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Presentation

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Therapeutic alternatives in ungueal psoriasis. Brief review of its pros and cons

NAVARRO-DURÁN Llary´*†, TORRES-GUERRERO Edoardo´´, GONZÁLEZ-MEDINA Elisa Monserrat´´, RAMOS-BETANCOURT Laura´´ and LACY, Rosa María´´

´Universidad Autónoma Metropolitana, Xochimilco. Mexico
´´Hospital "Dr. Manuel Gea González ". Mexico City.

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Abstract

Psoriasis is an inflammatory, autoimmune disease. Nail affection occurs in 40 to 50%, however, frequently the management is not adequate. Nowadays a wide range of therapeutic options are availa-ble that includes topic, intralesional or systemic drugs and also biologic agents; none has been 100% effective. Every option present advantages, and adverse effects, that must be considered based on the severity of nail compromise, clinical type of psoriasis and patient to treat.

Nail psoriasis, topic treatments, systemic treatments, biological agents, indications, side effects.

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* Correspondence to Author (email: lalotorresg@yahoo.com.mx)
† Researcher contributing first author.

Introduction

Psoriasis is an autoimmune inflammatory disease that occurs in genetically susceptible individuals-mind. It comes with exacerbations and remissions and involves the development of injuries of varying severity that affect not only skin but also nails and joints [1]. Is estimated to affect about 2% of the world population [2]. This calculated figure may be far from the number of actual cases because a large percentage of the population is not in a position to go or no medical services, and that this condition may be underdiagnosed or subject to misdiagnoses [3].

Although the first concepts regarding the pathogenesis of psoriasis are mainly focused on keratinocyte hyperproliferation, deregulation of the immune system is now recognized as a critical factor in this disease. Studies have supported the concept that Interacci-tions between different cells and cytokines probably contribute to the initiation and perpetuation of skin inflammation. A basic theoretical sequence immunological events occurring in psoriasis are described below: antigenic stimuli contribute to the activation of dendritic cells and other innate immune cells in the skin; proinflammatory cytokines produced by the innate immune cells, including interferon alpha (IFN), active-ing stimulate myeloid dendritic cells in the skin; they produce cytokines such as IL interleukin IL -23 and -12 that stimulate attraction, activation and differentiation of T cells recruited These T cells produce cytokines that stimulate keratinocyte proliferation and production of proinflammatory cytokines and peptides that perpetuate the process inflammatory [4].

The nail involvement occurs in 40-50% of adult patients with psoriasis; up to 13% of pediatric patients with this disease and this prevalence increases to 87% in those with psoriatic arthritis [5, 6].

There is a positive association between psoriasis and nail the duration and severity of skin lesions [6]. 5% of the patients with psoriasis, nail lesions may occur without presenting cutaneous manifestations [7]. Despite its relative frequency, nail psoriasis often overlooked and not treated effectively. This has important implications for patients with negative impact on the fun-tioning and quality of life. In addition, it can be a predictor of future inflammatory damage arti-lar (precursor of psoriatic arthritis) and is a visible indicator of disease activity [8].

The nail apparatus consists of the following anatomical structures: the nail plate, the periungual folds, matrix and bed [9]. Clinical morphology depends on the anatomical location of the pathological process [1] may affect any element, whether the bed and / or the matrix. This results in different clinical signs.

The most common findings are alterations of the bed comprising the following:

Onycholysis: One of the most frequent alterations and features. A distal detachment of the sheet occurs with respect to the bed, in which a more or less large whitish area surrounded by an erythematous appearance collarette with an oil stain is observed. Sometimes the whitish hue takes a greenish or brown due to colonization by bacteria or fungi. Subungual hyperkeratosis: It is important because parakeratotic proliferation of cells, which results in a dense, powdery, whitish mass distally off the nail; and it is the most clinically confused with onychomycosis. Oil stains or salmon patch: It is the only exclusive nail psoriasis lesion. Round or oval areas are seen in the center of the sheet of orange color. Splinter hemorrhages: These are linear, with threadlike appearance. Usually they are seen only in the fingers [1, 6, 10].

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These lesions can present isolated or associated with lesions of the matrix, in which case it can be observed pits or dimples, which are usually multiple and irre gular-punctate depressions, which are caused by transient focal involvement of the proximal matrix. These dimples correspond to paraqueratosis islets that by eliminating the appearance of the nail, leave blues-making in the film. Trachyonychia appears as the result of a permanent alteration of the proximal matrix, where a rough, dull surface is observed. Other related findings are leukonychia (partial or total white coloration of the nail plate, due to the involvement of the intermediate parent), Beau lines (horizontal depressions that represent involvement of the proximal or intermediate matrix along its length) and red crescent (involvement of the distal matrix) [1, 6, 10].

This condition resembles other onicodistrofias, the most common differential diagnosis of onychomycosis, so it is recommended to rule out this disease before treatment instituted (and if confirmed, must be resolved first), and in patients with psoriasis of the nails the risk of a fungal superinfection increases to 27% [10], plus there are hypotheses that suggest that onychomycosis can be a factor that aggravates or perpetuates psoriatic nail dystrophy [11].

In a publication by Fischer-Levancini, it was reported that up to 50% of patients with nail psoriasis referred pain associated with these events [5], a finding consistent with an en-study by De Jong et al, with a total of 1728 patients with nail psoriasis, who reported the presence of this symptom as much as 51.8% of the total and recorded more than 90% patients concerned about their aesthetic appearance. The activities of daily living were negatively affected in 58.9% of patients and a similar number (56.1%) reported that nail psoriasis inhibited normal activities of cleaning staff.

Regarding professional activities, 47.9% of these patients were adversely affected [12].

Therapeutic strategies and resources.

So far, it has a wide range of drug options for the treatment of nail psoriasis, something that represents a therapeutic challenge for dermatologists, and motivates the search for new solutions thereof including topical medications, intralesional, and systemic with biological action; none of which has been 100% effective. Each of these options has advantages and themselves, these side effects to be taken into account depending on the degree and type of nail condition of the patient being treated; not to mention the variability in costs.

Table 1 topical and systemic treatments tested until now are listed.

Due to many factors, treatment of nail psoriasis is complicated. On the one hand, this is due to the slow growth of this Annex and the difficulty in penetrating topical treatments; besides there are few available topical treatments. Meanwhile, oral medica-ments have limited use because of its systemic toxicity, in addition to the literature reports little attachment to them when the nails are the only affected (or where skin lesions are not important structure for the patient in the presence of nail lesions) [13, 14, 15].

For local treatment, steroid injections are considered the measure of choice. These drugs have anti-inflammatory, immunosuppressive and antiproliferative effects (which sig-nifica that these drugs act on all aspects of the disease physiopathogenic) [16].

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However, these infiltrations generate great pain, influence the development of atrophy, hipocromías, superinfection, inclusion cysts and tendon ruptures, so many PATIENT-tes discontinue treatment. Given the above, such as UL-esteem traquioniquias intense treatment option recommended only in severe cases and only if the condition is one or two fingers. Triamcinolone acetonide is the most commonly used agent bimonthly, at doses of 2.5 to 10 mg / ml to a maximum of four injection points (two in the proximal nail fold and two on the side fold) for 6 months [17] .

As another alternative within this family of drugs, clobetasol propionate concentrated-ing to 0.05% cream or gel, it is used twice a week for 4 months. The disadvantages are the percentage improvement reported (51%) and side effects (may cause atrophy, de-pigmentation, and telangiectasia presence of bone resorption) [12,17]. This same steroid at a concentration of 8% lacquer in scheme application once daily for the first week and second week onwards with applications 2-3 times a week for up to 9 months has proven both have adequate penetration into the bed and matrix, without adverse effects, with good results [18]. However, in Mexico there is available a commercial formulation of this substance, and although it can be appealed to the master formulations to address this issue, candidal superinfection has been reported after treatment with this steroid (Figs. 1-A and 1- B) [10].

Other topical therapeutic modalities used include 5 - fluorouracil, which is used dissolved in a 1% solution of propylene glycol or urea cream with 20% applied twice a day for at least six months. This drug is a pyrimidine analog that acts by irreversible inhibition of DNA synthesis, blocking cell proliferation [19].

Its use is recommended especially in the presence of dimples and subungual hyperkeratosis; besides improving dystrophies origin matrix up to 50% [20]; in cam-bio, clearly worse onycholysis, so it is suggested to avoid its application to this manifest-ing and as side effects can cause irritation and hyperpigmentation (Fig. 2) [10, 21,22].

Derivatives of vitamin D (Fig. 3), such as calcipotriol, tacalcitol and calcitriol, interac-They act with vitamin D receptors, promote cell differentiation and immunomodulation, while inhibit cell proliferation and expression of cutaneous lymphocyte associated antigen (CLA, for its acronym in English -Associated Cutaneous Lymphocyte Antigen), which translates clinically in improved nail bed alterations [16], acting positi-tively in reducing hyperkeratosis, however, its effects are limited to the parent company level, observed in a study conducted by Urbina and Sudy in Chile, little improvement pique-teado [23] so it should always be combined with corticosteroids, which raises even the cost of treatment. Recommended treatment schemes are: Calcipotriol twice daily or calcipotriol + betamethasone once a day for 3 months [24, 25].

Tazarotene (third generation derivative of retinoic acid from [Fig. 4]), exerts its effect by activating and regulating gene transcription, which modulate and induce the expression of epidermal growth factor, promote epidermal cell differentiation, exert effects immunomodulatory and regulate the growth of hyperproliferative epithelia; so it is an excellent modality for the treatment of disorders such as psoriasis [16].

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This drug presentation hydrophilic ointment gel or 0.1% applied once daily has been shown to significantly improve subungual hyperkeratosis, onycholysis, oil spots and the dimples, after 12 weeks of daily use. Side effects were rare and were Prolon-gada improvement (especially hyperkeratosis) to stop treatment, but remember that it is contraindicated in pregnancy (teratogenicity), it is expensive and not very effective in removing other changes resulting from damage to the nail matrix [5, 26, 27].

Tacrolimus is a macrolide immunosuppressant that acts by inhibiting calcineurin phosphatase. This lock prevents lymphocytes can dephosphorylate the cytoplasmic subunit of nuclear factor of activated T cells (NFAT by its acronym Nuclear Factor of Activated T Cells), which in turn prevents transcription of numerous inflammatory cytokines [19]. In studies with this drug has been observed generally good response, however, it has also been a frequent association with acute paronychia, aspect to take into consideration with-[14].

As for systemic treatment, it has cyclosporine, which is considered a PROFAR-maco, which acts by binding to cyclophilin, exerting a similar tacrolimus effect (Figs. 5-A and 5-B) [16]. This drug dose of 5 mg / kg daily produces improved nail lesions. However, due to their profile of liver and kidney toxicity, there is no justification to indicate it to isolated nail psoriasis [28]. In a study investigating the efficacy and safety of methotrexate (which blocks cell division in S phase and exerts an anti-inflammatory effect by the increase in tissue levels of adenosine) at doses of 15 mg / week and cyclosporin 5mg / kg was compared after 6 months of treatment, concluded that both drugs were equally effective in nail psoriasis. However, methotrexate was more effective in lesions of the matrix, whereas cyclosporin was more effective on the injury bed [16, 29].

Besides the above, we must not forget the major side effects associated with methotrexate, such as pancytopenia (by myelotoxicity) and liver toxicity, same that occurs with long-term treatment (as in the case of psoriasis) [16].

Among the physical treatment modalities are photochemotherapy with 8-MOP (I put-xipsoraleno), same as minister topically or orally, with subsequent exposure to UVA rays and generates good results, especially when the damage to nail apparatus It presents with pustules and / or hyperkeratosis, but on the contrary, onycholysis worsens [5, 10]. Until the advent of the derivatives of vitamin D, this was one of the handiest therapeutic strategies [10]; however, remember the side effects that include fotooni-cólisis and burns of the periungual tissues, this method can also predispose photosensitivity reactions, as well as exposing the patient to the side effects of psoralens (when they are indicate systemically), not to mention that UVA is limited number of sessions, beyond which it can no longer exposed [19], so that if a relapse occurs at the end of this treatment, not It is possible to re-use this mode.

Also, good results with photodynamic therapy and laser dye, which have shown to be effective in correcting alterations in both the bed and matrix are mentioned; However, the former has the disadvantage of discomfort and pain associated with their application and both are to the disadvantage of its cost (taking into account that talking about long-term treatment) [5].

At present there are relatively few studies that have examined the use of modifiers specific biological response in the treatment of nail psoriasis.

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These compounds work by means of mechanisms including interaction with receptors that alter or block the activation and differentiation of T cells, as well as, inhibit the release of cytokines and eliminate pathogenic B cells. The main target of action of these drugs created by bioengineering is the Tumor Necrosis Factor (TNF) [16]. Current evidence suggests that TNF as inhibitors infliximab, adalimumab and etanercept are effective for this pur-site, but have a very high cost and its use is not approved for the treatment of nail ma-isolated manifestations; [30] besides their dosing schedules are com-plex, for example infliximab (which in turn, is the antibody with the best results have been observed with the most experience trials have) the dosage is 5 mg / kg, intravenous infusion at weeks 0, 2, 6 and every 8 weeks until week 38-46 [31].

So it has recently tested a water soluble nail lacquer containing hidroxipropilqui-cough (copolymer of glucosamine and N-acetylglucosamine units linked by glucosídi-cos links; very abundant in crustaceans, fungi and insects), horsetail extract (Equisetum vense ar-rich silicates, which bind to the nail keratin reinforcing structure) and methyl sulfonyl methane (which gives elasticity to the nail and reduces their fragility). This product is effective for strengthening nails and reduces their roughness; plus it has some antimicrobial activity which has resulted in combination with antifungals such as ciclopirox to treat onychomycosis. In a clinical trial conducted to verify if this lacquer was able to improve the signs of nail psoriasis, dimples and decreasing the degree of onychodystrophy it was shown without any adverse effects (except mild irritation periungual folds) will report ; although in some cases observed in the authors' experience, no significant changes (unpublished data) [32,33, 34] showed.

Conclusions

The condition of the nails in patients with psoriasis is very common, with the percentage involvement is between 10 and 78%. The fingernails and in turn are affected more than usual is that it affects more than a fingernail.

Current therapy is often ineffective or generates modest results and includes the application of topical agents or local steroid injections are very painful. Furthermore, systemic therapies are generally not recommended for patients with nail disease only. Added to this, one of the problems by checking the level of evidence available treatments is the lack of sufficient studies in literature, in addition to the validity between the different trials reported.

It is not yet has a "gold standard" in the treatment of this manifestation, sometimes as in-capacitating or as traumatic for some patients, so the choice of therapeutic approach based on the points raised in this article I allow to individualize, choosing those tools and best suited to each patient therapeutic combinations (in the case of being necessary), taking into account (among other things) the degree of nail condition -and cutaneous-, there is-State General health, the presence or absence of comorbidities, and prescribed treatments for each of them (with the consequent potential interactions) and possible side effects in each case.

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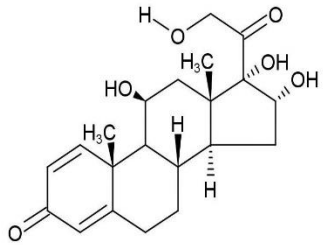
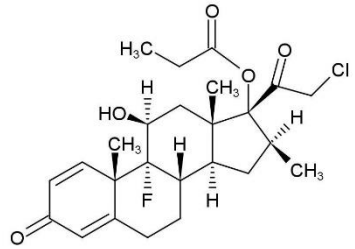


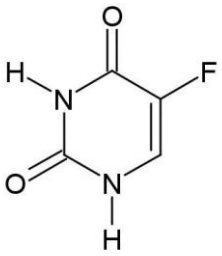
TRIAMCINOLONA

Figura 1A



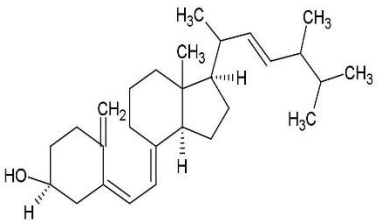
CLOBETASOL

Figura 1B



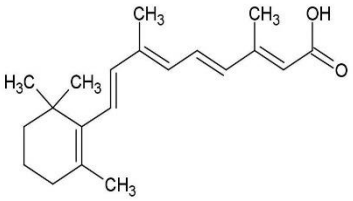
5-fluorouracilo

Figura 2



VITAMINA D
(CALCIFEROL)

Figura 3



ÁCIDO RETINOICO

Figura 4

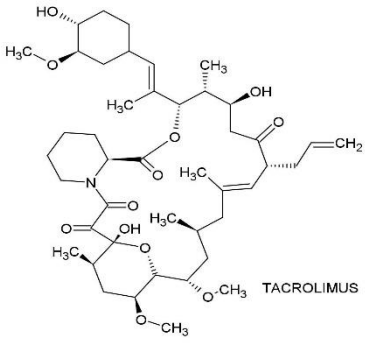
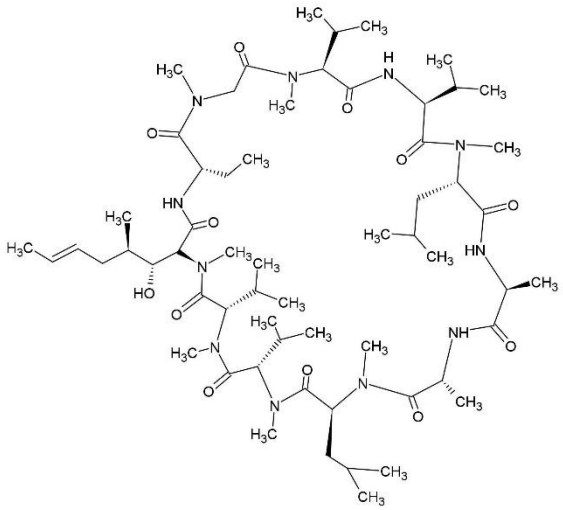


Figura 5A



CICLOSPORINA A

Figrua 5B

Tratamiento de aplicación tópica o intralesional	**Nivel de evidencia**
- Ciclosporina tópica al 70% en aceite de maíz	IB
- Ungüento tópico de tacrolimus al 1%	III
- Clobetasol al 0.05, 0.1 y 0.8% en laca	III
- Tazaroteno en gel al 0.1%	III
- Calcipotriol (con o sin betametasona)	IIB
- Fototerapia (con psoraleno y/ o acitretina)	III
- Luz pulsada de 595nm	III
- Terapia fotodinámica con MAL*	III
- Rayos Grenz	IIB
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Alopecia HIV. A review

MORENO-COUTIÑO, Gabriela*†

General Hospital "Dr. Manuel Gea González, Department of Dermatology.

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Abstract

The term alopecia (the Gr. Alopex) applies to abnormal hair loss or rarefaction in any hairy area. However, the area to which most commonly referred is to the scalp.

For patients living with HIV, not androgenetic alopecia has been associated with the ingestion of medicines, infections, or diseases of inflammatory origin and / or autoimmune.

Alopecia, HIV, hair loss.

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* Correspondence to Author (email: gmorenocoutino@gmail.com)
† Researcher contributing first author.

Alopecia of the scalp has been reported in association with antiretroviral therapy; particularly indinavir is a protease inhibitor. This adverse effect is dose-dependent and is not associated with age, gender or CD4 lymphocyte counts. It is the only dermatological adverse effect of this drug; however, the negative impact on the patient's self-image is the main cause for discontinuation.

Alopecia that occurs in up to 12-30% of patients receiving the drug, the scar type and cannot be diffuse or patchy alopecia occur anywhere in the body. It is believed that brings retinoid-like as xerosis, cheilitis, and alopecia onicocriptosis effects. Often that is more of a demonstration in affected patients. The pathophysiology is unknown but is believed to indinavir may interfere with the metabolism of retinoids, and seen from the first two months of treatment. The main differential diagnosis must be made with alopecia areata.

The best solution for hair loss is to replace the drug by another protease inhibitor [1-3]. It has also been associated with lopinavir and ritonavir. One patient had alopecia totalis of the scalp, eyebrows and eyelashes that were recovered by changing these antiretroviral nelfinavir. Another patient had diffuse loss of scalp hair also decided to replace lopinavir and ritonavir efavirenz [4].

And another in association with ritonavir and zidovudine [5,6]. The cause Darunavir indinavir similar to the adverse effects, which sometimes have been reported cases of alopecia [2].

Conversely, zidovudine (AZT), which was the first drug used for the treatment of HIV in the 80s, apparently caused increased hair growth, and even reported the reversal of alopecia areata after the start of AZT treatment [7,8]. and only report what associated with alopecia [9].

Besides association with antiretroviral alopecia, it is known that there is a higher incidence of alopecia areata is one of the most common autoimmune diseases compared to the HIV negative population [10].

And Conversely, only one report mentioned that a patient who since childhood had presented refractory alopecia areata treatment, spontaneously resolved with treatment for HIV infection almost 40 years later [11].

The diagnosis of alopecia areata should be confirmed with a biopsy, and so differentiate it from other types of alopecia patch.

As for infectious causes, tinea capitis in adults is rare, with less than 3% of all cases. In particular, adults living with HIV, is extremely rare to find cases. Some authors explain this as a result of increased colonization of Malassezia spp competitively inhibiting the development of dermatophytes.

Therefore, to diagnose a high level of suspicion is needed, in addition to relying on traditional diagnostic methods as drecto KOH examination and culture. The treatment is the same as any other case of tinea capitis [8, 12].

The co-infection of HIV and syphilis is extremely high, reported in some cities in the US to 60%

Alopecia caused by secondaries can be described as "mouse bites" fuzzy form or a combination of both. Its prevalence varies 4-12.5% of secondary alopecia, predominantly homosexual men.

Syphilitic alopecia is no scar, and may be the only manifestation of secondaries or accompanied by other mucocutaneous lesions.

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It is a common manifestation of syphilis, but should be considered for differential diagnosis should also include telogen effluvium, androgenetic alopecia, alopecia areata, tinea capitis and trichotillomania.

As no scar, it resolves with treatment of the infection. Treatment is with penicillin G benzathine 2.4 million units once a week for three weeks [13].

However, most of alopecia associated with HIV are classified as TE, triggered as a result of damage caused systemic infection such as HIV infection, endocrine diseases, immunological disorders, nutritional deficiencies, wasting syndrome, among many other causes that must be addressed properly [14].

Conclusions

Hair loss is, in most cases an aesthetic problem. However, we can not let it pass without taking into account the psychological impact it has on our patients, which may be as important to influence adherence to antiretroviral therapy. That is why, it is important to make a correct diagnosis and to influence changes that promote hair growth.

Most of the information in the literature focuses on the alopecia associated with drugs, because it is part of the long list of adverse reactions that are published. But we must not forget that there are multiple causes and should inevstigar.

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Folliculitis by *Malassezia* sp., An epidemiological study in Dominican Republic

PORRAS-LÓPEZ, Carlos*†, COMPRES-ESPINAL, Adriana, CRUZ, Cecilia and ISA-ISA, Rafael

Institute of Dermatology and Skin Surgery "Dr. Fernando Cordero C. Unit of Medical Mycology, "Guatemala City. Dominican Dermatology Institute and Skin Surgery "Prof. Dr. Hubert Bogaert Diaz. Department of Mycology, "Dominican Republic.

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Abstract

*Malassezia´*s folliculitis is a pathology characterized by the presence of papules and pustules in which *Malassezia* sp. can be isolated. This is indistinguishable from *Candida* sp. folliculitis, acne vulgaris, acneiform reaction, and some bacterial folliculitis so it may be underdiagnosed. The objective of this study was to characterize the epidemiology of *Malassezia* folliculitis through a retrospective cross-sectional study from 2009 to 2012 at the Institute Dermatological and Skin Surgery "Prof. Dr. Hubert Bogaert ", Dominican Republic. It was observed that the disease is more common in women, the most prevalent age group is 21 to 30 years, concomitant pathology is more often associated with tinea versicolor, the evolution time is less than 1 year, the most often associated site is the back. The use of Gram stain and culture is recommended to fully establish the diagnosis.

***Malassezia* sp., Gram stain, folliculitis.**

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* Correspondence to Author (email: cfporrasl_gt@hotmail.com)
† Researcher contributing first author.

Introduction

Malassezia folliculitis is a pathology characterized by papules and pustules chronic. [1.2] is associated with immunocompetent patients and can also be found in patients inmunosupresos, [3] considered a benign pathology associated with lipophilic fungus *Malassezia* sp, this entity is not distinguishable from a common acne, acneiform reaction and some bacterial folliculitis, usually in some cases it is subdiagnosticada.[4] This condition can be complicated by causing fungemia use catéter.[5,6] Clinical lesions are frequently located in the trunk, abdomen and extremities. This ringworm occurs frequently in tropical and temperate countries. Among the risk factors and are occlusion moisture and antibiotics, corticosteroids, presence of malignancies, transplant and diabetes.[7]

The genus *Malassezia* was described and characterized by Eichsted and Sluter in 1846 and 1847 respectively. The taxonomic grouping was resolved by Guillot et al., In 1995. [8]

The yeast *Malassezia* is part of the normal microbiota skin, the etiologic agent is recognized as the cause of tinea versicolor, and plays a role in the development of seborrheic dermatitis, confluent and reticulated papillomatosis, atopic dermatitis and type injuries psoriasis.[9] currently 14 known species of *Malassezia*: *Malassezia* furfur, M.

Pachydermatis, M. sympodialis, M. globosa, M. slooffiae, M. Restricted, M. obtusa, M. dermatis, M. japonica, M. yamatoensii, M.nana, equine M., M. caprae, M cuniculi. The requirement for its development in the skin is the presence of lipids, and for obtension in culture media (modified Dixon) is used commonly glycerol agar supplemented with Tween 20, 40, 60 and 80 (to achieve their isolation and identification). [10,11]

The presence of *Malassezia* in pustular lesions can be demonstrated using KOH 10% of chlorazol black, white calcoflour or Gram stain. In this case unipolar budding yeast are observed. [12,13]

Among the various treatments used in the case of *Malassezia* folliculitis selenium sulfide, econazole, clotrimazole, miconazole and ketoconzol are in the case of patients inmunocompetentes.[14] For neutropenic patients treatment often fails, Hair and Cermak (2004) report the use of ketoconazole orally without recurrence of injury in the event of a neutropenic patient. [10]

Methodology

A retrospective cross-sectional study was conducted in the department of medical mycology Dominican Dermatological Institute and Skin Surgery "Prof. Dr. Hubert Bogaert Diaz, "in which patients who came to present disseminated pustular lesions were analyzed. Patients were evaluated to determine the presence of *Malassezia* sp. lesions, achieving found 22 cases of immunocompetent patients from 2009 to 2012. Gram staining was used to establish the presence or absence of unipolar budding yeast.

Results

Of the 22 cases it found that most were female (63.64%).

Sex	**N. of cases**	**Frequency**
Female	14	63.64
Male	8	36.36

Table 1 Sex of patients

As for the age group folliculitis by *Malassezia* sp it is observed. I predominates 21-30 years (54.55%), followed by 10 to 21 years (31.82%) and 0-10 years (13.64%). The average age was 19 years.

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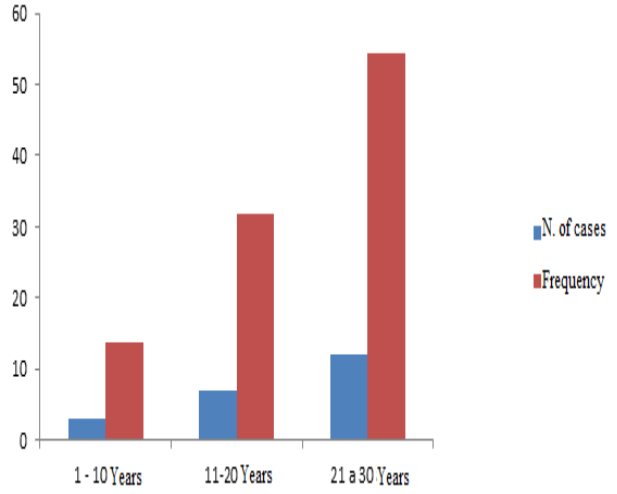
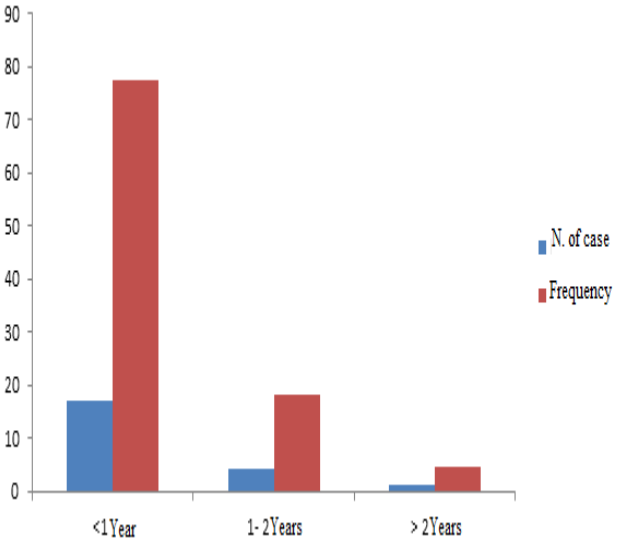


Graphic 1 Age Group

As for the time of evolution, most patients consulted before 1 year (77.27%), the average development time of six months.



Graphic 2 Time evolution

As for the distribution of lesions by *Malassezia* sp., It was found that most dominated back sole associated anatomical site and others like chest and arms.

Anatomic site	N. of cases	Frequency
Back	8	36.36
Back, chest	5	22.73
Back, chest, arms *	4	18.18
Arm, back	3	13.64
Arm, chest	1	4.55
Chest, face	1	4.55

* Three anatomical sites

Table 2 Anatomical Site

Of the 22 cases analyzed it was found that in 15 of them there was no evidence of associated disease, 3 of them had tinea versicolor, and found that there was 1 case of hypertension, 1 of atopic dermatitis, seborrheic dermatitis 1 and 1 with onychomycosis tinea pedis.

Comorbidities	N. of cases	Frequency
Missing	15	68.18
versicolor	3	13.64
Hypertension	1	4.55
Atopic dermatitis	1	4.55
Seborrheic dermatitis	1	4.55
Onychomycosis and tinea pedis	1	4.55

Antifungal most commonly used in these cases was orally itraconazole (81.82%), followed by oral ketoconazole (18.18%).

Antifungal	N. of cases	Frequency
itraconazole V.O	18	81.82
topical ketoconazole	4	18.18

Tabla 4 Antifungal used

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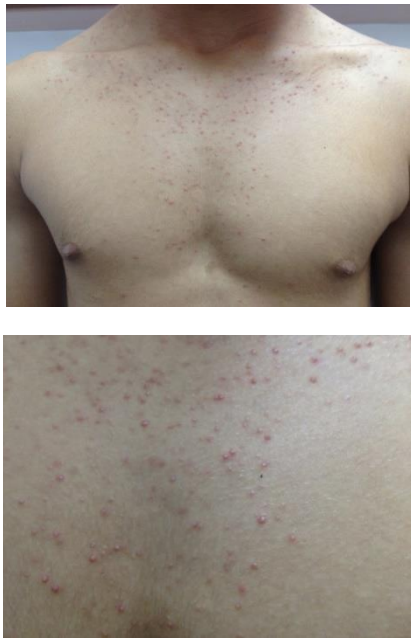

Figure 1 Clinical presentation of folliculitis by *Malassezia* sp.

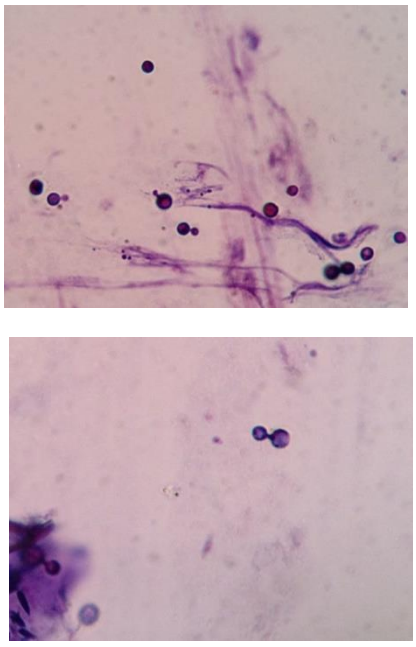

Figure 2 Observation of yeast *Malassezia* sp. Gram stain

Discussion

Regarding diagnosis of *Malassezia* folliculitis sp. one of the most common problems is the underdiagnosis that arises due to the lack of experience in identifying the injuries as pustules produced in this disease are very similar to those observed by acne, Candida sp. and acneiformes.[4,15] reactions In the present study a compilation of cases of folliculitis by *Malassezia* sp performed. in order to establish the epidemiology of the disease.

As for sex a higher frequency in women was observed 2: 1 as has been described in other studies such as that conducted Abdel et al. in Saudi Arabia in 1995.14 Other studies such as that conducted by Guzman et al. in Mexico in 2005, where more frequent point men.[12]

When performing mycological study and observe the gemantes yeast, you should be careful in establishing a diagnosis with Candida sp. since in both cases it is yeast. Among the important features that differentiate folliculitis by *Malassezia* sp. folicultis respect to Candida sp. location is, in many cases folliculitis Candida sp. It is located in hairy skin while *Malassezia* folliculitis is located on the back and pecho.16 In our study we found the presence of lesions predominantly back (49%), of which in 22% of cases have spread to the abdomen and chest (thorax).

In the present study we found the presence of folicullitis most frequently in adults (21-30 years), which correlates with the study of Guzman et al. in Mexico in 2005, which it states that the average age is 28 years in cases *Malassezia* folliculitis sp.[12]

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In this study patients attended the dermatology clinic in term of under 1 year of getting the problem time. So the presence of diseases associated with *Malassezia* folliculitis also evaluated sp., And found that the disease was more tinea versicolor association with 13.64% in a study by Abdel et al. in 1995, it was established that at a frequency of 17% was no association between *Malassezia* folliculitis sp. and tinea versicolor. [14]

It is important to note that the climate in which these cases could be identified was warm as temperatures on the island of Dominican Republic, fluctuate between 25-35 ° C, this correlates with what was said by Guzman et al., Who established the climate hot as a predisposing factor in cases of *Malassezia* sp.[12] folicultis

It has been observed in vitro sensitivity of *Malassezia* sp. ketoconazole and itraconazole. It also has a variability with respect to fluconazole, bifonazole, econazole, miconazole, clotrimazole and new azole as albaconazole and voriconazole, in the Dominican Dermatology Institute and Skin Surgery "Prof. Dr. Hubert Bogaert Diaz "the antifungals ketoconazole and itraconazole were selected. Itraconazole being the most frequently used (81.82%).

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Molecular biology in the diagnosis of invasive candidiasis

FRÍAS-DE-LEÓN, María Guadalupe*†

Juarez of Mexico Hospital, Division of Research. Av. 5160 National Polytechnic Institute, Col. Magdalena de las Salinas 07760, Mexico DF, Mexico

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Abstract

Between invasive mycosis, invasive candidiasis (IC) ranks first worldwide for its morbidity and mortality associated with immunocompromised patients, representing an important public health problema. *Candida albicans* is the most common etiologic agent of CI; however, other non-albicans species also can cause it. Some of the non-albicans species differ from *C. albicans* in their susceptibility pattern to antifungal, even some are intrinsically resistant. For these reasons, the rapid and accurate identification of *Candida* species plays an important role in the selection of appropriate therapy. Conventional methods for laboratory diagnosis of IC have restrictions that have been tried to overcome with the development of molecular and proteomic methods; however, they also have disadvantages. So it is appropriate that the diagnosis of IC is based on the results of conventional tests and complemented with some molecular for greater accuracy in identifying the pathogen that involves proper management of patients.

Molecular biology, candidiasis, diagnostic.

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* Correspondence to Author (email: magpefrias@gmail.com)
† Researcher contributing first author.

The incidence of invasive fungal infections has increased in immunocompromised patients and in critically ill patients [26]. Within invasive infections, invasive candidiasis (IC) prevails in first place worldwide, and thus represents an important public health problem [22]. The CI is caused by *Candida* yeasts, which includes more than 200 species, of which at least 17 have been reported to cause CI, being *Candida* albicans the most common, with more than 50% of cases, followed by *C. glabrata* (18%), *C. parapsilosis* (13%) and *C. tropicalis* (10%) and other rare species such as *C. guilliermondii*, *C. lusitaniae*, *C. norvegensis*, *C. inconspicuous*, *C. famata*, *C. intermedia*, C. zeylanoides, *C. pelliculosa*, *C. dubliniensis*, *C. rugosa*, *C. stellatoidea*, *C. kefyr*, *C. dubliniensis*, *C. famata*, *C. lusitaniae*, *C. norvegnesis*, *C. pelliculosa* [1,4,6,7,13,22,23]. Some of these species have low intrinsic resistance (*C. krusei*) or (*C. glabrata*) to azoles or echinocandins (*C. parapsilosis*) [19].

Importantly, the number of species associated with the CI may be higher, and that at least three species are part of complex: *C. glabrata* (*C. glabrata* strict sense, *C.* and *C. nivariensis bracariensis*), *C. parapsilosis* (*C. parapsilosis sensu stricto*, *C.* and *C. metapsilosis orthopsilosis*) and *C. guilliermondii* (sensu stricto C. guilliermondii, *C. fermentati*, *C. carpophila* and *C. xestobii*). Within these complexes, virulence and susceptibility to antifungal varies, so it is important to correctly identify [5]. C. parapsilosis complex, González et al. (14) report in CI prevalence of *C. parapsilosis sensu stricto* (95.3%), *C. orthopsilosis* (3.1%) and *C. metapsilosis* (1.6%).

Due to the etiological diversity of the CI, it is essential to identify, at the species level, the causal agent not only for diagnostic but also therapeutic and epidemiological.

For the diagnosis of IC, the clinical laboratory plays an important role in the isolation and identification of the pathogen by growing in different media and application of tests such as serological germ tube and assimilation of carbohydrates (VITEK 2 API and C AUX), among others [10]. However, these tests have several disadvantages, among which are: 1) low crop sensitivity to isolate the fungus and the need to require the fungal isolate to characterize, 2) the time required to get the result, 3) its low specificity, as they fail to differentiate between closely related species such as *C. albicans* and *C. dubliniensis*, and the species of complex *C. glabrata*, *C. parapsilosis* and *C. guilliermondii*, which are often mistakenly identified [2.3]. These disadvantages have limited the utility of phenotypic methods in clinical practice [3], so that several molecular methods have been developed to overcome the limitations of conventional tests. Molecular methods intended rapid and specific identification of the pathogen in clinical samples directly, without isolation of fungal [33].

In this paper, the advantages and disadvantages of recently developed molecular methods are reviewed, and that have shown promise in the diagnosis of IC: the polymerase chain reaction (PCR), fluorescent in situ hybridization probes peptides nucleic acids (PNA-FISH) and microarray.

PCR

The amplification of nucleic acids by PCR represents an alternative to improve early diagnosis of IC. This technique has been presented in different formats, including simplex PCR, multiplex, semi-nested, nested, coupled with enzyme immunoassay (PCR-EIA) in real time [9.29].

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White amplification (molecular markers) used to identify more *Candida* spp., In different formats PCR fragments corresponding to cytochrome P450 genes, topoisomerase II, heat shock, pH regulation, and rRNA (18S, 28S and 5.8S); however, rRNA are the most commonly used markers, because of its universal nature and number of copies [32] also contain regions of internal transcribed spacer 1 and 2 (ITS1 and ITS2) which show variability between species of the same gender [20]. Because of these characteristics, markers designed from rRNA genes have been specific and sensitive for detecting and identifying low concentrations of DNA of *C. albicans*, *C. tropicalis*, *C. parapsilosis*, *C. glabrata*, *C. dubliniensis*, *C. kefyr*, *C. krusei*, *C.* and *C. lusitaniae guililermondii* [16,17,20]. Of different formats PCR, real time PCR has the advantage of being able to quantify the fungal load, making it possible to monitor the persistence or resolution of infection, after treatment.

The PCR has proved a useful tool for the detection of *Candida* spp.,. Including commercial systems have been developed, as SeptiTest or SeptiFast for pathogen detection in clinical samples directly. The SeptiTest system employs a method of extracting DNA from blood and subsequently amplified by multiplex PCR and sequenced for identification of diverse *Candida* species. SeptiFast system in DNA extracted from blood samples and amplified by real-time PCR, wherein the amplicons are hybridized with specific fluorescent and 25 for identifying pathogens, including *Candida* probes. This procedure is completed in approximately six hours [1].

However, the PCR in different formats, has certain limitations that have prevented its applicability in the diagnosis of IC.

The main constraints are the lack of standardization of procedures for DNA extraction from clinical samples, and in some cases, the need for sequencing the amplicons, as this increases the cost of the test and time to get the results. Furthermore, in the case of real-time PCR, it has not been established between the limits of detection of DNA associated with *Candida* colonization or invasion, which may generate false positive results [8].

PNA-FISH

PNAs are synthetic peptides consisting pseudo base pairs having the same conformation of a nucleic acid. The PNA probes have neutral charge and very favorable characteristics for hybridization, as its high specificity, high affinity, fast kinetics and the lack of electrostatic repulsion. Additionally, the relative hydrophobicity of the PNA probes allows them to penetrate the hydrophobic cell wall of the microorganisms more easily [31]. Such probes are designed based on the region of the 26S rRNA of *C. albicans*, in order to identify the yeast in positive blood culture bottles. Upon hybridizing the probe with yeast, specific fluorescence is emitted for each species of *Candida*, which can be detected by fluorescence microscopy or flow cytometry. In addition, the PNA-FISH technique has had a positive impact on the selection of antifungal therapy, because when properly identify *C. albicans*, has managed to avoid excessive consumption of some antifungals, such as caspofungin [18,28,31].

Despite its advantages, the use of this technique has limited the clinical laboratories because of the cost of equipment (fluorescence microscopy) and reagents [30].

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Microarrays

The technology of microarray detection system represents a broad spectrum which can be very useful in view of the large number of pathogenic *Candida* species, it also allows the analysis of different types of biological samples (tissues, proteins, nucleic acids) [25]. Therefore, a microarray was developed for rapid diagnosis and simultaneous identification of 12 common fungal species of the genera *Candida* and Aspergillus. The probes were designed based on the analysis of changes in the ITS regions of the rRNA gene. Through the use of general primers (ITS1 and ITS4) directed towards the conserved regions of the 18S rRNA genes and 28S, respectively, the white region STI is simultaneously amplified and fluorescently labeled. This method has been validated with fungal isolates and clinical samples, showing satisfactory results in only four hours after DNA extraction, so it has a potential use in clinical laboratories mycology [21].

A great advantage of using microarrays is that can be analyzed simultaneously, multiple molecules in a single assay, making it possible not only to identify the pathogen, but also detect mutations that are associated with resistance to antifungal [25]. However, widespread use of microarrays is limited by the relatively small amounts of DNA pathogen found in biological samples. Trying to overcome these limitations,-Saltini Palka et al., [27] they proposed a large scale multiplex PCR (LSplex PCR) for amplification of several dozen genes nine pathogenic species, including *C. albicans*. This protocol employs a large number of pairs of oligonucleotides, 800 different to selectively amplify gene segments for the specific pathogen, and the amplicons are hybridized to a microarray probes.

This protocol increases 10 times LSplex detection sensitivity of the microarray. However, the high cost of this technology has prevented its use as a diagnostic tool.

As can be seen, the proposed molecular methods for the rapid and sensitive identification of clinical isolates of *Candida* spp., Also have limitations. Among the more important of the cost of equipment or reagents for implementation in routine clinical laboratories. Another disadvantage is that most of the methods have focused mainly on the unique identification of *C. albicans*, without taking into account the diversity of non-albicans species that can cause IQ.

Currently, it has also introduced the use of mass spectrometry (MALDI-TOF MS, for its acronym in English matrix-assisted laser desorption / ionization time-of-flight mass spectrometer) for the rapid identification of *Candida* spp., including closely related species [11,15,24]. However, although this novel method allows specific identification, isolation of the pathogen requires [12], which represents an important diagnostic use in IC disadvantage, as is common for the isolation of yeast is not achieved.

So due to the advantages and disadvantages of both the conventional methods such as molecular and proteomic, the most suitable is the diagnosis of IHD, like all infectious diseases, based on the result of conventional tests and is complemented by some of the molecular for greater accuracy in identifying the pathogen and therefore better management of patients.

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Pitted Keratolysis: Primarily a Clinical Diagnosis

MARTÍNEZ-HERRERA, Erick*†, TEJADA-GARCÍA, Diana´´, GARCÍA-REMENTERÍA, Carlos´´´ and ARENAS-GUZMÁN, Roberto´´´´

´´Universidad Nacional Autónoma de México, Departamento de Microbiología y Parasitología, Facultad de Medicina, Ciudad de México.
´´Dermatology Institute and Skin Surgery "Prof. Dr. Fernando A. Cordero C", Guatemala.
´´´Dawson Medical Group. 4805 S. Western Ave Oklahoma City, Oklahoma, USA (405) 636-1506.
´´´´General Hospital "Dr. Manuel Gea González". Chief, Section of Mycology, México City, DF.

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Abstract

Pitted keratolysis, is a bacterial skin infection that commonly affects the weight-bearing surfaces of the feet. The most frequent etiological agents are various species of Gram-positive bacteria including *Dermatophilus congolensis*, *Micrococcus sedentarius*, *Corynebacterium* spp. We report a case of pitted keratolysis in a 25-yearold male blacksmith from Guatemala. A Gram stain was performed of the lesions and showed grouped Gram positive cocci. The subsequent culture grew *Staphylococcus auricularis*

Pitted Keratolysis, Clinical Diagnosis.

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* Correspondence to Author (email: martinezerickh@gmail.com)
† Researcher contributing first author.

Pitted keratolysis is a bacterial skin infection that commonly affects the weight-bearing surfaces of the feet. It is caused by various species of Gram-positive bacteria including *Dermatophilus congolensis, Micrococcus sedentarius, Corynebacterium* spp., and others. It is characterized by crateriform depressions in the affected area and is often associated with hyperhidrosis, malodor, and sliminess of the area. We report a case of a 25-year-old blacksmith with pitted keratolysis who was successfully treated with aluminum sulfate soaks and Clindamycin 0.6% lotion BID. Pitted keratolysis (PK) is a bacterial skin infection primarily affecting the weight-bearing surfaces of the feet, including the ventral aspect of the toe, the ball of the foot, and the heel. Occasionally non-weight-bearing areas, such as the palmar surfaces of the hands may be affected, although this is rare. The disease is characterized by many circular or longitudinal, crateriform depressions in the skin surface [1]. Hyperhidrosis is the most frequently observed symptom, but malodor and sliminess of the skin are common features as well [2]. Although most cases are asymptomatic, painful plaque-like lesions can be seen. This is a disease that is most frequently observed in young men wearing protective shoes for occupational reasons which do not allow for ventilation of the feet, and thus promote a moist and warm environment for the growth of organisms [3]. We report a case of pitted keratolysis in a 25-year-old male blacksmith from Guatemala.

Case Report

A 25-year-old male blacksmith from Guatemala presented to our clinic with an 18- month history of excessive wrinkling, itching, and fetid sweating of bilateral feet. On physical exam, his feet were mal odorous and multiple punctiform depressions forming geographic plaques were appreciated on the soles of his feet.

In Figure 1 superficial erosions forming geographic plaques can be seen on the plantar surface of the foot. **In Figure 2** confluent depressed plaques with minimal scales can be seen on the plantar surface of the foot.

A Gram stain was performed of the lesions and showed grouped Gram positive cocci. The subsequent culture grew *Staphylococcus auricularis,* which was identified by the innovative microbial identification system VITEK® 60 (bioMérieux, France). A diagnosis of pitted keratolysis was established of according to the clinical triad (bromohidrosis, skin maceration and plantar keratolytic lesions) characteristics of the same and the patient received topical treatment with aluminum sulfate soaks and Clindamycin lotion 0.6% BID. The plantar lesions improved markedly after just one week of the prescribed treatment.

Comment

Pitted keratolysis was first described by Castellani in 1910 and was termed “keratoma plantaresulcatum” at the time [4]. It was later renamed “keratolysis plantare sulcatum” by Acton and Maguire in 1930 [5] as keratolysis was a more appropriate term than keratoma as keratoma implies a hypertrophic growth which is not the case with PK. It is now most commonly referred to as pitted keratolysis.

A variety of gram-positive species, including *Dermatophilus congolensis, Micrococcus sedentarius*1 *Corynebacterium* spp. [3], and others have been implicated as the causative agent of pitted keratolysis. These bacteria share a feature of producing proteinases that allow them to degrade keratin and destroy the stratum corneum thus producing the characteristic pits of PK. *D. congolensis* produces a keratinase and *M. sedentarius* produces proteinases [6].

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The malodor associated with PK is thought to be due to the production of sulfur-compound by-products, such as thiols, sulfides and thioesters [7]. Pitted keratolysis eruptions are limited to the stratum corneum layer of the skin, therefore no inflammation is observed.

The treatment of PK includes both educating the patient on preventative measures as well as various medications that have proven effective. "Various preventive measures recommended are, avoiding use of occlusive footwear, reduction of foot friction with properly fitting footwear, using absorbent cotton socks, wearing open toed sandals whenever possible, washing feet with soap or antibacterial cleanser twice a day, and avoiding sharing of footwear or towels" [8]. Topical clindamycin has also been shown to be an effective treatment for PK and is very commonly used for its bactericidal effect on Corynebacterium [9]. It also appears to be effective on other causative agents of PK, as is evidenced by our case. Aluminum sulfate or aluminum chloride soaks are used to treat the hyperhidrosis that is commonly associated with PK [1]. Other common treatments include acne medications such as topical erythromycin, oral erythromycin, and alcohol-based benzoyl peroxide applied twice a day. Mupirocin has also shown to be effective [10]. Vlahovic et. al. demonstrated the efficacy of clindamycin 1%-benzoyl peroxide 5% topical gel in the treatment of PK in their 2009 study.

This was the first known use of this combination treatment and it proved effective [11]. Botulinum toxin has also proven effective in treatment-refractory cases of PK associated with hyperhidrosis [12]. There is not a universal consensus on the treatment of PK, but in general a combination of the various treatments is often employed.

Conclusion

We report a case of a patient with pitted keratolysis who responded well to aluminum sulfate soaks and Clindamycin lotion 0.6% BID. This case should serve as a reminder to keep a clinical suspicion for pitted keratolysis and know the distinguishing characteristics as it is primarily a clinical diagnosis and which could be confused with other skin lesions as *tinea pedis*, hyperhidrosis, chronic arsenic and erythrasma.

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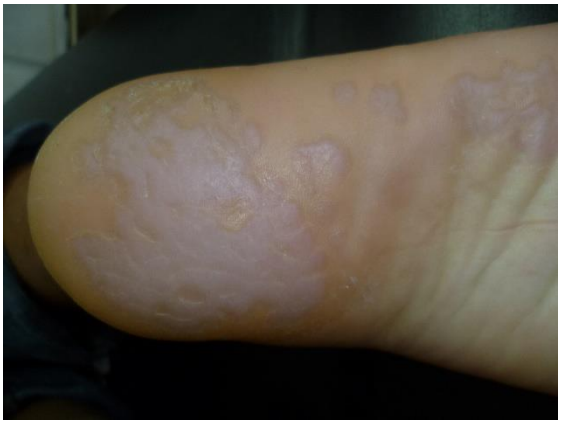

Figure 1 Forming shallow erosions geographically defects in heel

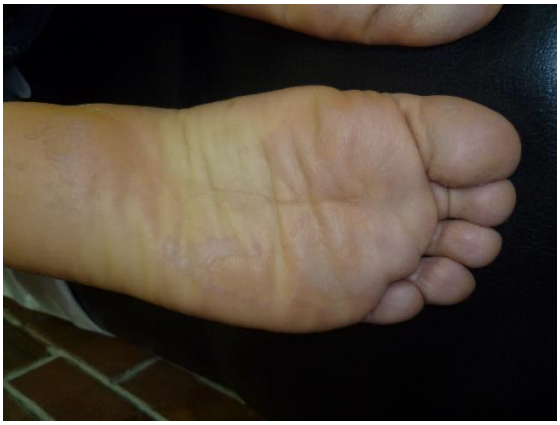

Figure 2 Forming shallow erosions geographically defects in foot and toes

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Rhinoscleroma: A case report

MARTÍNEZ-HERRERA, Erick´*†, PORRAS-LÓPEZ, Carlos´´, SÁNCHEZ-RODRÍGUEZ, José Luis´´ and ARENAS-GUZMÁN, R´´´

´Universidad Nacional Autónoma de México, Departamento de Microbiología y Parasitología, Facultad de Medicina, Ciudad de México.
´´Instituto de Dermatología y Cirugía de Piel "Prof. Dr. Fernando A. Cordero C", Unidad de Micología Médica, Ciudad de Guatemala
´´´Hospital General "Dr. Manuel Gea González", Sección de Micología, Deparamento de Dermatología, Ciudad de México

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Abstract

Rhinoscleroma is an unusual chronic and progressive infection caused by *Klebsiella rhinoscleromatis*, associated with chronic upper airways obstruction. It is endemic in Latin American countries. We present a 42 year-old female, with a five year history of a chronic nodular affection localized in nasal and infra-nasal region.

Rhinoscleroma, *Klebsiella rhinoscleromatis*, Mickulickz´s cells.

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* Correspondence to Author (email: martinezerickh@gmail.com)
† Researcher contributing first author.

Introduction

First described in 1870 by Von Hebra, rhinoscleroma is a rare progressive chronic granulomatous infection that mainly affects the airways altas.[1,2] The apparatus is initially located in the nasal passages and upper respiratory tract invade and tear and sometimes invasion traqueobronqueal.[3,4] tract produces an invasive granuloma with a marked tendency to sclerosis and obstruction subsecuente.[5-6]

It is caused by Klebsiella rhinoscleromatis, and is endemic in Central and South America. It appears in the third decade of life, predominantly in women 13: 1 and is associated with low socioeconomic status population. Its mechanism of transmission is directo4 contact.

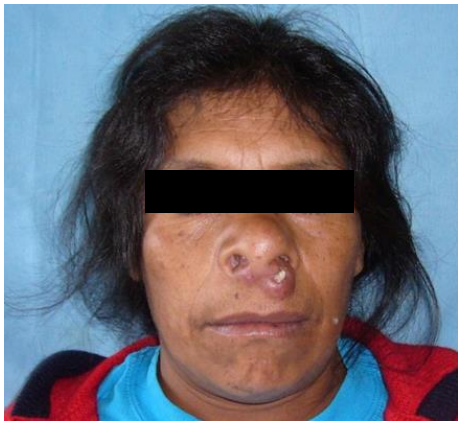
The pathogenesis is due to iron deficiency and specific fagocitosis7 deficit. The clinical picture is manifested through three stages: Stage I exudative, or catarrhal rinítico; that arise as symptoms of common cold with fetid purulent rhinorrhea, nasal obstruction unilateral or bilateral, crusting, burning and dryness of the pharynx. Granulomatous or proliferative stage II; rhinitis symptoms diminish, no infiltration and obstruction of the lower portion of the nostril, granulation tissue is exuberant, friable, crusty and induration. Stage III sclerotic; where clinical improvement, previously inflamed tissues are replaced by collagen dense, there is a process of spontaneous or after treatment leads to healing but anatomical distortion and stenosis of the affected structures during proliferación [8,9] stadiums.

Recommended treatments are based on the use of antibiotics and surgery for a long time. Good results being obtained using flouroquinolonas as ciprofloxacin or levofloxacino[10].

Surgery is needed if there is a stenotic laringotraqueal commitment to life-threatening and in the case of nasal obstruction with impaired quality of life. It must monitor the therapy as this has shown up to 76% of recurrenes[11].

Clinical case

A female patient 42-year-old from San Marcos Guatemala, treated at the Institute of Dermatology and Skin Surgery "presented Prof. Dr. Fernando A. Cordero C ", who came to present injury infranasal region of 5 years of evolution, which grows as time passes, accompanied by foul odor, and no response to previous treatment with amoxicillin. Denies history. A physical examination is: chronic dermatosis localized in nasal and infranasal region, characterized by erythematous and infiltrated lesion nodular, reaching 1/3 proximal septum, about 5 centimeters in diameter, with regular edges, defined , which in its central zone is serum and purulent crust on the surface neovascularization (Fig. 1) is located. Laboratory tests that were performed; Full, TTP and normal HIV negative test TP and hematology. Treatment was ciprofloxacin 500 mg twice daily and referred to the otolaryngology service.



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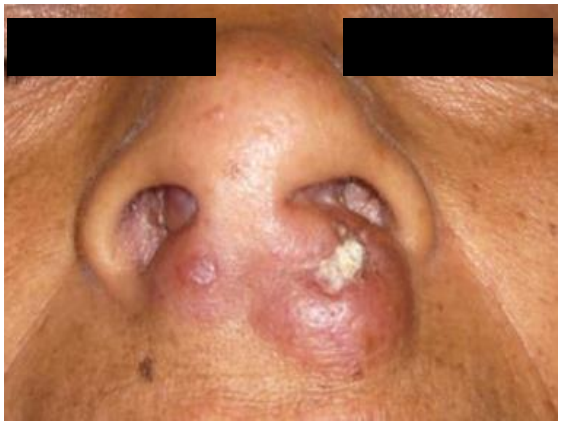
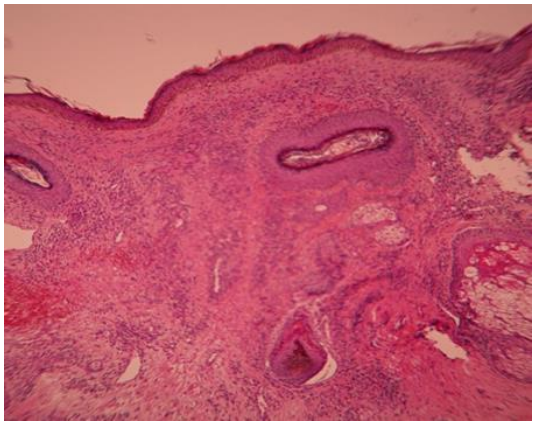


Nodular lesions caused by Klebsiella rhinoscleromatis are observed.

Figure 1 Rinoescleroderma

The secretion culture was positive for Staphylococcus aureus sensitive to β-hemolytic amoxicillin and ciprofloxacin. Two biopsies were performed in 4 mm punch. The first was macerated which was negative, and the second stained with hematoxylin and eosin showed that stratum corneum network basket and atrophic epidermis. Resurfacing to deep dermis with interstitial inflammatory infiltrate composed of plasma cells with bright eosinophilic material presence inside that corresponded to Russell bodies and Mikulickz cells, Giemsa positive (Fig. 2 and 3).



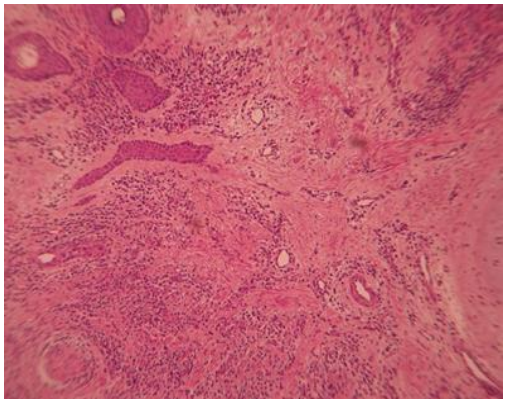
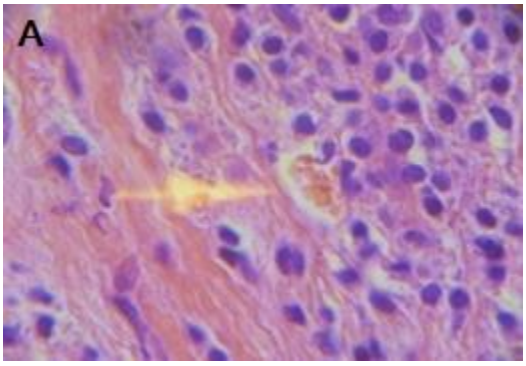


Figure 2 Biopsy inflammatory infiltrate with plasma cells (HE 40X)



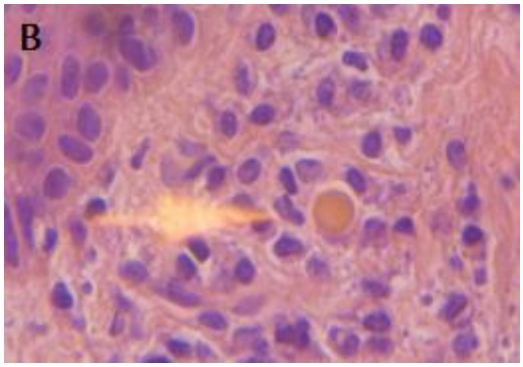


Figure 3 Mikulicz cells (A) and Russell bodies (B).

Discussion

Since its discovery in 1870 rhinoscleroma diagnosis in endemic areas, showing an upturn with fungal infections of the oropharynx due to the emergence of HIV, both in developed countries and in the process of development.[12].

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Rhinoscleroma in turn is associated with low socioeconomic status, overcrowding, malnutrition and poor hygiene [9-13]. Diagnosis is based on clinical display, histopathology, imaging findings and the visualization of the lesion by endoscopy, being useful to rule out other diseases and determine the extent of being lesiones.[14] histopalógico clinical diagnosis and in our case imagenoscopía I showing the presence of vacuolated cells containing the Gram negative bacillus, Klebsiella rhinoscleromatis encapsulated in Mickulickz called cells (Figure 3) [15].

Bacteriology in the case of rhinoscleroma has low sensitivity, around 50-60% 16, not the exception being the case where only managed to establish a co-infection with S. Areus.

In our case catarrhal presentation, the septum and inferior turbinate were the ideal places to get the sample. Ideally, the biopsy of affected areas of granulomas. Laryngeal dyspnea exist characterized by the presence of stridor, retractions, bradypnea and dysphonia (not always present) and mass occupying upper airway should always do rhinoscleroma differential diagnosis, as suggested by Villaverde N et al.[17]

In our case, a patient forty-two years is shown, which correlates with the reported epidemiology, since it is the genre where it is observed more frequently (13: 1) and also from the third decade of life, which correlates with the incubation period five years.[18]

As for treatment, it has reported excellent results flouroquinolonas and in some cases depending on the pattern of susceptibility or resistance, has also been used streptomycin and minocycline between otros.[19,20]

Given the good results with flouroquinolonas decided treat our patient with ciprofloxacin 500 mg twice daily, and referring it to otolaryngology.

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Tinea nigra: A case report in Dominican Republic

PORRAS, Carlos´*†, RODRÍGUEZ, Edita´´, CRUZ, Cecilia´´ and ISA-ISA, Rafael´´

´Institute of Dermatology and Skin Surgery "Dr. Fernando A. Cordero C " Medical Mycology Unit Guatemala City
´´Instituto Dermatológico "Prof. Dr. Huberto Bogaert" Republica Dominicana

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Abstract

Tinea nigra is a superficial mycoses, it usually is asymptomatic and generally affects palms. These lesions are seen as a macula straightedge and a dark halo at the periphery. We report a case of a 6-year-old pediatric patient from Dominican Republic, which came for a dermatological consulting to Institute of Dermatology and Skin Surgery "Dr. Huberto Bogaert" with a dark macula in the left palm. A mycological study was carried out in which it was isolated *Hortaea werneckii*, confirming the clinical diagnosis of *tinea nigra* . A good response to ketoconazole cream 2% was observed.

Tinea nigra, *Hortaea werneckii*, **dermatomycosis, Dominican Republic.**

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* Correspondence to Author (email: cfporrasl_gt@hotmail.com)
† Researcher contributing first author.

Introduction

Tinea nigra (black palmar *tinea*) is a rare superficial mycosis caused by *Hortaea werneckii*[1]. It was first described in Brazil Cerqueira (1891), who called keratomycosis palmaris[2] nigricans.

Hortaea werneckii is distributed in the tropics and subtrópicos.[3] is considered that this fungus is present in soil and decaying material. The inoculation occurs through trauma.[4] The incubation period is 2 to 7 weeks, affecting only the stratum corneum without any invasion of the deeper layers epidermis.[5] Brazil has reported traumatic inoculation through conejo.[6] bite. H. werneckii is characterized by a halophilic organism, which means it grows on substrates with high salt and low pH, besides presenting melanina.[7]

Regarding the epidemiology of *tinea nigra*, described in Southeast Asia, in Africa and in tropical and subtropical areas of America. Cases have been reported in the UK, Spain and France, and some of these cases are due to travel to areas endémicas.[8]

Among the treatments used for *tinea nigra* are topical ketoconazole, miconazole, terbinafine and butenafine accompanied by keratolytic agents, which can reduce pigmentación.[9,10,11] In some cases, it reported the resolution espontánea.[12]

Case report

Female patient of 6 years old resident of Santo Domingo, Dominican Republic, who came to see mycology department of Dermatology Institute and Skin Surgery "Prof. Dr. Hubert Bogaert "for filing a coffee stain left palm with regular boundaries, about 1.5 centimeters in diameter and asymptomatic, approximately 1 year; refers no other pathology.

The patient was treated with ketoconazole cream 2%, with complete recovery in two weeks.

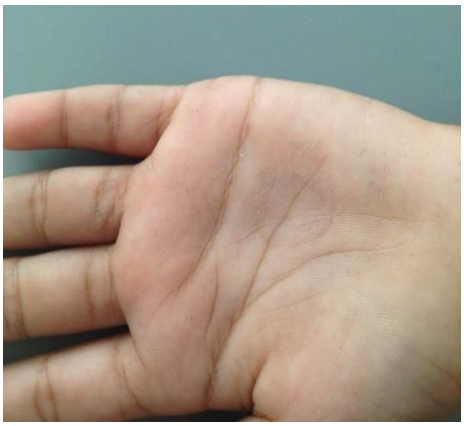

Figure 1 Left palm patient showing a lesion approximately 1.5 cm in diameter.

He proceeded to take sample of the affected area by scraping with a scalpel blade No. 15, it was observed under a microscope using potassium hydroxide (KOH) 10% finding pseudohyphae yeast structures and dark green. Cultivation was done on Sabouraud agar with chloramphenicol and cycloheximide (Mycosel) for subsequent microscopic identification**.**

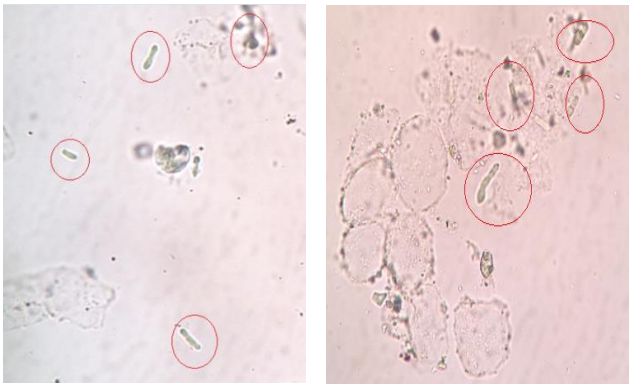

Figure 2 Yeasts with a central depression dark green

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The sample was cultured on Sabouraud agar with chloramphenicol and cycloheximide (Mycosel) at 25 ° C. After about three weeks, the growth of a colony of black color, smooth and shiny surface was observed. At the microscopic examination of the culture with lactophenol blue blastoconidia presence septum with a central dark evidenced, which were consistent with the morphology of *Hortaea werneckii*.

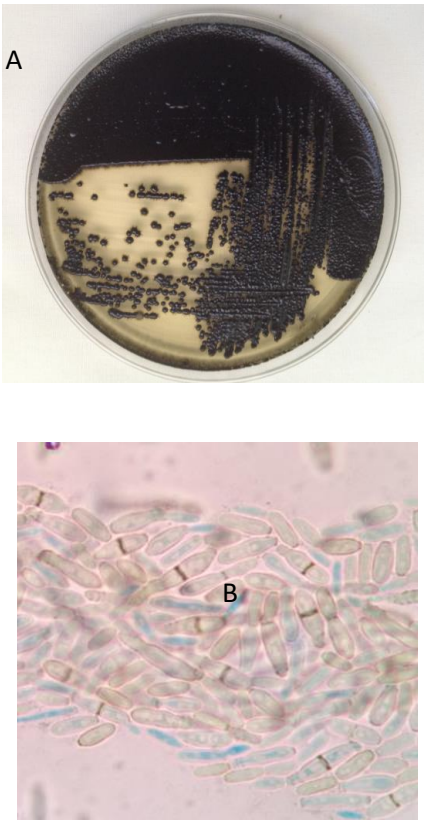

Figure 3. A) Culture of *Hortaea werneckii* agar Mycosel, B) Multiple blastoconidia with a central septum

Discussion

Tinea nigra is a rare dermatomicosis whose etiologic agent is the fungus H. dematiaceous werneckii, is a pathology usually located on the palms and soles and may be injuries from millimeters to centimeters and darker with defined limits to the affects of periphery.[13]

Preferably people who are among the first and third decades of life, 14 reported at a higher frequency Venezuela between 3-28 years, 15 as I observed in the present case. Since the lesions are asymptomatic, this problem can be underdiagnosed.[1]

From a clinical point of view, the injury was considered characteristic of this pathology, as reported in the literature; 13 however, this condition is rare in patients with dark skin, so that the case is considered atypical. Among the differential diagnoses for this pathology are melanocytic nevi, malignant melanoma, pigmentation Addison's disease, fixed pigmented erythema or exposure to químicos.[16]

There are few cases reported globally, without knowledge of the presence of the disease in Dominican Republic even if it is a tropical region with temperatures ranging between 25-35 ° C and a salt concentration is abundant.[3]

Ketoconazole cream was used 2%, with a total resolution of the lesion at 2 weeks. In some cases there has been spontaneous healing injuries.[12]

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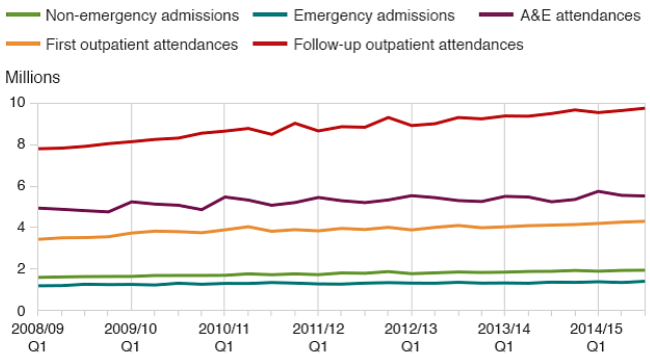

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