Hidradenitis suppurativa: a disease with male predominance in Tunisia

A. Mebazaa, R. Ben Hadid, R. Cheikh Rouhou, S. Trojjet, D. El Euch, M. Mokni, M. Zitouna, and A. Ben Osman

- A bstract

Background: hidradenitis suppurativa (HS) is a chronic inflammatory, suppurating, fistulizing, and scar-producing disease of apocrine gland-bearing skin. The diagnosis is primarily clinical, based on the presence of both sinus tracts and abscesses with a characteristic distribution.

Objective: Review of epidemiological, clinical, and prognostic characteristics of HS and discussion of the etiopathogenic aspects of this chronic problem.

Patients and methods: We retrospectively report all cases of HS followed at the Department of Dermatology between January 1985 and December 2008.

Results: Eleven patients (10 male and 1 female), with a mean age of 35.2 years (range 21-53 years) at HS diagnosis were followed for HS. The average age of disease onset was 23.9 years. The median delay between onset of symptoms and diagnosis was 144 months (range 1-408 months). Clinical features showed inflamed discharging papules or nodules, painful tender erythematous nodules, and double-ended comedones. The disease mainly affected the axillary, anal, perineal, and genital areas. Histologically, dermal features showed active folliculitis or abscess, sinus tract formation, fibrosis, and granuloma formation. Pathological associations (Darier's disease and Down syndrome) were noted in two patients. Treatment consisted of antibiotics in eight patients, retinoids (1 mg/kg/ WORDS day) in three patients, and surgery in three patients. The mean follow-up was 13 months (range 2-30 months). Recurrence of lesions was observed in all patients approximately 1 month after hidradenitis treatment withdrawal. The Down syndrome patient developed vaginal hydrocele of the testis as a complication of his staphylococcic ulcers. In all cases healing occurred with substantial scarring.

> Discussion: An obvious male predominance was noted in our patients as well as a delay in the diagnosis of HS, which could be explained by non-recognition of the disease by non-specialists that see the patients at the primary-care level.



suppurativa, Verneuil. acne inversa

Introduction

Hidradenitis suppurativa (HS), also known as Verneuil's disease or acne inversa, is a chronically relapsing inflammatory skin disease characterized by recurrent draining sinuses and abscesses, predominantly in skin folds with terminal hairs and apocrine glands. It was first described by Velpeau in 1839 then by Verneuil (2) in 1854, who associated it with the sweat glands. HS was then classified as a member of the follicular occlusion triad, along with acne conglobata and dissecting cellulitis of the scalp (3). In 1975, pilonidal sinus was added to this triad, forming the follicular occlusion tetrad (4). In 1989, Plewig and Steger (4) introduced the term acne inversa based on the follicular origin of the disease.

HS is usually diagnosed clinically. It has a chronic course and may be extremely painful and severely debilitating. This article reviews the epidemiological, clinical, and prognostic characteristics of HS.

Patients and methods

We retrospectively collected all cases of HS clinically diagnosed at the department of dermatology of La Rabta hospital, Tunis, between January 1985 and December 2008.

Relevant data included age, sex, delay between onset of symptoms and diagnosis, extent of disease, frequency of exacerbations, histological exam, and response to different treatment modalities.

Results

Eleven patients (10 males and 1 female) were referred for HS. The average patient's age was 35.2 years (range 21-53 years) and the average age of disease onset was 23.9 years. At the time of the survey, patients had suffered average disease duration of 144 months (range 1-408 months). No family history of HS was noted in our patients. All patients had multifocal involvement. Clinical features showed inflamed discharging papules or nodules, painful tender erythematous nodules, and double-ended comedones (Figs. 1-3). The disease affected mainly the axillary, anal, perineal, and genital areas (Table 1). All patients were assessed according to Hurley's clinical classification and eight of them were evaluated according to the Sartorius severity score (Table 2). In six patients, histological findings showed active folliculitis or abscess, sinus tract, fibrosis, and granuloma formation. Bacterial samples realized in seven cases revealed methi-S Staphylococcus aureus in all cases. Pathological



Fig. 1. Discharging nodules and folliculitis of the axillary fold.



Fig. 2. Purulent draining sinuses and discharging nodules.



Fig. 3. Bridle hypertrophic scars.

Table 1. Clinical and prognostic characteristics of HS patients.

Patient data	Description
Case 1 Age: 41 Sex: M Onset age: 25 Duration: 16 yr Follow-up: 18 mo	Personal history: Smoking, acne conglobata, Darier's disease Aspect of lesions: Cysts, comedones, discharging sinuses Topography: Face, back, axillary and perineal areas Treatment: Acitretin 1 mg/kg/d Evolution: Improvement +++, relapses at retinoid withdrawal
Case 2 Age: 29 Sex: M Onset age: 21 Duration: 8 yr Follow-up: 3 mo	Personal history: Smoking Aspect of lesions: Nodules, boils, abscesses, ulcers, discharging sinuses, fibrosis, pustules, comedones Topography: Axillary, inguinal, perianal areas Treatment: Doxyclines 200 mg/day, antiseptics, surgery Evolution: Improvement +, frequent relapses, lost to follow-up
Case 3 Age: 28 Sex: M Onset age: 20 Duration: 8 yr Follow-up: 4 mo	Personal history: Smoking Aspect of lesions: Abscesses, crypts, pustules, comedones Topography: Perianal, axillary areas Treatment: Oxacillin, antiseptics Evolution: Improvement +, lost to follow-up
Case 4 Age: 21 Sex: M Onset age: 18 Duration: 3 yr Follow-up: 30 mo	Personal history: Obesity, Down syndrome, hydrocele testis Aspect of lesions: Abscesses, sinus tract fistulae, nodules, comedones Topography: Axillary folds, groin, perianal, gluteal crease, perineum Treatment: Doxycyclines 200 mg/day, antiseptics Evolution: Improvement +++, relapses at antibiotic withdrawal
Case 5 Age: 43 Sex: M Onset age: 28 Duration: 15 yr Follow-up: 6 mo	Personal history: Diabetes type 2 Aspect of lesions: Sinus tract fistulae, ulcerations Topography: Inguino-scrotal, perineum, gluteal areas Treatment: Doxycyclines 200 mg/day, antiseptics Evolution: Improvement ++, relapses at antibiotic withdrawal
Case 6 Age: 53 Sex: F Onset age: 19 Duration: 34 yr Follow-up: 24 mo	Personal history: Acne vulgaris Aspect of lesions: Fistulizing lesions, folliculitis Topography: Axillary, inguinal, perianal areas Treatment: Isotretinoin 1 mg/kg/day, topical erythromycin Evolution: Improvement ++, relapses at retinoid withdrawal
Case 7 Age: 27 Sex: M Onset age: 45 Duration: 1 mo Follow-up: 3 mo	Personal history: – Aspect of lesions: Suppurating lesions Topography: Axillary folds Treatment: Oxacillin antiseptics Evolution: Improvement +, lost to follow-up
Case 8 Age: 45 Sex: M Onset age: 45 Duration: 2 mo Follow-up: 6 mo	Personal history: Smoking Aspect of lesions: Bridle scars, folliculitis, sebaceous cysts Topography: Axillary folds, perianal area Treatment: Doxycyclines 200 mg/day, antiseptics, topical erythromycin Evolution: Improvement ++, relapses at antibiotic withdrawal

Case 9	Personal history: Smoking
Age: 38	Aspect of lesions: Bridle scars, purulent fistulas, abscess, keloidal atrophic
Sex: M	scars
Onset age: 24	Topography: Axillary folds, perianal, intergluteal regions
Duration: 14 yr	Treatment: Rifampicin, antiseptics, topical erythromycin
Follow-up: 20 mo	Evolution: Improvement ++, relapses at antibiotic withdrawal
Case 10	Personal history: Acne vulgaris
Age:	Aspect of lesions: Nodulo-cystic lesions, comedones, papulo-pustules,
Sex:	bridles, interconnecting abscesses, keloidal atrophic scars
Onset age:	Topography: Face, back, axillary folds
Duration: yr	Treatment: Doxycyclines 200 mg/day, antiseptics, surgery
Follow-up: 6 mo	Evolution: Improvement ++, relapses at antibiotic withdrawal
Case 11	Personal history: Acne vulgaris, smoking
Age:	Aspect of lesions: Nodulo-cystic lesions, comedones, papulo-pustules,
Sex:	interconnecting abscesses
Onset age:	Topography: Axillary folds, perianal, intergluteal regions, face
Duration: yr	Treatment: Isotretinoin 1 mg/kg/day, surgery
Follow-up: 6 mo	Evolution: Improvement ++, relapses at retinoid withdrawal

Table 2. Clinical classification of HS in our patients

Hurley's clinical classification (stages)	Sartorius score
Ι	-
III	-
II	35
III	90
III	56
II	38
II	19
III	85
II	33
III	-
III	42
	III III III III III III III III III II

associations (Darier's disease and Down syndrome) were noted in two patients.

Treatment consisted of antibiotics (oxacillin, doxycyclin, or rifampicin) in eight patients, retinoids (1 mg/kg/day) in three patients, and local surgery of discharging nodules and abscesses in three patients. Mean follow-up was 16 months (range 2–36 months). Follow-up was marked with recurrence of lesions in all patients approximately 1 month after treatment withdrawal, leading to cyclic readministration of antibiotics or retinoids. The Down syndrome patient developed vaginal hydrocele of the testis as a complication of staphylococcic ulcers. Healing occurred with substantial scarring (keloidal atrophic scars) in three patients. One patient had limited surgical excision of keloidal scars.

Discussion

Hidradenitis suppurativa (HS) is a rare chronic suppurative condition that is associated with significant morbidity.

The exact incidence of HS is uncertain, but the literature suggests from 1 in 300 to 600 (4–6). HS is probably more common than once thought, but the diagnosis is frequently ignored or missed.

The disease seems to be more common in females, with reported female: male ratios ranging from 2:1 to 5:1 (4–6). In our series, a marked male preponderance is noted with a sex ratio (M/F) of 10/1.

The average age of onset of HS, as in our patients, is 23 years (4-6). In less than 2% of cases, the disease appears before age 11 and, in extremely rare cases, it occurs after menopause (7). In addition, the disease tends to ease or subside in women after menopause, probably in relation to hormonal changes (7, 8).

An increased frequency of HS is observed in blacks, possibly because blacks have a greater density of apocrine glands than whites (4, 8). In our series, all patients had dark phototype (IV or V). HS has polymorphic clinical and evolutive courses. Disease onset is insidious, and early symptoms may include discomfort, itching, erythema, burning, and hyperhidrosis. Occlusion of a hair follicle results in large multiheaded comedones, then nodules or cysts (3, 9, 10). The cyst may rupture spontaneously, leading to purulent discharge and chronic draining sinuses. Otherwise indurated inflammatory deep abscesses may occur. The lesions then heal with fibrosis, leading to hypertrophic or keloid scarred skin and subcutaneous tissues (6, 9, 10).

The clinical course varies from occasional axillary lesions to diffuse abscess formation in multiple sites. The sites of predilection of lesions are genitofemoral areas in women and perianal involvement in men. No gender predilection is seen in the axillary lesions (4, 6).

Patients are evaluated using the simplified Hurley's clinical classification (9) or the Sartorius severity score (10).

In the case of suspicious lesions as well as perianal involvement, biopsies should be performed to exclude the possibility of coexisting cancer, and Crohn's disease should also be considered. In the majority of specimens, the histological examination reveals follicular involvement, including poral occlusion and folliculitis. Apocrinitis as the dominant histological feature is found in only a small number of specimens (11). Furthermore, a paucity of apocrine glands is noted in the genitofemoral region. This finding supports the theory that apocrine gland inflammation is not the pathogenetic mechanism of HS, but rather a secondary manifestation of follicular involvement (6, 12).

Bacteriological analyses of HS have also been carried out. Deep needle aspiration and the carbon dioxide (CO_2) laser method have revealed *Staphylococcus aureus* and coagulase-negative staphylococci to be the most commonly found bacteria (13, 14). Imaging studies including MRI and ultrasound have otherwise been used to determine locoregional involvement (15, 16). In our patients, bacteriological analysis revealed *Staphylococcus aureus* and coagulase-negative staphylococci in seven patients tested.

The etiology of HS is still being debated. Although several studies have failed to demonstrate human lymphocyte antigen (HLA) associations (17), others have suggested an autosomal dominant mode of inheritance (18). Otherwise, hyperandrogenism, obesity via occlusion and maceration, heat, humidity and friction from clothing, smoking, lithium, chemical irritants, and oral contraceptives may be associated with HS, possibly as triggering factors (19-24). The bacterial infection is thought to occur secondary to the disease process (25). In addition, HS has been reported to coexist with other skin diseases that show poral occlusion; for example, Fox-Fordyce disease, pityriasis rubra pilaris, steatocystoma multiplex, and Dowling-Degos disease (4). Associations with Down syndrome, Behçet's disease, acanthosis nigricans, pyoderma gangrenosum, pyoderma vegetans, and arthropathy have also been described (4). Regarding the relationship with Crohn's disease (CD), some have hypothesized that perianal involvement of HS appears to be another cutaneous manifestation of CD (26). Although foreign body-type granulomas are a common finding in HS, the presence of discrete epithelioid granulomas in the dermis away from the site of active inflammation is unusual and should alert the pathologist to the possibility of a systemic granulomatous disease such as CD (26, 27). In our study, pathological associations have included Down syndrome and Darier's disease, and to our knowledge there has been no previous report of such an association in the literature. The association between diabetes and HS as in patient 5 is sporadically described (28). HS is often a diagnostic challenge, and in the early stages the differential diagnosis includes a painful nodule, abscess, furuncle, carbuncle, lymphadenitis, and ruptured inclusion cyst (4, 27). In later stages, when inflammation of more than one gland is present, the differential includes lymphogranuloma venereum, donovanosis, scrofuloderma, tuberculous gumma, actinomycosis, sinus tracts, and fistulas occurring with ulcerative colitis and regional enteritis (4, 27, 28).

HS is a recurrent disease with a chronic and progressive clinical course. When measured by the Dermatology Life Quality Index (DLQI), patients experience a significant degree of morbidity, with the highest scores resulting from pain caused by disease. Additionally, quality of life seems to be lower than in other dermatologic diseases such as urticaria, psoriasis, atopic dermatitis, and neurofibromatosis (29).

Potential complications include dermal contraction, local or disseminated infection, lymphedema caused by lymphatic injury from inflammation and scarring, rectal or urethral fistulas, restricted limb mobility from scarring, and arthritis secondary to inflammatory injury (4). Reports of squamous cell carcinoma following chronic lesions of HS have been described (30). Other rare but serious complications of HS are bacterial meningitis, bronchitis, pneumonia, and systemic amyloidosis (4). In our study, the Down syndrome patient developed vaginal hydrocele of the testis as a complication of staphylococcic ulcers. Two patients presented severe keloidal and atrophic scars.

There is no single effective treatment for HS. In mild cases we can begin with conservative measures such as warm baths, hydrotherapy, cryotherapy, and topical cleansing agents to reduce bacterial load (31). Nonsteroidal anti-inflammatory drugs may alleviate pain as well as inflammation. Antibiotics, although not proven to be effective, are the mainstay of medical treatment, especially for lesions suspected of being superinfected. The only topical antibiotic that has been proven effective in a randomized controlled trial is clindamycin (32). Antistaphylococcal agents are best for axillary disease, and more broadspectrum coverage is better for perineal disease. Dicloxacillin, erythromycin, tetracycline, and minocycline have been used with various results (32). Other hormonal medications have been tried (oral contraceptive agents that contain a high estrogento-progesterone ratio and low androgenicity of progesterone, anti-androgen cyproterone acetate in conjunction with ethinylestradiol in females and

the 5-alpha-reductase inhibitor, finasteride in males) with various responses (33, 34). Oral retinoids (isotretinoin, acitretin, etretinate) have also been used with prolonged remissions (35, 36). Systemic or intralesional corticosteroids, immunosuppressant agents, and intramuscular human immunoglobulin are other treatment possibilities (6, 31). Significant clinical improvement with long remissions was also reported after administration of infliximab and etanercept (37, 38). Radiotherapy has been investigated as a potential treatment option (39). Aminolevulinic acid photodynamic therapy for HS has also been reported as an interesting alternative (40). Otherwise, carbon dioxide laser has been used in conjunction with second-intention healing to provide relief for a few patients (41).

Finally, although incision, drainage, and exteriorization of individual lesions may be useful in some instances, radical surgical excision at the earliest recognized stage remains a mainstay of therapy. Postoperative recurrence is common after incision and drainage with limited surgical excision (42).

In our study, nine patients had antibiotics (1to 3-month cures) and three patients had systemic retinoids (1 mg/kg/day) with relapses after the first month of treatment withdrawal. Three patients had incision, drainage, and exteriorization of individual lesions, and one had limited surgical excision of keloidal scars.

Conclusion

HS remains a challenging disease for patients and physicians. In addition to treating the physical illness, it is crucial to acknowledge and treat the psychological burden associated with the disease. Because of the areas of the body that are affected, the malodorous discharge, the chronic discomfort, and the general unsightliness of the disease as well as years of inadequate treatment, HS may lead to frustration, depression, and isolation.

References

1. Verneuil A. Etudes sur les tumeurs de la peau et quelques maladies des glandes sudoripares. Arch Gen Med. 1854;4:447-68, 693-704.

2. Pillsbury DM, Shelly WB, Kligman AM. Bacterial infections of the skin. In: Pillsbury DM, editor. Dermatology, 1st ed. Philadelphia: W. B. Saunders; 1956. p. 482–4, 489.

3. Attanoos RL, Appleton MA, Douglas-Jones AG. The pathogenesis of hidradenitis suppurativa: a closer look at apocrine and apoeccrine glands. Br J Dermatol. 1995;133:254–8.

4. T Jansen, Altmeyer P, Plewig G. Acne inversa (alias hidradenitis suppurativa). J Eur Acad Dermatol Venereol. 2001;15:532–40.

 Jemec GBE, Heidenheim M, Nielsen NH. The prevalence of hidradenitis suppurativa and its potential precursor lesions. J Am Acad Dermatol. 1996;35:191–4.

6. Alikhan A, Lynch PJ, Eisen DB. Hidradenitis suppurativa: a comprehensive review. J Am Acad Dermatol. 2009 Apr;60(4):539–61; quiz 562–3.

 Mengesha YM, Holcombe TC, Hansen RC. Prepubertal hidradenitis suppurativa: two case reports and review of the literature. Pediatr Dermatol. 1999;16:292–6.

8. Paletta C, Jurkiewicz MJ. Hidradenitis suppurativa. Clin Plast Surg. 1987;14:383-90.

9. Hurley HJ. Axillary hyperhidrosis, apocrine bromhidrosis, hidradenitis suppurativa, and familial benign pemphigus: surgical approach. In: Roenigk RK, Roenigk HH, editors. Dermatologic surgery. New York: Marcel Dekker; 1989. p. 729–39.

10. Sartorius K, Lapins J, Emtestam L, Jemec GB. Suggestions for uniform outcome variables when reporting treatment effects in hidradenitis suppurativa. Br J Dermatol. 2003;149:211–3.

11. Jemec GB, Hansen U. Histology of hidradenitis suppurativa. J Am Acad Dermatol. 1996;34:994-9.

12. Yu CC, Cook MG. Hidradenitis suppurativa: a disease of follicular epithelium, rather than apocrine glands. Br J Dermatol. 1990;122:763–9.

13. Jemec GB, Faber M, Gutschik E, Wendelboe P. The bacteriology of hidradenitis suppurativa. Dermatology. 1996;193:203-6.

14. Lapins J, Jarstrand C, Emtestam L. Coagulase-negative staphylococci are the most common bacteria found in cultures from the deep portions of hidradenitis suppurativa lesions, as obtained by carbon dioxide laser surgery. Br J Dermatol. 1999;140:90–5.

15. Kelly AM, Cronin P. MRI features of hidradenitis suppurativa and review of the literature. AJR Am J Roentgenol. 2005;185:1201-4.

16. Wortsman X, Jemec GBE. Real-time compound imaging ultrasound of hidradenitis suppurativa. Dermatol Surg. 2007;33(11):1340–2.

17. O'Loughlin S, Woods R, Kirke PN, et al. Hidradenitis suppurativa. Glucose tolerance, clinical, microbiologic, and immunologic features and HLA frequencies in 27 patients. Arch Dermatol. 1988;124:1043–6.

18. Fitzsimmons JS, Fitzsimmons EM, Bilbert G. Familial hidradenitis suppurativa: evidence in favour of single gene transmission. J Med Genet. 1984;21:281–5.

19. Mortimer PS, Dawber RPR, Gales MA, Moore RA. Mediation of hidradenitis suppurativa by androgens. Br Med J. 1986;292:245–8.

20. Yosipovitch G, DeVore A, Dawn A. Obesity and the skin: skin physiology and skin manifestations of obesity. J Am Acad Dermatol. 2007 Jun;56(6):901–16; quiz 917–20.

21. König A, Lehman C, Rompel R, et al. Cigarette smoking as a triggering factor of hidradenitis suppurativa. Dermatology. 1999;198:261–4.

22. Revuz JE, Canoui-Poitrine F, Wolkenstein P, Viallette C, Gabison G, Pouget F, Poli F, Faye O, Roujeau JC, Bonnelye G, Grob JJ, Bastuji-Garin S. Prevalence and factors associated with hidradenitis suppurativa: Results from two case-control studies. J Am Acad Dermatol. 2008;59:596–601.

23. Morgan WP, Leicester G. The role of depilation and deodorants in hidradenitis suppurativa. Arch Dermatol. 1982;118:101–2.

24. Stellon AJ, Wakeling M. Hidradenitis suppurativa associated with use of oral contraceptives. Br Med J. 1989;298:28–9.

25. Jemec GBE, Faber M, Gutschick E, et al. Microbiology of hidradenitis suppurativa. Dermatology. 1996;193:203-6.

26. Roy MK, Appleton MAC, Delicata RJ, Sharma AK, et al. Probable association between hidradenitis suppurativa and Crohn's disease: significance of epithelioid granuloma. Br J Surg. 1997;84:375–6.

27. Church JM, Fazio VW, Lavery IC, Oakley JR, Milsom JW. The differential diagnosis and comorbidity of hidradenitis suppurativa and perianal Crohn's disease. Int J Colorectal Dis. 1993;8:117–9.

28. Von der Werth JM, Jemec GBE. Morbidity in patients with hidradenitis suppurativa. Br J Dermatol. 2001;144:809–13.

29. Wolkenstein P, Loundou A, Barrau K, Auquier P, Revuz J. Quality of life impairment in hidradenitis suppurativa: a study of 61 cases. J Am Acad Dermatol. 2007 Apr;56(4):621–3.

30. Diaz D, Calvo-Serrano M, Martinez-Hijosa E, et al. Squamous cell carcinoma complicating perianal hidradenitis suppurativa. Int J Colorect Dis. 1995;10:225–8.

31. Slah N. Hidradenitis suppurativa: a treatment challenge. Am Fam Physician. 2005;72(8):1547-52.

32. Jemec GB, Wendelboe P. Topical clindamycin versus systemic tetracycline in the treatment of hidradenitis suppurativa. J Am Acad Dermatol. 1998;39:971–4.

33. Mortimer PS, Dawber RP, Gales MA, Moore RA. A double-blind controlled cross-over trial of cyproterone acetate in females with hidradenitis suppurativa. Br J Dermatol. 1986;115:263–8.

34. Farrell AM, Randall VA, Vafaee T, Dawber RP. Finasteride as a therapy for hidradenitis suppurativa. Br J Dermatol. 1999;141:1138–9.

35. Boer J, van Genert MJP. Long-term results of isotretinoin in the treatment of 68 patients with hidradenitis suppurativa. J Am Acad Dermatol. 1990;40:73–6.

36. Chow E, Mortimer P. Successful treatment of hidradenitis suppurativa on retroauricular acne with etretinate. Br J Dermatol. 1992;126:415–9.

37. Sullivan TP, Welsh E, Kerdel FA et al. Infliximab for hidradenitis suppurativa. Br J Dermatol. 2003;149:1046-9.

38. Cusack C, Buckley C. Etanercept: effective in the management of hidradenitis suppurativa. Br J Dermatol. 2006;154:726–9.

39. Frohlich D, Baaske D, Glatzel M. Radiotherapy of hidradenitis suppurativa-still valid today? Strahlenther Onkol. 2000;176:286-9.

40. Gold MH. Aminolevulinic acid photodynamic therapy for hidradenitis suppurativa. Dermatol Clin. 2007 Jan;25(1):67-73.

41. Lapins J, Sartorius K, Emtestam L. Scanner-assisted carbon dioxide laser surgery: a retrospective follow-up study of patients with hidradenitis suppurativa. J Am Acad Dermatol. 2002;47:280–5.

42. Slade DE, Powell BW, Mortimer PS. Hidradenitis suppurativa: pathogenesis and management. Br J Plast Surg. 2003;56:451-61.

A U T O R S ' A D D R E S S E S	Amel Mebazaa, MD, Dermatology Department, La Rabta Hospital, Rue jabbari 1007, Tunis, Tunisia, corresponding author, Tel.: +216 23 218 392, Fax: +216 71 570 506, E-mail: amebazaa@yahoo.fr
	Rym Ben Hadid, MD, same address
	R. Cheikh Rouhou, MD, same address
	Sondes Trojjet, MD, same address
	Dalenda El Euch, MD, same address
	Mourad Mokni, MD, Professor, same address
	Moncef Zitouna, MD, Professor, Department of Histopathology, La Rabta Hospital. Tunis, Tunisia
	Amel Ben Osman, MD, Professor, Department of Dermatology, La Rabta
	Hospital. Tunis, Tunisia