot and to screen them more often if they have high risk for foot ulcerations. Patients at high risk of foot complications including those peripheral neuropathy, long standing diabetes, smokers, improper foot wear, previous history of ulceration and amputations.

Screening Tools for Diabetic Foot

High-risk foot identification is a vital component of comprehensive diabetes care. Furthermore, risk classification allows timely and precise follow-up for different levels of risk.[15] According to the International Working Group on Diabetic Foot (IWGDF) patients with a low risk should be screened in a year or sooner if a foot problem arises whereas patients who have loss of protective sensation can be seen more frequently i.e. every three to six months.[4] Those who have previous ulceration, and or amputation and or evidence of peripheral vascular disease must be seen every month. Many practical tools to screen for diabetic foot problems exist including those for peripheral neuropathy such as the 10 gram monofilament, neurothesiometer, the 128 KHZ tuning fork, etc. Peripheral vascular disease can be assessed by palpation of the dorsalis pedis pulse and tibialis posterior and the use of hand held doppler and measuring the ankle-brachial index (ABI).

Several elements are essential to ensure a valid and optimal screening test including simplicity, quick to conduct, have high inter and intra-observer reliability, validity, and generalizability. In their assessment of the 60 seconds Inlow's diabetic foot screen Murphy et al[16] concluded that the tool demonstrates excellent interrater and intrarater reliability. However, these results have to be considered with caution since the numbers which were tested were only 69 and the sample was a convenient sample, therefore bias cannot be ruled out.

However, there are few shortcomings that need to be examined when utilizing the Inlow's screening tool in daily practice and these include: under the skin section the authors included only.

Skin
0=intact and healthy
1=dry with fungus or light callus
2=heavy callus build up
3=open ulceration or history of previous ulcer

Firstly, the combination of (dry, fungus and light callus) cannot be justified (placed together) since they are three separate conditions with different etiologies which can co-exist together or be on their own, so the numbering has to be modified. Additionally, patient with long standing diabetes may suffer from autonomic neuropathy leading to dysfunctional sweat glands thereby leading to cracked/fissured skin which is also had been omitted from the screening tool.

Secondly, there is no mention of the following condi-

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Inlow's 60-second Diabetic Foot Screen[36]

Screening Tool			www.cawc.net
Patient Name:		Clinician Signature:	
ID number:		Date:	
1. Skin 0=intact and healthy 1=dry with fungus or light callus 2=heavy callus build up 3=open ulceration or history of previous ulcer			
2. Nails 0=well-kept 1=unkempt and ragged 2=thick, damaged, or infected			
3. Deformity 0=no deformity 2=mild deformity 4=major deformity			
4. Footwear 0=appropriate 1=inappropriate 2=causing trauma			
5. Temperature – Cold 0=foot warm 1=foot is cold			
6. Temperature – Hot 0=foot is warm 1=foot is hot			
7. Range of Motion 0=full range to hallux 1=hallux limitus 2=hallux rigidus 3=hallux amputation			
Assess – 30 seconds	**Left Foot**	**Right Foot**	**Care Recommendations**
8. Sensation – Monofilament Testing 0=10 sites detected 2=7 to 9 sites detected 4=0 to 6 sites detected			
9. Sensation – Ask 4 Questions: i. Are your feet ever numb? ii. Do they ever tingle? iii. Do they ever burn? iv. Do they ever feel like insects are crawling on them? 0 = no to all questions 2=yes to any of the questions			
10. Pedal Pulses 0=present 1=absent			
11. Dependent Rubor 0=no 1=yes			
12. Erythema 0=no 1=yes			
Score Totals=			

Screening for foot ulcers and/or limb-threatening complications. Use the highest score from left or right foot.
Score=0 to 6→recommend screening yearly Score = 7 to 12→recommend screening every 6 months
Score=13 to 19→recommend screening every 3 months Score = 20 to 25→recommend screening every 1 to 3 months

Comments:

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tions which may accompany diabetic foot common conditions:

Maceration

Around 55% of wounds under investigation are reported to have macerations according to one clinical study[18] and in diabetic ulcers maceration represents a challenge for the treating health care provider.[19] Maceration is a frequent phenomenon in heavily exuding ulcers of all types, and in order to avoid damage to the periwound area frequent dressing is required.[20,21]

Corns

Many patient with diabetes have improper footwear and according to a study done by Gayle et al,[22] 39% of patients with diabetes who were attending a specialist diabetic clinic were reported wearing pointed toe shoes. Regular debridement in patients with diabetes, may reduce the incidence of subcutaneous bleeding, subsequent ulceration thereby avoiding the need for surgery.[23] Therefore, the inclusion of corns is needed in the screening tool.

Dermatitis

Topical treatments and other sensitizers are a common cause of allergic dermatitis among patients with type II diabetes. A retrospective study conducted in Jordan in 2002 by Najdawi and Fa'ouri.[24] Of 232 elderly patients with diabetes reported eczema/dermatitis as the commonest skin disorder seen (25.9% of cases). Another study in Turkey showed the prevalence of dermatitis to be 15.2%.[25]

Fragility of the Skin

This is a common finding among elderly patients and worsened by autonomic neuropathy thereby making the patient susceptible to skin integrity breakdown and subsequent microbial invasion.

Shiny Skin and Loosing Hair

The risk of peripheral artery disease (PAD) is markedly increased among individuals with diabetes[26] and accurate estimation of the prevalence of PAD in patients with diabetes is difficult as the condition may be often asymptomatic, pain perception may be altered by co existing peripheral neuropathy and worst still, the presence of intermittent claudicating and absence of peripheral pulses, are non-sensitive diagnostic indicators.[27] However studies utilizing the ankle-brachial index (ABI) showed the prevalence of PAD in patients with diabetes to be between 20% to 30%.[28-30] In patients with peripheral arterial disease the skin may be smooth, cool and shiny with hair loss, and nails tend to be dystrophic or thickened.[31]

Uncommon Conditions

Although some of the following conditions are rare, they may co-exist in patients with diabetes. These include but not limited to; verruca plantaris, psoriasis, hemosiderin deposition, naevi, moles, malignant melanoma, greenish discoloration due to pseudomonas infection on top of tinea pedis.

Diabetes Specific Conditions

Healthcare practitioners must also be aware of other diabetes mellitus-specific conditions, including, granuloma annulare, necrobiosis lipoidica diabeticorum which occurs in 0.3-1.6% of patients with diabetes,[32] granuloma annulare, diabetic dermopathy affecting 7% to 70% of diabetics,[33] waxy skin syndrome, and bullosis diabeticorum affecting 0.5% of those aged between 40-77 years old suffering from long standing diabetes and neuropathy.[34]

Sensation

Sensation – Ask 4 Questions:
i. Are your feet ever numb?
ii. Do they ever tingle?
iii. Do they ever burn?
iv. Do they ever feel like insects are crawling on them? 0=no to all questions
2=yes to any of the questions

The sensation items have omitted very essential elements including unsteadiness while walking and aching pain or tenderness in legs. These are essential components of the neuropathy disability score (NDS). This is of great clinical significance since diabetic peripheral neuropathy (DPN) is one of the most common microvascular complications in patients with diabetes.[35] Furthermore, DPN is the most common element in the cascade to diabetic foot ulceration. The maximum score of DNS is four points, one point or more indicates neurological abnormalities and as such omitting two components will make the scoring system unreliable.

The screening tool has failed to include (presence/loss of hair) in their assessment since loss of hair on the toes represent a significant marker of reduced perfusion to the periphery along with dystrophic toenails, dry and fissured skin.

This review article may serve to encourage further scientific enquiry into wound assessment tools and more importantly advanced wound care products, where prescription and utilization has been largely influenced by drug companies for many decades with very few rigorous scientific data to support most of those products out there in the market.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

1. Sibbald RG, Ostrow B, Lowe J, et al. Screening for the high-risk diabetic foot: A 60-second tool. *Adv Skin Wound Care*. 2012; 5(2): 72-82. doi: 10.1097/01.ASW.0000421460.21773.7b

2. Sage RA, Webster JK, Fisher SG. Outpatient care and mor-

ISSN 2473-4799 http://dx.doi.org/10.17140/DRMTOJ-2-121

bidity reduction in diabetic foot ulcers associated with chronic pressure callus. *J Am Podiatr Med Assoc*. 2001; 91(6): 275-279. doi: 10.7547/87507315-91-6-275

3. Najdawi F, Fa'ouri M. Frequency and types of skin disorders and associated diabetes mellitus in elderly Jordanians. *East Mediterr Health J*. 2002; 8(4-5): 574-578. Web site. http://apps.who.int/iris/handle/10665/119202. Accessed January 24, 2017.

4. Ewan A. Masson EA. Dermatological care of the diabetic foot. *Am J Clin Dermatol*. 2002; 3(7): 463-474. doi: 10.2165/00128071-200203070-00003

5. European Wound Management Association (EWMA). *Position document. wound bed preparation in practice*. London: MEP Ltd; 2004.

6. Woodbury MG, Sibbald RG, Ostrow B, Persaud R, Lowe JM. Tool for rapid & easy identification of high risk diabetic foot: Validation & clinical pilot of the simplified 60 second diabetic foot screening tool. *PLoS One*. 2015; 10(6): e0125578. doi: 10.1371/journal.pone.0125578

7. Carreau L, Niezgoda H, LeBlond S, Trainor A, Orsted H, Woodbury MG. A prospective, descriptive study to assess the reliability and usability of a rapid foot screen for patients with diabetes mellitus in a complex continuing care setting. *Ostomy Wound Manage*. 2013; 59(1): 28-34. Web site. http://www.o-wm.com/article/prospective-descriptive-study-assess-reliability-and-usability-rapid-foot-screen-patients-di. Accessed January 24, 2017.

8. Murphy CA, Laforet K, Da Rosa P, Tabamo F, Woodbury MG. Reliability and predictive validity of inlow's 60-second diabetic foot screen tool. *Adv Skin Wound Care*. 2012; 25(6): 261-266. doi: 10.1097/01.ASW.0000415343.45178.91

9. Murphy-Chutorian B, Han G, Cohen SR. Dermatologic manifestations of diabetes mellitus: A review. *Endocrinol Metab Clin North Am*. 2013; 42(4): 869-898. doi: 10.1016/j.ecl.2013.07.004

10. Al-Lenjawi B, Mohamed H, Abu Salma M, Abo Gouda Z. Natural honey in the management of thermal burn of the foot in a type 2 diabetic patient: A case report. *Dermatol Open J*. 2016; 1(1): 14-18. doi: 10.17140/DRMTOJ- 1-105.26

11. Al-Lenjawi B, Mohamed H, Al-Ali A, Kherallah B. Are all wound products created equally? The re-emergence of natural honey. *J Diabetic Foot Complications*. 2015; 7(2): 26-41. Web site. http://jdfc.org/spotlight/are-all-wound-products-created-equally-the-re-emergence-of-natural-honey/. Accessed January 24, 2017.

12. Demirseren DD, Emre S, Akoglu G, et al. Relationship between skin diseases and extracutaneous complications of diabetes mellitus: Clinical analysis of 750 patients. *Am J Clin Dermatol*. 2014; 15(1): 65-70. doi: 10.1007/s40257-013-0048-2

13. Van Hattem SI. "Skin manifestations of diabetes." *Cleve Clin J Med*. 2008; 75(11): 772-774.

14. Boulton AJM. The diabetic foot: From art to science. *Diabetologia*. 2004; 47(8): 1343-1353. doi: 10.1007/s00125-004-1463-y

15. Boulton AJ, Armstrong DG, Albert SF, et al. Comprehensive foot examination and risk assessment: A report of the task force of the foot care interest group of the American Diabetes Association, with endorsement by the American Association of Clinical Endocrinologists. *Diabetes Care*. 2008; 31(8): 1679-1685. doi: 10.2337/dc08-9021

16. Murphy CA, Laforet K, Da Rosa P, Tabamo F, Woodbury MG. Reliability and predictive validity of Inlow's 60-Second diabetic foot screen tool. *Adv Skin Wound Care*. 2012; 25(6): 261-266. doi: 10.1097/01.ASW.0000415343.45178.91

17. Bower VM, Hobbs M. Validation of the basic foot screening checklist: A population screening tool for identifying foot ulcer risk in people with diabetes mellitus. *J Am Podiatr Med Assoc*. 2009; 99(4): 339-347. doi: 10.7547/0980339

18. Jergensen B, Price P, Andersen KE, et al. The silver-releasing foam dressing, Contreet Foam, promotes faster healing of critically colonised venous leg ulcers: A randomised, controlled trial. *Int Wound J*. 2005; 2(1): 64-73. doi: 10.1111/j.1742-4801.2005.00084.x

19. Rodgers A, Watret L. Maceration and its effect on periwound margins. *Diabetic Foot*. 2003; 6(3 Suppl): S2-S5.

20. Hilton JR, Williams DT, Beuker B, Miller DR, Harding KG. Wound dressings in diabetic foot disease. *Clin Infect Dis*. 2004; 39(Suppl 2): S100-S103. doi: 10.1086/383270

21. Gayle KAT, Tulloch Reid MK, Younger NO, et al. Foot care and footwear practices among patients attending a specialist diabetes clinic in Jamaica. *Clin Pract*. 2012; 2(4): e85. doi: 10.4081/cp.2012.e85

22. Sasmaz S, Buyukbese M, Cetinkaya A, Celik M, Arican O. The prevalence of skin disorders in type-2 diabetic patients. *Internet J Dermatology*. 2004; 3(1): 1-4. Web site. https://ispub.com/IJD/3/1/8588. Accessed January 24, 2017.

23. Marso SP, Hiatt WR. Peripheral arterial disease in patients with diabetes. *J Am Coll Cardiol*. 2006; 47(5): 921-929. doi: 10.1016/j.jacc.2005.09.065

24. American Diabetes Association. Peripheral arterial disease in people with diabetes. *Diabetes Care*. 2003; 26(12): 3333-3341. doi: 10.2337/diacare.26.12.3333

25. Elhadd TA, Robb R, Jung RT, Stonebridge PA, Belch JJF. Pilot study of prevalence of asymptomatic peripheral arterial

ISSN 2473-4799 http://dx.doi.org/10.17140/DRMTOJ-2-121

occlusive disease in patients with diabetes attending a hospital clinic. *Practical Diabetes Int*. 1999; 16: 163-166. doi: 10.1002/pdi.1960160605

26. Hirsch AT, Criqui MH, Treat-Jacobson D, et al. Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA*. 2001; 286(11): 1317-1324. doi: 10.1001/jama.286.11.1317

27. Beks PJ, Mackaay AJ, de Neeling JN, de Vries H, Bouter LM, Heine RJ. Peripheral arterial disease in relation to glycemic level in an elderly Caucasian population: The Hoorn study. *Diabetologia*. 1995; 38(1): 86-96. doi: 10.1007/BF02369357

28. Gardner AW, Afaq A. Management of lower extremity peripheral arterial disease. *J Cardiopulm Rehabil Prev*. 2008; 28(6): 349-357. doi: 10.1097/HCR.0b013e31818c3b96

29. Paron NG, Lambert PW. Cutaneous manifestations of diabetes mellitus. *Prim Care*. 2000; 27: 371-383.

30. Sibbald RG, Landolt SJ, Toth D. Skin and diabetes. *Endocrinol Metab Clin North Am*. 1996; 25: 463-472.

31. Lipsky BA, Baker PD, Ahroni JH. Diabetic bullae: 12 cases of a purportedly rare cutaneous disorder. *Int J Dermatol*. 2000; 39(3): 196-200. doi: 10.1046/j.1365-4362.2000.00947.x

32. Soliman E, Gellido C. Diabetic Neuropathy. *Medicine*. 2002.

33. Reiber GE, Vileikyte L, Boyko EJ, et al. Causal pathways for incident lower-extremity ulcers in patients with diabetes from two settings. *Diabetes Care*. 1999; 22(1): 157-162. doi: 10.2337/diacare.22.1.157

34. Meijer JW, van Sonderen E, Blaauwwiekel EE, et al. Diabetic neuropathy examination: A hierarchical scoring system to diagnose distal polyneuropathy in diabetes. *Diabetes Care*. 2000; 23(6): 750-753. doi: 10.2337/diacare.23.6.750

35. American Diabetes Association. Preventive foot care in people with diabetes (Position Statement). *Diabetes Care*. 2003; 26 (Suppl 1): S78-S79. Web site. http://journals.sagepub.com/doi/abs/10.1177/107110070002100115?journalCode=faib. Accessed January 24, 2017.

36. Inlow S. A 60 second foot exam for people with diabetes. *Wound Care Canada*. 2004 ;2(2): 10-11.

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Case Series

*Corresponding author
Rosa Giménez-García, MD
Clinical Assistant
Department of Dermatology
Hospital Universitario Rio Hortega;
Associate Professor
Faculty of Medicine
Calle Carabela 115, Boecillo
Valladolid 47151, Spain
E-mail: rosagim@hotmail.com

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Gouty Tophi: Two Case Report

Rosa Giménez-García, MD*; Gonzalo Cabezón-Villalba, MD; Laura Perez-Gimenez, MD; María Jesús Gimenez-Mazuelas, MD

Department of Dermatology, Hospital Universitario Río Hortega, Valladolid, Spain

INTRODUCTION

Gouty tophi represent a symptom of chronic form of gout resulting from accumulation of monosodium urate crystals in tissues, which is the most prevalent form of inflammatory arthritis.The tophus represents a granulomatous inflammatory response to monosodium urate crystals.[1,2] Women are less likely to have gout than men but they develop it in the postmenopausal years and have comorbidities such renal disease, diabetes or concomitant use of diuretics more common as compared with men.[3] We present two cases of gouty tophi.

CASE REPORTS

Case 1

A 66-year-old man presented with an intense joint pain and deformities on his toes (Figure 1). Furthermore, the patient had an additional lump in his left elbow. He had personal history of hypercholesterolemia and hypertriglyceridemia. He had elevated levels of uric acid a year ago but in treatment with allopurinol the levels had descended to normality; uric acid 3.33 mg/dL (normal range 3.5-7.2), Histological study was consistent with gouty tophi. He was referred to rheumatologist.

Case 2

A 84-year-old man with multiple co-morbidities such as dyslipidemia, hypertension and hyperuricemia, presented to us with multiple soft tissue masses over several metacarpals associated with severe joint deformities (Figure 2). He had not been treated regularly for gout. Laboratory tests included urea 78.8 mg/dL (normal range 17.1-49.2), uric acid levels of 9.98 mg/dL (normal range 3.5-7.2), creatinine 1.63 mg/dL (normal range 0.8-1.3). We establish diagnosis of gouty tophi and referred to his physician for appropriate treatment. The patient started therapy with allopurinoll 100 mg daily with resolution of symptoms.

Figure 1: Goutytophionthe Right Toes.

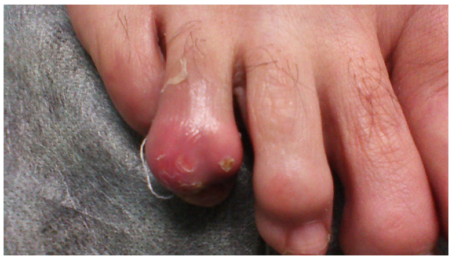
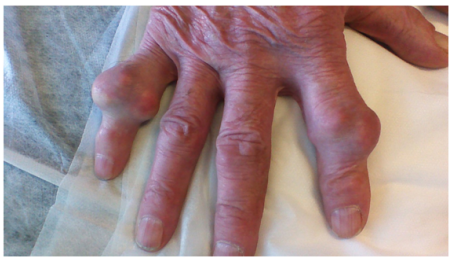


Figure 2: Tophion the Right Hand Associated with Severe Joint Deformities



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DISCUSSION

The prevalence of gout and hyperuricemia is on the rise in developing countries probably related to population aging, alcohol intake, hypertension, obesity, metabolic syndrome and use of diuretics. The prevalence increases with age. Being male and black person are also risk factors. Gout is caused by the deposits of monosodium urate crystals (MSU) in the synovial fluid and other tissues and it is associated with hyperuricemia. Crystal deposition then triggers immune activation. Tophi are subcutaneous nodules comprised of aggregates of crystals in and around joints or soft tissues. Commonly affected sites are the first metatarsophalangeal joint (MTPJ), midtarsal joints, ankles, knees, fingers and ankles. It usually appears in chronic hyperuricemia but occasionally the patient may develop them without previous gouty arthritis episodes. Superficial tophi can lead to ulcerations of the overlying skin. Histopathological features include deposit of urate crystals surrounded by an intense inflammatory reaction of macrophages, lymphocytes and large foreign body giant cells. The birefringence of the crystals is a specific sign of urate crystals. Suboptimal management of gout has been shown.[1-4]

The diagnosis of an acute gout attack in the elderly can be a challenge. Management of gout must include a definitive diagnosis (clinical, and laboratory features, presence of tophi, ultrasound examination, and demonstration of MSU crystals in synovial fluid or in the tophus); a swift treatment of acute attacks, use of urate-lowering therapies for prevention and lifestyle advice (optimizing weight, restriction intake of purines-rich food and limiting alcohol consumption).[5,6]

Treatment of acute attacks includes non-steroidal anti-inflammatory drugs, low-dose colchicine regimen and oral, intramuscular or intraarticular corticosteroids. Allopurinol is the first-line medication for reducing serum uric acid. Probenecid, colchicine, other xanthine oxidase inhibitors as febuxostatmay also be used as urate-lowering therapies (ULT). The 2012 American guidelines support ULT initiation during an acute attack of gout. ULT should be started at a low-dose, and the dose gradually increased. Despite the low levels of uric acid of the analysis of the patient is possible the presence of tophi and arthritis. A patient starting ULT are at risk of gout arthritis due to the deposit of acid uric crystals in joints. To avoid this arthritis is recommended a concomitant treatment based on colchicine or COX-2 inhibitors or low-dose prednisolone.[6-8]

CONCLUSIONS

The prevalence of gout increases with the population aging and it is associated with comorbidities. If no hyperuricemia treatment is given the disease may develop into chronic tophaceous gout involving soft tissues or joints. It is important for clinicians be able to diagnose and improve the quality of gout management.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

CONSENT

The authors have taken oral consent from the patients.

REFERENCES

1. Thissen CA, Frank J, Lucker GP. Tophi as first clinical sign of gout. *Int J Dermatol*. 2008; 47(Suppl 1): 49-51. doi: 10.1111/j.1365-4632.2008.03961.x

2. Chhana A, Dalbeth N. The gouty tophus: A review. *Curr Rheumatol Rep*. 2015; 17: 19. doi: 10.1007/s11926-014-0492-x

3. Harrold LR, Etzel CJ, Gibofsky A, et al. Sex differences in gout characteristics: Tailoring care for women and men. *BMC Musculoskelet Disord*. 2017. 14; 18: 108. doi: 10.1186/s12891-017-1465-9

4. Cottrell E, Crabtree V, Edwards J, Roddy E. Improvement in the management of gout is vital and overdue: An audit from a UK primary care medical practice. *BMC Family Practice*. 2013; 14: 170-180. doi: 10.1186/1471-2296-14-170

5. Schlee S, Bollheimer LC, Bertsch T, Sieber CC, Härle P. Crystal arthritides - gout and calcium pyrophosphate arthritis: Part 2: Clinical features, diagnosis and differential diagnostics. *Z Gerontol Geriatr*. 2017; doi: 10.1007/s00391-017-1198-2

6. Ting K, Graf SW, Whittle SL. Update on the diagnosis and management of gout. *Med J Aust*. 2015; 203: 86-88. doi: 10.5694/mja14.00953

7. Hainer B, Matheson E, Wilkes T. Diagnosis, treatment and prevention of gout. *Am Fam Physician*. 2014; 90(12): 831-836. Web site. http://www.aafp.org/afp/2014/1215/p831.html. Accessed March 22, 2017

8. Abhishek A, Roddy E, Doherty M. Gout - A guide for the general and acute physicians. *Clin Med (Lond)*. 2017; 17: 54-59. doi: 10.7861/clinmedicine.17-1-54