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Editorial

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Microneedling: An Update

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Aesthetic treatments are on the rise and according to the American Society of Plastic Surgery (ASPS), minimally invasive procedures comprise of approximately 89% of all cosmetic treatments. Microneedling is one of the new kids on the block. Though it has been in the market for a little over 10 years, it has become popular in the last 2 years.

Microneedling (MN) may be done with a handheld device which has a rolling cylinder at one end. This cylinder has tiny medical grade stainless steel needles protruding from it. This is rolled on the skin in all four directions to create multiple channels in the skin. Depth of the channels created depend on the length of the needles on the rolling device. The length of the needles ranges from 0.5 mm to 3 mm. These channels allow easy transdermal drug delivery and this makes it useful for mesotherapy and platelet rich plasma (PRP). Microinjuries to the upper dermis release numerous growth factors which cause neocollagenesis.

Microneedling is a simple, inexpensive, clinical procedure with minimal downtime. Topical anaesthesia is most commonly used. There may be erythema and oedema for 3 to 4 days post-treatment. Use of sunscreen and emollients is encouraged after treatment to prevent post-inflammatory hyperpigmentation especially in ethnic skin.

Side effects may be seen in the form of bruising, mild scabbing for a few days, hematoma on the bony areas, secondary bacterial infection, milia, tram track effect seen mainly on bony prominences and post-inflammatory hyperpigmentation.

This procedure is performed to treat acne scars, traumatic scars, striae, skin rejuvenation, dyschromias, alopecia and it is also used for transdermal drug delivery.

Microneedling should be avoided, if there is active bacterial infection, herpes labialis, molluscum contagiosum, verrucae vulgaris, acne in the area to be treated. Other contraindications are rosacea, uncontrolled diabetes, neuromuscular disease, collagen vascular disease, skin malignancy, solar keratosis and chronic skin condition like scleroderma, psoriasis and eczema.

The advantage of microneedling is that there is neither heat nor light involved reducing the chances of long standing side effects in ethnic skin. It is thus safe in skin types III-VI as there is neither any thermal damage nor is the epidermis injured.

It can be combined with other treatment modalities such as chemical peels, nonablative and fractional lasers, and fillers to enhance results.

Various types of devices are now available with the growing popularity of the technique. Enlisted below are a few devices:

1. The needles in dermarollers are mostly made of medical grade stainless steel, but some instruments have needles made of titanium or gold coating that are believed to be less traumatic and safer than the uncoated.

2. Automated rollers: Instruments with disposable heads and hence can be used for many patients. They are battery driven.

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3. Instruments with a vibration mode to reduce pain.

4. Scalp rollers: They have titanium needles unlike stainless steel rollers and are used to treat thinning hair.

5. Instruments with a light-emitting diode (LED) photon light attached, mainly used for scarring and wrinkles. However, no published data is available regarding its efficacy. The various lights available are 405 nm blue, 633 nm red, 590 nm yellow and 560 nm green.

6. Home care dermarollers: The Needle size is less than 0.15 mm in length. They are used for treatment of open pores, fine lines, cellulite, stretch marks and to reduce sebum production. They are also used for transdermal delivery of substances like lipopeptides and other antiaging products. They can be used twice a week for up to 100 times. After use, the rollers have to be cleaned in hot tap water and shaken dry.

7. Dermastamp: They are the miniature versions of the Dermaroller® (c) with diameter of 0.12 mm and needle length varying from 0.2-3 mm. They are used for localized scars (e.g., varicella scars) and wrinkles. The procedure with the derma-stamp can be performed in 2 minutes.

8. Dermapen: It is an automated microneedling device which looks like a pen. This ergonomic device makes use of disposable needles and guides to adjust needle length for fractional mechanical resurfacing. It has 9-12 needles arranged in rows. It makes use of a rechargeable battery to operate in two modes, namely, the high speed mode (700 cycles/min) and the low speed mode (412 cycles/min) in a vibrating stamp-like manner. The needles are disposable and hence it is re-usable. It is safe as the needle tips are hidden inside the guide, and more convenient to treat narrow areas such as the nose, around the eyes and lips without damaging the adjoining skin.

9. DermaFrac™: It is a newer modification of microneedling combining microdermabrasion, microneedling, simultaneous deep tissue serum infusion, and LED. It can be used for acne, uneven skin tone, fine lines and wrinkles, open pores and photo damaged skin. It is non invasive, and cost effective with less down time with individualized selection of serums for infusion.

10. Dermarollers with various types of Microneedles

a. Hollow microneedles—for insertion into the skin and infusion of drug through the MN pore created.
b. Coated microneedles—for deposition of active ingredient into the epidermis, followed by removal of the MN array.
c. Solid microneedles—for skin pretreatment, followed by the application of an active-loaded reservoir
d. Dissolving microneedles—for delivery of active compound, incorporated into the matrix of the needles, into the skin.
e. Swellable microneedles—for drug delivery through the hydrogel matrix from a drug-loaded reservoir

Microneedling like chemical peels is here to stay owing to its safety and efficacy in all skin types.

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Editorial

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Emerging Age of Opportunity for More Effective Treatments in Atopic Dermatitis

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Atopic dermatitis (AD) is a chronic inflammatory disease characterized by widespread lesions, which manifest as erythematous areas with crusting and/or scaling, oozing, excoriations, and lichenification. Additionally, pruritus/itching is one of the major symptoms of the disease which has prompted a description of AD as "the itch that rashes". Combined, these symptoms significantly impact on the quality of life (QoL) for millions of individuals worldwide. For years, AD was primarily considered a disease that affected children and adolescents, but recent epidemiological studies have highlighted the increased incidence of disease among the adult population as well.

Until recently, individuals with AD had very few options for effective treatment of their disease. Individuals were predominantly managed through the use of topically applied steroids, calcineurin inhibitors (i.e., pimecrolimus, tacrolimus), or immunosuppressive agents (i.e., cyclosporine, mycophenolate mofetil) to suppress the inflammatory response and restore skin barrier function; allergen avoidance to prevent disease exacerbation and acute flares; and antimicrobial agents to prevent secondary skin infections. In severe cases, patients received oral antihistamines and anti-anxiety medications to alleviate the insatiable itching. These treatments are relatively effective in individuals with more mild disease, but the treatment effect is short-lived and does not provide long-term relief or remission for the more moderate to severe patients. Additionally, long-term use of steroids may be associated with significant side effects which subsequently increases the anxiety of patients and may reduce patient compliance.

Recent advances in our understanding of AD have further illustrated the complex nature of the disease. Specifically, research has demonstrated that AD is a complex interaction of barrier disruption and systemic inflammation which drives disease pathogenesis. For years, it was believed that T-helper 2 (Th2)-mediated inflammation in the skin was the predominant culprit for the disruption of the skin barrier and increased risk of skin infections. Additionally, the increased Th2 signaling was responsible for the increased pruritus observed in these patients as well. More recently, genomic and proteomic approaches have shown that AD patients are more heterogeneous that initially thought. This heterogeneity has prompted further characterization of the different inflammatory axes and the corresponding subsets of AD patients. As a result of these investigations, it's now known that AD patients can have elevated Th2 inflammation as well as increased T-helper 22 (Th22)- and T-helper 17 (Th17)-mediated inflammation.

Corresponding with our increased understanding of the inflammatory axes in AD, there has been an increase in novel therapeutic approaches targeting these specific inflammatory pathways. A monoclonal antibody targeting interleukin-4 receptor alpha (IL-4Rα; dupilumab) was recently approved for the treatment of moderate to severe AD and additional monoclonal antibodies targeting IL-13, thymic stromal lymphopoietin (TSLP), IL-33, and OX-40 are currently in clinical trials as well. Similarly, crisaborale, a topical ointment that targets phosphodiesterase 4 (PDE4), was approved for the treatment of mild to moderate AD. Additional therapies are currently in clinical trials evaluating the effects of janus kinase (JAK), aryl hydrocarbon receptor (AHR), and chemoattractant receptor-homologous molecule expressed on T-helper 2 cells (CRTH2) antagonists in various severities of AD.

As our understanding of AD continues to increase, we deviate from the idea that AD is

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a cosmetic disease with severe itching to a more appropriate acceptance that AD is a systemic autoimmune disease with significant skin inflammation. As such, the emergence of new therapeutic options provides the hope that resolution and remission may soon be a reality for the millions of patients suffering from AD.

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Case Report

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Successful Treatment of Multiple Alopecia Areata With Contact Immunotherapy: Supportive Usage of Oral Antihistamine and Topical Corticosteroid

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ABSTRACT

Contact immunotherapy using diphenylcyclopropenone (DPCP) is a commonly used first-line therapeutic technique for patients with alopecia areata (AA). We present here a successful immunotherapy for AA female case with atopic diathesis, using oral antihistamine and topical corticosteroid supportively on DPCP. Courses of clinical and laboratory findings suggested that indirect effect to allergic inflammation by antihistamine drugs contributed to AA regression in our case.

KEY WORDS: Alopecia areata; Atopic dermatitis; Contact immunotherapy.

ABBREVIATIONS: DPCP: Diphenylcyclopropenone; AA: Alopecia Areata; AD: Atopic Dermatitis; UV: Ultraviolet; PUVA: Phototherapy ultraviolet radiation; UVB: Ultraviolet B.

INTRODUCTION

Alopecia areata (AA) is a most common cause of hair loss,[1] characterized by patchy, confluent, or diffuse hair loss in normal-appearing skin. Area of hair loss usually involve in the scalp and region of the beard, or even on the whole body. Together with clinical presentation of hair-loss, exclamation-mark hairs, cadaver hairs and nail pitting often render the diagnosis of AA.[1] About 16% of AA patients are reported to associate with other allergic or autoimmune diseases including atopic dermatitis (AD), vitiligo, autoimmune thyroid disease.[2,3] Helpful therapeutic options with immunosuppressive or immune-deviation strategy are suggested to clinician on these days.[1] Among them, only 2 treatments are considered to reach the level of evidence-based medicine; intralesional injection of corticosteroid and contact immunotherapy.[1,4-6] Still, there is no curative therapy for AA. On the other hand, 'modified' immunotherapy for AA such as combination therapy with contact immunotherapy and steroid pulse and contact immunotherapy without sensitization at a starting point are also reported, currently.[7]

We experienced promoted hair regrowth in refractory multiple AA after the combination therapy of contact immunotherapy using diphenylcyclopropenone (DPCP) with oral bepotastine besilate, oral hydroxyzine hydrochloride and intermitted application of topical corticosteroid.

CASE REPORT

A female case, with a history of polycystic kidney and AD from 27-years-old, experienced onset of AA when she was 40 years old. She has not responded to steroid pulse therapy for one year of intermittent (weekend) oral corticosteroid with targeted ultraviolet (UV) B phototherapy for her scalp. She visited our hospital in 2015, when she was 47-years-old. She neither responded

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to 6 months of weekend oral corticosteroid with topical phototherapy ultraviolet radiation (PUVA) for her head, nor 6 months of oral antihistamine and intralesional corticosteroids with ultraviolet B (UVB) irradiation for her body in our hospital. After tapering oral corticosteroid, we began biweekly contact immunotherapy using DPCP on 2016. Simple application of 10^{-8}10^{-7}% DPCP for 3 months aggravated her skin especially in her hairy scalp, ears, face and neck. Hairs of pariental region remained to be completely lost after 3 months of biweekly contact immunotherapy (Figure 1). However, it showed remarkable recovery not only of eczema of her body but also of hair growth after double dose of oral bepotastine besilate (40 mg/day), hydroxyzine hydrochloride (20 mg/day) and topical corticosteroid lotion were added (Figure 2). Application of topical corticosteroid was only permitted in last three days before the day of DPCP application. Changes in laboratory data were shown in Table 1.

DISCUSSION

Inducing allergic reaction by applying contact allergens to the affected skin is a principle of contact immunotherapy.[7] Among several reported prognostic factors of contact immunotherapy until today, AD is not a negative factor for DPCP.[8] We have decided to start the therapy on this case because firstly depend on patient's strong will, secondly depend on this note described above, in spite of concerned aggravation of AD eczema by excessive allergic reaction. Combinative use of oral antihistamine and topical corticosteroid, aimed to cure worsen AD skin symp-

Figure1: After 3 Months of DPCP Therapy. Pariental Area of Hair Loss of the Scalp Reached About 70% of the Scalp.

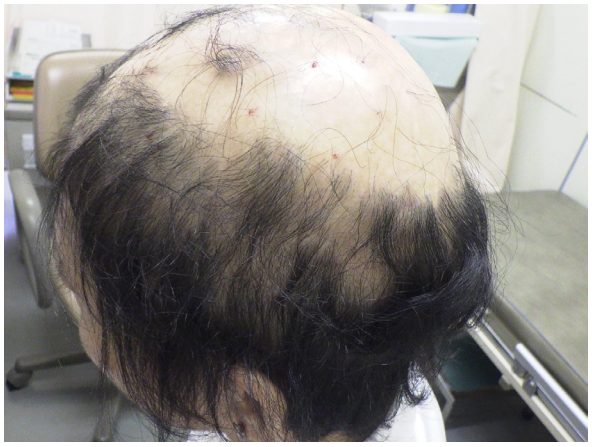

Figure 2: After 10 Months of DPCP with Oral Antihistamine and Topical Corticosteroid. AA Patch Nearly Recovered. The Combination Therapy is Being Continued Until Now.

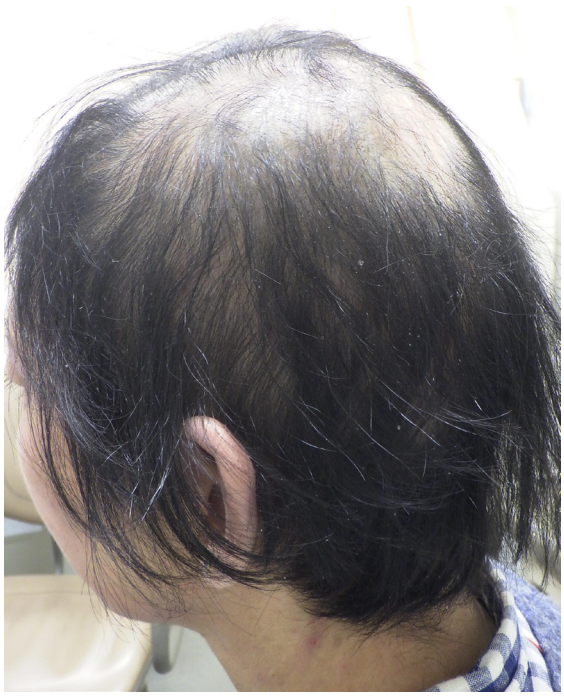

Table 1: Laboratory Data of the Year 2015-2016. Thymus and Activation-Regulated Chemokine (TARC) was Decreased whereas Serum Immunoglobulin (Ig) E and Eosinophil Level Showed no Remarkable Changes.

Date	Therapy	TARC (-450 pg/mL)	IgE (-500 IU/mL)	Eosinophil (70-440 /μL)
Jan. 2015	Weekend oral corticosteroid		517	461
Aug. 2015	UVB Antihistamine Intralesional corticosteroid Weekend oral corticosteroid	2416	651	600
Dec. 2016	DPCP Antihistamine (4 months after changing to bepotastine besilate and hydroxyzine hydrochloride) Topical corticosteroid	985	588	575

TARC: Thymus and Activation-Regulated Chemokine; DPCP: Diphenylcyclopropenone; UVB: Ultraviolet B.

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toms with this case, were casually started, though we principally stop using oral antihistamine at the beginning of contact immunotherapy in order to induct sufficient contact dermatitis by the therapy in our hospital. Supplementary effects of second-generation antihistamine such as fexofenadine and ebastine are previously reported.[9-11] Among them, Inui et al presented the effect of fexofenadine in 121 cases of contact immunotherapies on AA patients. AA patients with atopic diathesis treated with fexofenadine showed marked hair regrowth than AA patients with atopic diathesis untreated with fexofenadine.[10] Interestingly, there has been reported no difference in hair regrowth between AA patients with or without fexofenadine therapy, who had no atopic diathesis. They concluded that combination therapy of fexofenadine with contact immunotherapy is helpful option in the treatment of AA with atopic diathesis. Our case showed remarkable and sudden hair recovery after new generation antihistamine drugs and topical corticosteroid were added to simple DPCP therapy. These results bring us insights that antihistamine could have contributed to hair growth *via* indirect effect through improvement of allergic inflammation. According to the data (Table 1), thymus and activation-regulated chemokine (TARC) was decreased whereas serum immunoglobulin E level and eosinophil level were not influenced by this treatment modality. Her skin symptom are being still fluctuated and managed to control with topical corticosteroid and moisturizer as well.

We also suspect intermitted using of topical corticosteroid contributed controlling inflammatory cell infiltration and promoting recover from erosive dermatitis commonly which is common complication of contact immunotherapy. We suggest that usage of topical corticosteroid shouldn't be eliminated in contact immunotherapy on AA patients with atopic diathesis such as AD. Ohyama et al reported that there were not statistically significant difference in serum levels of IL-12 and substance P, but there were decreased infiltrating T-cells around follicles were seen pathologically in local skin with AA patchof ebastine-treated C3H/HeJ litter mice. In their report, they speculated antihistamine modulate local behaviors of mast cells in AA.[11] Our case in this report also suggests that TARC derived from keratinocytes at a local skin with AA patch directly accelerated hair fall. Among AA as multifactorial disease, we consider susceptibility to contact immunotherapy in AA with atopic diathesis may depend on severity of AA.

CONCLUSION

As far as we are aware, this is a first report that bepotastine besilate and hydroxyzine hydrochloride showed beneficial effect in an AA patient with AD undergoing contact immunotherapy. Effective and capable combinative options with contact immunotherapy such as anti-allergic inflammation drugs are required for AA patient with AD.

CONFLICTS OF INTEREST

The authors have no conflict of interest to declare.

CONSENT

The authors have received oral informed consent from the patient whose photographs are involved in the manuscript.

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Systematic Review

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Systematic Review of Oral and Topical Botanicals in Reducing Photosensitivity

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ABSTRACT

Objectives: Various treatment options are available for the management of photosensitivity. The objective of this review is to evaluate the use of botanicals rich in antioxidants for photosensitivity reduction.
Design: Embase and Ovid/MEDLINE databases were searched for clinical studies evaluating antioxidants from botanical sources in the management of photosensitivity.
Results: Of 339 citations, 10 met the inclusion criteria. Four studies evaluated *Polypodium leucotomos*, two evaluated *Camelia sinensis*, while *Hamamelis*, *Pistacia vera L.*, *Citrus sinensis* varieties Moro, Tarocco and Sanguinello, and *Capparis spinosa* were investigated in one study each. Five studies evaluated oral supplementation, four evaluated topical formulations, and one study evaluated both oral and topical antioxidants. Main results were summarized.
Conclusions: There is some evidence that antioxidants derived from botanical sources may be beneficial in reducing skin erythema and photosensitivity. However, the studies included in this review have methodological limitations and large scale randomized, placebo controlled trials are needed to further evaluate the efficacy and safety of botanical antioxidants in photosensitivity reduction.

KEY WORDS: Botanical; Plant; Antioxidant; Erythema; Photosensitivity; Ultraviolet; Ultraviolet-A (UVA); Ultraviolet-B (UVB); Photoprotection; *Polypodium leucotomos*.

ABBREVIATIONS: UV: Ultraviolet; UVA: Ultraviolet-A; UVB: Ultraviolet-B; MED: Minimal Erythema Dose; MPD: Minimal Phototoxic Dose; IP: Idiopathic Photodermatoses; PL: *Polypodium leucotomos*; PLE: Polymorphic Light Eruption; IBS: Irritable Bowel Syndrome.

INTRODUCTION

Photosensitivity, commonly referred to as sun sensitivity, is a term used to describe inflammation triggered by ultraviolet (UV) rays from the sun. The American College of Rheumatology explains that photosensitivity is a 'skin rash as a result of unusual reaction to sunlight, by patient history or physician observation.'[1] Specifically, photosensitivity is caused by an abnormal reaction to a component of the electromagnetic spectrum of sunlight and a chromophore (reactive compound) within the skin. The characteristics of photosensitivity vary with the type of photosensitivity. The presentation can manifest as macular erythema, papules, plaques, vesicles, bullae, telangiectasias, or eczematous patches usually in sun-exposed areas of the skin. The differential diagnosis of photosensitivity is large and includes genetic and metabolic diseases, photochemical sensitivity, idiopathic photosensitivity and other systemic and cutaneous diseases where photosensitivity is a part of a larger symptom complex. Aggravation of skin symptoms from sunlight exposure is a common presentation of various rheumatologic diseases.[2] There have been recent advances in the understanding of photosensitive rheumatic diseases, especially cutaneous lupus erythematosus and dermatomyositis.[3] Studies suggest mechanisms for photosensitivity include: modulation of autoantibodies, production of free radicals, cytotoxic effects, apoptosis induction, upregulation cytokines, induction of nitric oxide

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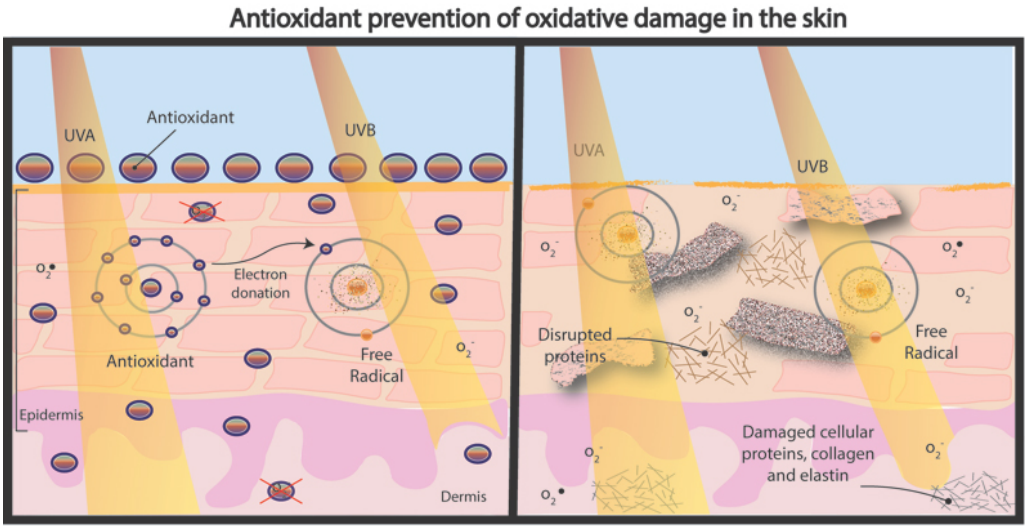
Figure 1: Plant Extract Antioxidants are a Promising Approach to Protecting Photosensitive Skin from Damaging UV Radiation. Free Radicals can Adversely Alter Lipids, Proteins, and DNA and Trigger a Number of Human Diseases. Antioxidants are Involved in the Prevention of Cellular Damage by Safely Interacting with Free Radicals and Halting Damage. The Application of External Sources of Antioxidants can Assist in Managing the Oxidative Stress.



synthase expression and ultraviolet-generated antigenic DNA.[1,4] In concert with the mechanisms discussed, some studies suggest that supplementation with antioxidants may be beneficial. Plant extract antioxidants and immune modulating mechanisms are a promising approach to protecting photosensitive skin from damaging UV radiation (Figure 1).

Current therapy for photosensitivity involves symptom specific relief, sun protection and treatment of the underlying disorder. Sun-protective measures such as sun avoidance and sunscreen are essential. If possible, any drugs or chemicals that could cause photosensitivity should be discontinued after consulting with a doctor. When a skin reaction has already developed, topical corticosteroids may be prescribed to reduce inflammation. Topical application of calcineurin inhibitor tacrolimus has also shown to be efficacious in some patients.[5] For more severe reactions, oral glucocorticoids may improve symptoms during acute exacerbations.[6] The use of steroids leads to numerous unwanted side effects such as skin thinning and bruising, weight gain, acne, and osteoporosis.[7] Given these adverse effects there is a need for safe and effective alternatives to treat photosensitivity. Our understanding of medicinal botanical extract efficacy and their mechanisms is growing, as is the demand for natural approaches for treatments. Here we review and discuss the evidence for the use of plant-derived components and their mechanisms in reducing photosensitivity.

MATERIALS AND METHODS

Search Strategy

Embase and Ovid/MEDLINE databases were searched in January 2017 for clinical studies examining the effects of plant based antioxidants on photosensitivity. A controlled vocabulary was used in the search, as outlined in Appendix Table A1.

Eligibility Criteria

Clinical studies that used antioxidants from botanical sources were included. Additionally, studies focusing on photosensitivity with outcome measures evaluating the change in skin erythema were included. Reports that did not describe human clinical studies (such as reviews, abstracts, and editorials), as well as those that did not study photosensitivity or did not evaluate a plant-derived antioxidant were excluded.

RESULTS AND DISCUSSION

Our search yielded 339 articles of which ten manuscripts met the inclusion criteria (Figure 2). The botanical sources of antioxidants included *Polypodium leucotomos* (tropical fern; four reports), *Camelia sinensis* (green tea, two reports), *Hamamelis* (witch hazel, one report), *Pistacia vera* L. (pistachio, one report), *Citrus sinensis* varieties Moro, Tarocco and Sanguinello (red orange, one report), and *Capparis spinosa* (one report) (Table 1). Five studies evaluated antioxidants in oral formulation, four studies evaluated topical formulations, and one study evaluated both oral and topical antioxidants. Main results are summarized in Table 2.

Polipodium leucotomos

Polypodium leucotomos (PL) is a tropical fern plant native to Central America. The benefits of PL have been reported in the treatment and prevention of skin conditions including psoriasis, sunburn, and polymorphic light eruption.[8] PL possesses potent antioxidant, photoprotective, and immune modulatory activities.[9]

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Table 1: Botanicals Used for Photosensitivity Reduction.

Scientific name	Common name
Polypodium leucotomos[10-13]	Tropical fern
Camelia sinensis[16,17]	Green tea
Hamamelis virginiana L.[21]	Witch hazel
Pistacia vera L.[23]	Pistachio
Citrus sinensis[24]	Red orange
Capparis spinosa L.[27]	Caper bush

Gonzalez et al[10] conducted a non-blinded randomized controlled trial to investigate the photoprotective activity of PL when applied topically or consumed orally among 21 healthy subjects aged 18-46 years (8 men, 13 women), with skin type III and IV. The subjects were either non-sensitized or psoralen-sensitized [oral 8-methoxy psoralen (8-MOP) or 5-methoxy psoralen (5-MOP)] and then randomized to receive topical PL (10%, 25%, 50%) or oral PL (1080 mg). Topical treatment was applied to subjects' backs once, in the amount of 2 µL/cm^2, at least 15-30 minutes prior to sun exposure. In the group receiving oral PL, a total of 720 mg PL in the form of capsules was consumed one day prior to sun exposure and 360 mg PL three hours prior to sun exposure. The outcome measures were minimal erythema dose (MED) and minimal phototoxic dose (MPD) before and after PL administration. MED is the minimal UV dose that leads to sunburn; MPD is the minimal ultraviolet-A (UVA) dose that leads to phototoxic reaction in the skin. The MED and MPD outcomes were evaluated visually and reported as minutes of sun exposure until visible skin reaction occurred. Subjective determination of MED was done 20-24 hours post-exposure to solar radiation; MPD was determined 48-72 hours post-exposure. The following criteria was used for evaluating erythema reaction: 0=none, ±= trace, +=pink, ++=pink red without edema, +++=strong red with edema, ++++=violaceous with painful edema. MED was reported as minutes of solar exposure until there was visible erythema; similarly, MPD values were reported as minutes of sun exposure until phototoxic reaction. The results showed that both topical and oral PL provided skin photoprotection. Specifically, PL significantly increased MED (p<0.001) and MPD (p<0.001). Oral PL provided better photoprotection than 10% PL topical (MED: 98±15.4 and 80±0, respectively; p<0.05). No adverse effects were reported.

Although, this study shows that PL in topical or oral formulation may provide photoprotective benefits, while not causing side effects, the results should be interpreted with cau-

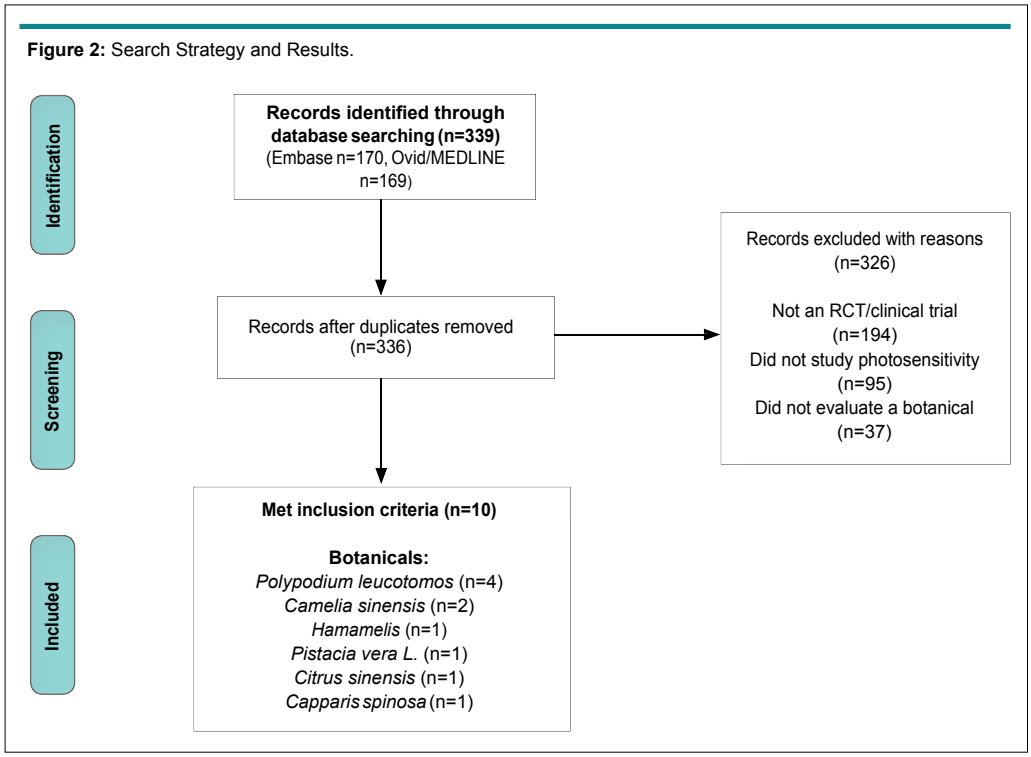

Figure 2: Search Strategy and Results.

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Table 2: Summary of Clinical Studies.

Author	Intervention	Study design	Comparison	Subjects	Affected region	Period of treatment	Outcome measure	Major results
Gonzalez et al[10]	*P. leucotomos* (PL) topical (10%, 25%, 50%)	Randomized trial	Oral PL (1080 mg daily) Sunscreen SPF 15 Control (no treatment)	N=21 Age 18-46 Skin type III and IV Subjects untreated or treated with oral psoralens [oral 8-methoxy psoralen (8-MOP) or 5-methoxy psoralen (5-MOP)]	Back	Topical–one time application 15-30 minutes before sun exposure Oral PL–one time consumption (720 mg day before sun exposure, 360mg three hours before sun exposure)	MED[a] MPD[b]	PL significantly increased MED (p<0.001) and MPD (p<0.001)
Caccialanza et al[12]	*P. leucotomos* (PL) extract 480mg daily for 15	Single group	None	N=28 subjects Age 21-68 with idiopathic photodermatoses	Sun exposed body areas	For 15 days prior to sun exposure	% improvement in skin condition[c]	80% had improvement in skin condition after PL consumption (p<0.05)
Caccialanza et al[11]	*P. leucotomos* (PL) extract 480mg daily for 15	Single group	None	N=57 Age 21-74 With idiopathic photodermatoses (IP)	Sun exposed body areas	For 15 days prior to sun exposure	% improvement in skin condition	74% had improvement in skin condition after PL consumption (p<0.05)
Tanew et al[13]	*P. leucotomos* (PL) extract 720 to 1200 mg daily for 2 weeks	Open, uncontrolled bicenter study	None	N=35 Skin type III, IV With polymorphic light eruption (PLE) Age not specified	Extensor surface of upper arms; alternatively, upper back	3 weeks	Photoprovocation of PLE lesions	Threshold for induction of PLE lesions increased significantly (p<0.05)
Farrar et al[16]	*Camelia sinensis* 1080mg daily for 12 weeks	RCT	Placebo capsules	N=50 Age 18-65	Buttocks	12 weeks	MED	MED not different between green tea and placebo group (p=0.47) MED not different at baseline vs post-green tea supplementation (p=0.17)
Li et al[17]	*Camelia sinensis* extract 2-5% topical	Single group, intra patient	None	N=20 Skin type III, IV Age not specified	Dorsal skin	6 days	Erythema index measured by chromamtery	2% and 3% green tea extracts were most protective from erythema
Hughes-Formella et al[21]	*Hamamelis (witch hazel)* aftersun lotions (distillate 1, 2, 3 each from different supplier)	Double blind, controlled trial	Vehicle 1 and 2 Dimethindene maleate 0.1% topical Hydrocorticone lotion 0.1%, 0.25%, 1% Control (no topical)	N=41 Age 19-50	Back	48 hours	Erythema suppression (assessed by visual inspection and chromametry)	Hamamelis lotions led to maximal erythema suppression at 72 hours (which is 36% that of hydrocortisone response at 1.2 MED, 66% at 1.4 MED, 56% at 1.7 MED)
Martorana et al[23]	*Pistacia vera L.* extracts TP–extract from pistachio skins SP–extract from decorticated seeds	Single group, intra patient	Tocopheryl acetate (TOC) Vehicle Control (no treatment)	N=12 Age 25-35	Ventral surface of each forearm	One time application	Percent induced erythema (PIE) (monitored by reflectance spectrophotometry)	PIE for TP, SP, and TOC were 66.8%, 33.2%, and 22.6% respectively TP formulation was significantly more protective than SP formulation (p<0.05) Difference between SP and TOC formulations was not significant (p>0.05)

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Puglia et al[24]	*Citrus sinensis* varieties Moro, Tarocco and Sanguinello (red orange) extract 100mg daily for 15 days	Single group, intra patient	None	N=20 Age 26-47 Skin type II, III	Ventral surface of each forearm	15 days	Erythema (monitored by reflectance spectrophotom-etry)	40% reduction in UV induced erythema compared to baseline
Bonina et al[27]	2% Lyophi-lized extract of *Capparis spinosa* (LECS)	Single group, intra-patient	Tocopheryl acetate (TOC) gel Control gel	N=6 Age 25-35	Ventral sur-face of each forearm	One time application	Percentage of erythema inhibition (PIE) (monitored by reflectance spectrophotom-etry)	60% PIE by LECS *vs.* 22% PIE by TOC gel (p<0.01)

[a]MED: minimal erythema dose (minimal UV dose that produces visible erythema)
[b]MPD: minimal phototoxic dose
[c]Normalization – no symptoms after UV exposure; clear improvement; slight improvement; no improvement
RCT: Randomized, controlled trial.

tion. First, subjects only received a one-time treatment with topical or oral PL. Long-term topical application, oral consumption, and potential side effects were not evaluated. Additionally, the non-sensitized group consisted mostly of women (1 man, 12 women) aged 18-46 years, while psoralen-sensitized group consisted mostly of men (7 men, 1 woman) aged 18-21 years. The small sample size and unequal gender distribution in the two groups are limitations of this study. Combined with the relatively narrow age range, the results of the study may not be generalizable. Lastly, no blinding was implemented in the study. Double-blinded-randomized controlled studies in healthy subjects, as well as those with skin disorders, are needed to further investigate the long-term effects and safety profile of topical and oral PL.

Idiopathic photodermatoses (IP) are a group of skin disorders in which skin reacts abnormally to sunlight.[11] The most common IP are polymorphic light eruption (PLE), actinic prurigo, chronic actinic dermatitis, and solar urticaria.[11] Caccialanza et al[11] conducted an uncontrolled, single arm clinical study to investigate the photoprotective activity of PL extract in 57 subjects (12 men, 45 women) with IP (PLE and solar urticaria) aged 21-74 years. Subjects consumed 480 mg PL extract daily for 15 days before UV exposure. The outcome was assessed by clinical evaluation and subjective assessment of improvement. Overall, 74% found it beneficial to consume PL extract during exposure to sunlight, which was significant at p<0.05. There were no reported adverse effects. Similarly, in another uncontrolled single arm study, Caccialanza et al[12] evaluated whether oral PL extract would provide photoprotection in 28 subjects (9 men, 19 women) with IP (PLE and solar urticaria), aged 21 to 68 years who consumed 480 mg PL extract daily for 15 days. Overall, 80% found it beneficial to consume oral PL extract during the summer (p<0.05). One subject with irritable bowel syndrome (IBS) stopped treatment due to worsening of symptoms. No other adverse effects were reported. The limitations of both studies include small sample size and no placebo control. Although both studies included a wide age range, the small sample size and lack of equal gender distribution make the results not generalizable to broader population. The lack of a placebo control prevents differentiation between the effects of the PL compared to the natural phenomenon of "hardening" that occurs with PLE and may account for the improvement in symptoms. Further controlled studies with a larger sample size and a placebo control are needed to investigate the benefits and potential side effects of PL extract supplementation.

Tanew et al[13] investigated whether PL extract would prevent or delay photoinduction of polymorphic light eruption (PLE) lesions by UV radiation. A total of 35 subjects of skin type III and IV and with PLE participated in an open, uncontrolled bicenter study. Age range and gender of study subjects were not specified. For one week, PLE lesions were induced *via* UVA and ultraviolet-B (UVB) photoprovocation, after which subjects initiated PL extract supplementation for three weeks (720 to 1200 mg daily, according to body weight). Subjects returned for second photoprovocation during week three of PL supplementation. Outcome measure was reported as the number of UVA and UVB exposures needed to induce PLE lesions before and after treatment. The results showed that the number of UVA exposures required to induce PLE increased significantly after the two-week PL extract supplementation (1.95±1.07 to 2.62±1.02, p<0.01). Additionally, the number of required UVB exposures also increased significantly after oral PL supplementation (2.38±1.19 to 2.92±0.95, p<0.05). There were no reported side effects and tolerance of PL extract was excellent. The limitations of this study include the open, uncontrolled study design. Like previously described studies, this study allowed for early UV exposure, which can lead to hardening of the skin and may account for the changes noted in the study. Age range and gender of study subjects were not specified; therefore, it would be difficult to make generalizations based on the results of this study. Further blinded, controlled studies are needed to investigate the efficacy, optimum dosage, and duration of treatment with PL extract.

Green Tea

Camelia sinensis is the plant that gives rise to a variety of teas, including green tea. Green tea contains polyphenols, which are naturally occurring compounds known for their antioxidant activity, including prevention of oxidative damage in the skin

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induced by UV radiation.[14] Catechins are polyphenolic compounds that are found in green tea, of which epigallocatechin-3-gallate (EGCG) is the most abundant and is thought to provide skin protection.[15]

Farrar et al[16] conducted a randomized, placebo-controlled trial using systemic green tea to evaluate its effects on minimal erythema dose (MED). MED is the lowest UV dose that produces visually detectable skin erythema. Fifty subjects aged 18-65 years ("nearly all females")[16] were randomly assigned to consume 1080 mg green tea catechins daily or placebo capsules. At baseline and 12 weeks post-supplementation, buttock skin was exposed to UV radiation and MED was recorded. After supplementation, the difference in MED between green tea and placebo group was not significant (p=0.47). Additionally, the difference between MED at baseline and post green tea supplementation was not significant (p=0.17). The study failed to demonstrate that oral supplementation with green tea catechins protects from UV induced erythema. The limitation of this study is that it did not compare the effects of green tea supplementation at different dosages. Additionally, the study included mostly white female subjects, thus results cannot be generalized and may raise the possibility of sex-specific effect. Future studies are needed to compare how different dosages affect the efficacy and adverse effects of green tea.

Li et al[17] conducted a single group controlled trial among 20 Chinese women (age not specified) to investigate the efficacy of 2-5% green tea extract for skin protection from UV induced erythema. Green tea extracts were applied to skin before and after UV irradiation (6 days total). The erythema intensity was measured by chromametry. The results showed that green tea extracts protected the skin from UV damage, with 2% and 3% green tea extract being the most effective. The study showed promise for the use of green tea extract in sunscreens and other topical formulations for skin photoprotection. The authors found that higher concentration was not protective and hypothesized that higher concentrations of green tea extracts may lead to cutaneous irritation and make the skin more sensitive. More studies are needed to assess how the concentration of green tea may relate to cutaneous irritation before the cost-benefit assessment can be made. The limitation of this study is that it only included a small sample of Chinese women, making generalizations difficult.

Hamamelis

Hamamelis virginiana L. (Hamamelidaceae) is a medicinal plant commonly known as witch hazel.[18] It has been used in the treatment of various conditions including rash, sunburn, swelling, inflammation, erythema, eczema, rheumatism, and tumors.[19] Hamamelis has anti-inflammatory, astringent, hydrating, and barrier stabilizing properties, which make it beneficial in the treatment of skin conditions.[20]

A double-blind study of 41 subjects (9 men, 32 women) aged 19-50 years, was conducted in order to optimize the development of hamamelis topical for after sun use.[21] Three lotions containing 10% hamamelis distillates from different suppliers were compared to 2 hamamelis free vehicles, 0.1% dimethindene maleate gel, 1% hydrocortisone cream, 0.25% hydrocortisone lotion, and control (no topical). Topicals were applied to the skin for 48 hours after skin irradiation with three UV doses (1.2 MED, 1.4 MED, 1.7 MED). The hamamelis topicals resulted in maximal erythema suppression at 72 hours which was 36% that of hydrocortisone response at 1.2 MED, 66% at 1.4 MED, and 56% at 1.7 MED. Overall, the study showed that the three hamamelis lotions exerted anti-inflammatory effects, and one of the distillates appeared more effective than the others although differences among the 3 distillates was not large. Although hamamelis topical formulations seem to be beneficial, all formulations were less effective than 1% hydrocortisone. Further, studies are needed to elicit the advantage of hamamelis formulations compared to currently available therapies for photosensitivity. As has been the case in other studies reviewed here, this study enrolled mostly women.

Pistachio

Pistachio nuts are thought to have high antioxidant potential.[22] Both the pistachio seeds and skins contain bioactive compounds which, when taken orally or applied topically, may protect human skin from damaging effects of UV radiation.

Martorana et al[23] investigated the antioxidant properties of two polyphenol-rich extracts from skins (TP) and decorticated seeds (SP) of Bronte pistachios. The results showed that both TP and SP had high levels of phenolic compounds, but TP had 10 times more phenols than SP. It was concluded that TP extract had higher antioxidant activity than SP. Due to their antioxidants properties, TP and SP extracts were investigated further for their ability to ameliorate skin erythema induced by acute UVB irradiation in 12 healthy subjects aged 25-35 years (gender not specified). The skin of both forearms was exposed to UVB radiation, and immediately treated with topical formulations containing SP, TP, TOC (tocopheryl acetate), or vehicle (blank formulation). Control site received no treatment. Induced erythema was monitored for 58 hours by reflectance spectrophotometry. Percentage inhibition of UVB-induced erythema (PIE) was calculated for each formulation in order to better compare their efficacy. The PIE values for TP, SP, and TOC formulations were 66.8%, 33.2%, and 22.6% respectively. The TP formulation was significantly more protective than SP formulation (p<0.05), while the difference between SP and TOC formulations was not significant (p>0.05). Overall, the results showed that both TP and SP extracts were protective against UVB induced skin damage and inflammation. The skin from pistachio nuts is typically industrially removed and becomes waste. This study points to the potential benefits of using this low cost ingredient in cosmetics for photoprotective applications. The limitation of this study is its small sample size and that it was only conducted on volunteers with healthy skin. Further studies investigating the

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protective effects of TP and SP extracts in individuals with photodermatoses may be beneficial.

Red Orange Extract

The red orange extract (ROE) comes from blood oranges (*Citrus sinensis* varieties Moro, Tarocco, Sanguinello)[24], which are a type of fruit with blood colored flesh. The extract possesses antioxidant properties and may protect the skin from UV damage.[25] The antioxidant and photoprotective properties of ROE are thought to be due to its content of phenolic compounds including anthocyanins, flavones, and hydroxycinnamic acids.[25]

Puglia et al[24] investigated the protective effects of ROE on UV induced skin erythema. Twenty healthy, Caucasian subjects aged 26-47 years (gender not stated) of skin types II and III were enrolled in the study. Two sites on ventral forearms were irradiated with UVB to induce skin erythema and the erythema was monitored for 48 hours by reflectance spectrophotometery. After a three-week rest period, the same subjects consumed ROE capsules in the amount of 100 mg daily for 15 days. At the end of supplementation period, the skin on ventral forearms was again irradiated and skin erythema monitored for 48 hours. After 15 days of oral supplementation with ROE there was a 40% mean reduction in UV-induced skin erythema. This study points to ROE may be a good candidate as an ingredient in skin products aimed at photoprotection. The limitation of this study is small sample size, lack of gender characterization, and that it did not report if the subjects experienced adverse effects from the supplement. Information on side effects would help in weighing the risks and benefits of such treatment.

Capparis spinosa L.

Capparis spinosa L. (CS) is a small shrub that is found in the Mediterranean and some parts of West and Central Asia.[26] It has been used in traditional medicine for the treatment of various diseases. It possesses antioxidant, anti-inflammatory, antimicrobial, antiviral, and immunomodulatory properties.[26]

Bonina et al[27] conducted an *in vitro* study showing that CS does possess antioxidant properties. They then evaluated the ability of topically applied lyophilized extract of CS (LECS) to reduce UVB induced skin erythema in six healthy subjects of both sexes, aged 25-35 years. The 2% LECS topical was compared with tocopheryl acetate (TOC; an antioxidant used in cosmetic formulations) and a control. The skin was irradiated with UV light and then the topicals were applied and remained on the skin for 3 hours, after which they were washed off and skin dried. The induced erythema was monitored for 58 hours using reflectance spectrophotometry and percentage of erythema inhibition (PIE) was determined. The PIE was approximately 60% for LECS and 22% for TOC gel formulations. These findings are not surprising considering that LECS is rich in flavonols, which have a rich antioxidant and anti-inflammatory profile. The limitation of this study is a small sample size. Additionally, the study did not provide the subject gender breakdown and it only enrolled individuals within a narrow age range of 25-35 years. Further studies with larger sample size and perhaps various LECS concentrations could help further determine the benefits of this antioxidant rich topical for skin photoprotection.

CONCLUSION

Overall, there is some evidence suggesting that botanical therapies rich in antioxidants may be beneficial in reducing skin erythema and photosensitivity when used topically or orally. Botanicals that are known to be rich sources of antioxidants include *Polypodium leucotomos* (tropical fern), *Camelia sinensis* (green tea), red orange, Hamamelis (witch hazel), pistachio, and *Capparis spinosa*, as reviewed here. It is important for clinicians to be aware of such studies evaluating plant sources of antioxidants as they could be used as adjunctive supplements in skincare. Our search produced a limited number of studies evaluating botanical antioxidants in reducing photosensitivity. Many of the studies had a small sample size and enrolled mostly women; therefore, it is difficult to generalize the results. Further, clinical studies involving larger sample sizes are needed to assess the use of botanically derived antioxidants in reducing photosensitivity. Regardless of the findings presented here, it is important that all individuals, including those with photosensitive skin, follow sun protective measures as their first line of protection, including the use of sun protective clothing, broad spectrum sunscreens, and limit their sun exposure. Supplements should only be used as adjunctive approaches and should not replace good sun protection hygiene.

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AUTHOR CONTRIBUTION

Suzana Saric and Ashley K. Clark wrote the first draft of this manuscript; Raja K. Sivamani performed critical editing and provided overall oversight for the review.

CONFLICTS OF INTEREST

The authors declare no conflict of interest. Dr. Sivamani serves as a scientific advisor for Dermveda.

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Appendix

Table A1: Search Strategies used on January 28, 2017 in Embase and Ovid Databases.

Database	Search syntax
Embase	1. 'photosensitivity disorder'/exp OR 'photosensitivity'/exp OR 'photodermatosis'/exp OR 'skin protection'/exp OR 'sunscreen'/exp 2. 'plant extract'/exp/dd_dt OR 'plant extract' OR 'herbaceous agent'/exp 3. 'antioxidant'/exp 4. 'phytotherapy'/exp OR 'chinese medicine'/exp OR 'dietary supplement'/exp 5. ('plant extract'/exp/dd_dt OR 'plant extract' OR 'herbaceous agent'/exp) OR ('phytotherapy'/exp OR 'chinese medicine'/exp OR 'dietary supplement'/exp) 6. 'antioxidant'/exp AND (('plant extract'/exp/dd_dt OR 'plant extract' OR 'herbaceous agent'/exp) OR ('phyto-therapy'/exp OR 'chinese medicine'/exp OR 'dietary supplement'/exp)) 7. ('photosensitivity disorder'/exp OR 'photosensitivity'/exp OR 'photodermatosis'/exp OR 'skin protection'/exp OR 'sunscreen'/exp) AND ('antioxidant'/exp AND (('plant extract'/exp/dd_dt OR 'plant extract' OR 'herbaceous agent'/exp) OR ('phytotherapy'/exp OR 'chinese medicine'/exp OR 'dietary supplement'/exp))) 8. ('photosensitivity disorder'/exp OR 'photosensitivity'/exp OR 'photodermatosis'/exp OR 'skin protection'/exp OR 'sunscreen'/exp) AND ('antioxidant'/exp AND (('plant extract'/exp/dd_dt OR 'plant extract' OR 'herbaceous agent'/exp) OR ('phytotherapy'/exp OR 'chinese medicine'/exp OR 'dietary supplement'/exp))) AND [humans]/lim AND [english]/lim
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Research

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Clinical and Epidemiological Aspect of Black African Adult Women With Facial Dermatosis

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ABSTRACT

Objective: To describe the clinical and epidemiological aspects of black African adult women affected by facial dermatosis.
Materials and Methods: It was a descriptive and retrospective study conducted at the Dermato-venereology Department of the Teaching Hospital of Treichville. It was conducted over a period of 5 years from January 2010 to December 2014. This study concerned women aged over 18 years who have attended consultation for any facial dermatosis and had a diagnosis after a paraclinical and/or clinical examination. Have not been taken into account in the study, all Caucasian women and all other women with uncompleted records which do not contain all the epidemiological and clinical data required for the study. Data were entered through Epi Info™ versions 3.5.1 software and analysed with Excel 2013 software.
Results: During the 5 years, we registered 7,898 patients over 18-years-old, 1,192 of these were affected by facial dermatosis that is 15.09%. The mean age was 37.7 ranging from 18 to 89-years-old. There were at least two facial dermatosis in 2.51% cases. The pigmentary disorders occurred mostly in 24.7% followed by acne group and seborrheic dermatitis (19.5%). The first three facial dermatosis of women were exogenous ochronosis, seborrheic dermatitis and lichen planus respectively 16.7%, 10.7% and 10%.
Conclusion: Pigmentary disorders due to depigmentation practices are predominant. Ochronosis exogenous, seborrheic dermatitis and acne are the most frequent.

KEY WORDS: Adult; Dermatitis; Face; Women.

INTRODUCTION

The face is the main interface of interhuman relations. Therefore, the facial dermatosis in their apparent character caused most often aesthetical damage and secondary psychic suffering in their apparent character. This psychic suffering are more intense in adult women whose face constitutes an important element of their charm and their seduction. Black African women are also concerned. In this context, it is important to identify all facial dermatosis in adult women in order to elaborate an effective treatment of these apparent pathologies. This study aimed at identifying all dermatosis occurring on the face of black African adult women specially. Specifically, this study aimed at describing the epidemiological and clinical characteristics of facial dermatosis of black adult woman (Figure 1).[1]

MATERIALS AND METHODS

Our study was conducted in the Dermatology and Venereology Department of the Teaching Hospital of Treichville, National Reference Centre for Skin Disease in Ivory Coast. This was

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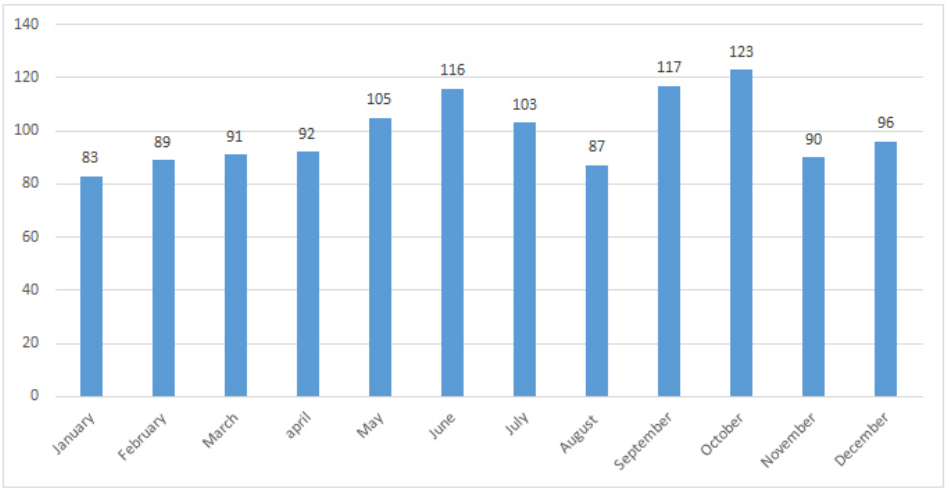
Figure 1: Evolution of Facial Dermatosis of the Black African Adult Woman in the Months of the Year.



a descriptive, retrospective study conducted over a period of 5 years, from January 2010 to December 2014. Medical records of outpatients were received during the period of the study. All medical records of women, older than 18, consulted with any facial dermatosis revealed by a clinical and/or paraclinical examination, were surveyed. Have not been included are all Caucasian women and women with uncompleted medical records which lacks all epidemiological and clinical data required for the study. Socio-demographic and diagnostic data of patients were identified on pre-established survey sheet. Data collection was done using Epi Info™ versions 3.5.1 software. Data were analysed with Excel version 2013 software.

RESULTS

Epidemiological Aspects

We identified over the period of 5 years 7,898 patients over 18-years-old, of these, 1,192 showed some facial dermatosis that is 15.09%. The mean age was 37.7 ranging from 18 to 89 years old. Women without monthly income were the most numerous with 22.4%, followed by pupils and students and women without official occupation 20.7% and 20.4% respectively. Most of the patients were from Abidjan and its suburbs in 86.2%. During the years, in June and October, facial dermatosis met two peaks rate of incidence at least two facial dermatosis in 2.51% cases (Table 1).

Clinical Aspects of Facial Dermatosis

Pigmentary disorders were most frequent 24.1% followed by acne group and seborrheic dermatitis in (19.5%) and endogenous inflammatory dermatosis (11.2%). The first three women facial dermatosis were as follow exogenous ochronosis, seborrheic dermatitis and lichen planus 16.7%, 10.7% and 10% respectively (Table 2).

DISCUSSION

Facial dermatosis affect the quality of life (QoL), are sometimes followed by some important psychic disorders and inspired most often an immediate consultation of the dermatologist. They are numerous and frequently lead to medical consultation.[2-4] This prevalence is related to race, sex and age. In literature, it is rare to find studies compiling all the facial dermatosis. The originality of this study reside in the overriding role played by adult women face for her psycho-social equilibrium. This study is therefore a compass for all dermatologists interested in black women from Africa. Nevertheless, some authors were interested in some dermatosis which may affect adult woman generally[2,5,6] and in particular black African's face.[7,8] The prevalence is relatively low, and it may be justified by the pauperization and the fact that some patients preferred to consult a beautician for any facial care. The average age of our patients is 37.7. It reflects the demography of sub-saharian African population in general and particularly in Ivory Coast. Actually, the census of the Ivorian population in 2014 showed that 77.7 % were youth under 35 years old that is just over 3 in 4 people.[9] These young girls are mostly with limited financial resources as revealed in our study, a prevalence of 22.4% of women without monthly income and 20.7% of pupils and students. This low-economic level may lead some women to use folk's African medicine and patent medicines which are relatively less expensive.[10,11] Two peaks rate of incidence of facial dermatosis were found during years, in June (116 cases) and (123) cases in October. These two months open the door to two main periods of the life of the young girl during years. Indeed, in June they are about to go on school holidays for students and annual holidays for workers whereas in October they are getting ready for the feast of the end of the year. We notice during these great periods that there is a particular care for the physical appearance. The period preceding these events allow them to look for good results of the treatment undertaken.

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Table 1: Distribution of Dermatosis Groups of Face of African Adult Woman by Major Dermatosis.

Dermatosis groups of face	Major dermatosis/frequency	Frequency of dermatosis groups	Percentage of dermatosis groups
Pigmentary disorder	Ochronosis/199	287	24.1%
	Melasma/42		
	Hyperpigmentation after Inflammatory/39		
Acne and seborrheic Dermatitis	Acne/104	232	19.5%
	Seborrheic Dermatitis/128		
Exogènes dermatosis	Eczema/119	159	13.3%
	Irritant dermatitis/40		
Drug eruption	toxic epidermal necrolysis/98	132	11.1%
	Fixed drug eruption/21		
	Maculopapular rash /13		
Tumoral dermatosis	Dermatosis papulosa nigra/45	131	11.0%
	Sebaceous cyst/25		
	Nevus/13		
Autoimmune disease	discoid Lupus/60	91	7.6%
	Pemphigus/13		
	Dermatomyositis/7		
Inflammatory endogenous dermatosis	Lichen planus/48	63	05.3%
	Xanthélasma/9		
	Sarcoidosis/6		
Infectious Dermatosis	Zona/20	56	4.7%
	Dermatophyte/11		
	superficial cutaneous bacterial infection/7		
Various dermatosis	peri oral Dermatitis/8	41	3.4%
	post traumatic Scar/7		
	Vitiligo/5		
Total		1192	100%

Table 2: Distribution of the Face Dermatosis of African Adult Woman by Rank.

Rank	Diagnostic	Frequency	Percentage
1st	exogenous ochronosis	199	16.7%
2nd	Seborrheic dermatitis	128	10.7%
3rd	Eczema	119	10.0%
4th	Acne	104	8.7%
5th	Toxic epidermal necrolysis	98	8.2%
6th	Discoid lupus	60	5.0%
7th	Lichen planus	48	4.0%
8th	Dermatosis papulosa nigra	45	3.8%
9th	Melasma	42	3.5%
10th	Irritant dermatitis	40	3.3%

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Photo 1: Ochronosis of the Face.

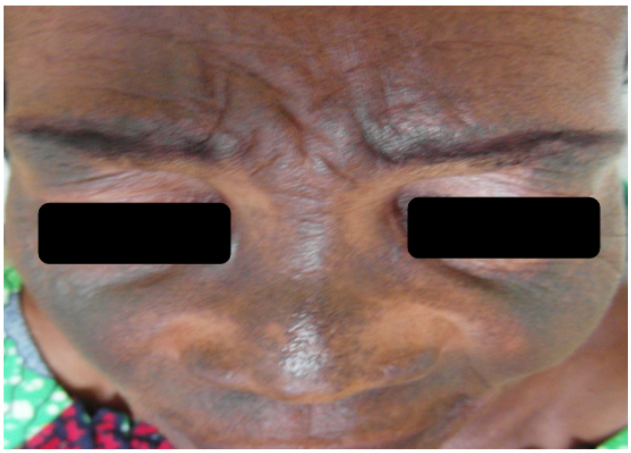


Photo 2: Seborrheic Dermatitis of the Face.

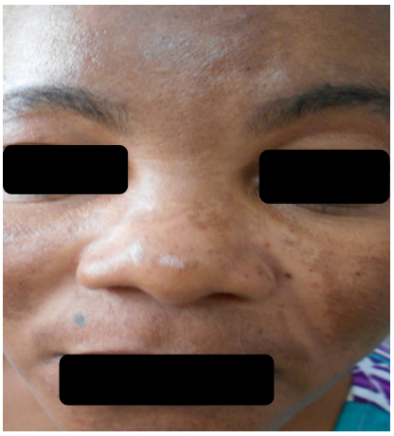
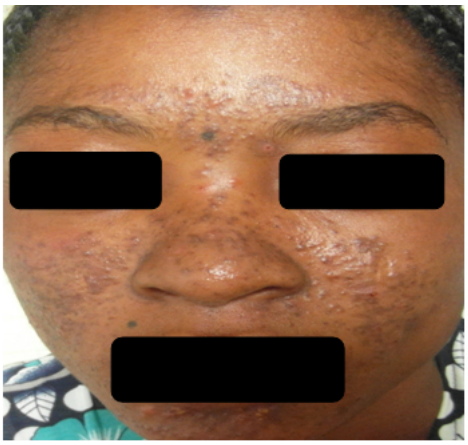


Photo 3: Acne of the Face.



Dermatosis leading to pigmentary disorders were the most frequent and they were followed by those composed of acne, seborrheic dermatitis with 24.1%, 19.5% of cases respectively. The group of pigmentary disorder were predominant because of the growth of the use of beauty products in black African generally and Ivory coast in particular. This practice is found in 49.2% of women who attended to consultation in Burkina according to Traore et al.[12] The prevalence of the people of Abidjan, the economic capital of Ivory Coast was estimated 53% in 2008.[13]

This practice was most often responsible for the first four facial dermatosis identified in our study (Exogenous ochronosis, eczema, seborrheic dermatitis and acne). Our study revealed the preponderance of exogenous ochronosis which are caused by the use of some beauty products containing hydroquinone.[6,14] Most often, women held surgery with the dermatologist because beautician and traditional doctors had failed to cure this dermatosis.[15-17] Infectious dermatosis occur rarely on the face of adult women whereas they occur frequently during our current general consultation as dermatologist.[15-17] Indeed infectious dermatosis are the first reason for consultation in the Teaching Hospital of Treichville, Abidjan.[15] The law prevalence of infectious facial dermatosis would be due to the improvement over years of the skin hygiene (Photos 1, 2 and 3).

CONCLUSION

Dermatosis which occurs on the face of black adult women are not rare. They occur most often in young girls with low financial income. The pigmentary disorders due to some depigmentation practices are preponderant. The most frequent are ochronosis exogenous, seborrheic dermatitis and acne.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

CONSENT

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

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Mini Review

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Spongiosis: A Short Review

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ABSTRACT

Spongiosis is the hallmark of eczematous dermatitis. The mechanism underlying it is somewhat complex and is not fully understood up till now. Many factors interplay to produce spongiosis. One of these factors is Fas receptor/Fas ligand interaction.

KEY WORDS: Spongiosis; Dermatitis; Fas.

ABBREVIATIONS: DSC: Desmocollins; DSG: Desmogleins; ECAD: E-cadherin; EPLIN: Epithelial Protein Lost in Neoplasm; IC: Intercellular; KCs: Keratinocytes; RCM: Reflectance Confocal Microscopy.

DEFINITION

Spongiosis is a process in which intercellular edema between the squamous cells of the epidermis causes an increase in the width of the spaces between them, separating the malphigian cells with stretching and eventually rupture of the intercellular prickles, and accentuation of honeycombed morphology of the upper epidermal layers appears accentuated, resulting in a sponge like appearance of the tissue (hence the name spongiosis).[1,2]

Another feature frequently observed is vesicle formation, which—either focal or widespread in extent—is seen on reflectance confocal microscopy (RCM) as well-demarcated that appear as dark hollow spaces between granular and spinous keratinocytes (KCs). Often small round, weakly refractile cells may be seen in the center of vesicles and microvesicles, these may correspond to apoptotic KCs or inflammatory cells.[1]

Exocytosis is regularly associated with spongiotic dermatitis, whereby the inflammatory cells are seen on RCM as bright, round highly refractile structures of about 8-10 mm, interspersed between KCs. Inflammatory cells may also be observed to various extents in perifollicular, perivascular or interstitial dermal distribution.[1]

MECHANISM

Spongiosis is a characteristic histopathologic appearance in eczematous dermatitis.[3,4] It entails condensation of KCs with widening of the intercellular (IC) spaces, IC edema and distention of the remaining IC contacts which give the epidermis a 'sponge-like' appearance. IC adhesion is normally anchored by desmosomes and adherens junctions.[5]

Fluid Accumulation

The two reasonable possibilities for the source of accumulated fluid in the intercellular space: (1) from the epidermal cells or (2) from the dermal fluids which arise, in turn, from the vessels. The epidermal cells alone cannot account for all the fluid, which is obvious in that an extremely spongiotic epidermis (e.g., a blister of contact dermatitis) often assumes a greater volume than the initial epidermis. If we accept that most of the fluid comes from the dermis, then why does that fluid accumulate in the epidermis.[6]

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There are two hypotheses for epidermal spongiosis fluid accumulation: (1) the fluid is pushed there by dermal hydrostatic pressure (due to decrease in the osmotic pressure of the dermis), or (2) the fluid is pulled there by epidermal osmotic pressure (due to increase in the osmotic pressure of inter-KCs space). In both cases a permissive basement membrane is assumed. The first possibility is clearly not operative; for under conditions in which there is massive dermal edema (increased dermal hydrostatic pressure), such as in urticaria or erythema multiforme, bullous formations often result in the upper dermis before any spongiosis is appreciable in the epidermis.[7]

While this mechanism may play a role in some situations, it is easier to visualize the epidermal cell as the agent influencing this process. The KC may respond to injury actively, for example, by altering its membrane cation pump, favoring an outflow of ions. A review of the cell physiology literature yields some experimental data from frog skin studies, suggesting that stratum spinosum cells may pump sodium ions into the epithelial interspaces,[8] and enough sodium salt may be transported into the interspaces to keep them expanded.[9]

In one report when sodium transport was stimulated, the IC spaces widened, and when sodium transport was inhibited by ouabain or dinitrophenol, the intercellular spaces closed.[10] A passive mechanism can also be hypothesized. Permeability of the KC plasma membrane could increase (e.g., from cell death), with leakage of cytoplasmic protein. This in turn would pull fluids to the inter-KC area.[6]

Detachment of Cells

Specific adhesiveness of KCs is provided by homophilic interactions of the cadherin superfamily.[11,12] Adherens junctions anchor actin microfilaments and contain E-cadherin (ECAD) as their transmembrane glycoprotein. The intracellular segment of ECAD associates with α-catenin, β-catenin, and ɤ-catenin (plakoglobin) .[12] Although, central to cellular adhesion, cadherins display physiologic functions beyond the mechanical interconnection of cells. It has been suggested that cadherins play a crucial role in regulatory pathways involved in various aspects of cell fate including developmental decisions, cell differentiation, and cell survival.[13]

CADHERINS

Cadherins are a superfamily of adhesion molecules that mediate Ca^{2+} dependent cell-cell adhesion in all tissues of that determine tissue architecture and control cell contact formation and dissociation during development, tissue homeostasis of all metazoans.[14]

This superfamily involves: Classical cadherins that are the major component of cell-cell adhesive junctions, desmogleins (DSG), desmocollins (DSC), protocadherins and some other cadherin-related molecules (e.g., The Fatprotein of Drosophila). Expression of particular cadherins often correlates with formation of discrete tissue structures, and in mature tissues discrete cell layers or other cell assemblies are often demarcated by particular cadherins.[15]

The majority of members of the cadherin superfamily are transmembrane glycoproteins that pass the membrane only once. The N- and C-termini of the cadherin protein chain are located outside and inside the cell, respectively.[16]

Classical cadherins contain five cadherin domains that are commonly designated as extracellular1 (EC1)-extracellular5 (EC5) (beginning with the N-terminus of the molecule). The conformation of the cadherin molecule is stable only in the presence of Ca^{2+}, whose binding with the EC portion of the polypeptide chain is prerequisite for cadherin-mediated cell-cell adhesion.[17] Removal of Ca^{2+} leads to a disordering of interdomain orientations, as can be seen by electron microscopy,[18] increased sensitivity to proteolysis, and increased motion between successive domains.[19]

THE EXTRACELLULAR DOMAIN (EC)

EC portion of the cadherin molecule consists of a varying number of so-called cadherin domains that are highly homologous to each other. Each domain is comprised of approximately 110 amino acid residues. EC cadherin domains per seared capable of hemophilic recognition and binding. It was shown that cells that express mutant cadherins lacking the cytoplasmic domains can bind with substrate covered with purified cadherin ectodomains. However, in this case adhesion is much weaker than in the case of cells bearing full-size cadherins.[17,20]

The Cytoplasmic Domain

The cytoplasmic region of classical cadherins, roughly 150 amino acids long, is the most highly conserved portion of these proteins. The juxta-membrane region binds to p120, and the carboxy-terminal ca. Hundred amino acids bind to β-catenin and to plakoglobin. Sequences homologous to the β-catenin/plakoglobin-binding region are also present in the desmosomes. The cytoplasmic domain of classical cadherins is associated with the cytoplasmic proteins catenins, which, in turn, serve as intermediate linkers between the cadherins and actin filaments**.** These data indicate that the formation of stable cell-cell junctions depends on the presence of functionally active cytoplasmic domain in the cadherin molecule and association of the latter with the cytoskeleton. Deletion of the cytoplasmic domain or the catenin-binding site suppresses stable cadherin-mediated adhesion of cultured cells.[17] Alternatively, over expression of the catenin-binding site also entails disruption of cell-cell junctions. This could be explained by competition of the expressed catenin-binding site with the endogenous cadherin for catenin binding.[21]

The Role of Cadherins in Mechanotransduction

Cadherins require anchoring to the cytoskeleton for properad-

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hesive function and junction organization. This is mediated by the catenins, a class of cytosolic proteins that were identified as cadherin-associated proteins necessary for cell adhesion.[22] Catenins form a protein family that is characterized by the armadillo repeat. Cadherins bind with their C-termini to β-catenin that in turn binds to α-catenin. The cadherin-catenin complex is connected *via* α-catenin with actin filaments, an interaction that may not be direct,[23] and might require further bridging proteins such as epithelial protein lost in neoplasm (EPLIN) or vinculin.[24]

Regulation of Cadherin Activity

Cadherin-mediated adhesion can be regulated by a variety of extracellular signals, including growth factors,[25] peptide hormones[26] signals from gap junctions and cholinergic receptor agonists.[27]

In response to these external stimuli, different signals are generated in the cell, of which protein phosphorylation is apparently, the most important for the regulation of cadherin function.[28]

Another mechanism of regulation of cadherin activity is changing the extent of clustering of cadherin molecules in the junction area which can significantly affect the strength of cell-cell interaction.[29]

Implications and Indications of Cadherins in Diseases

1) Cancer: Mutations that lead to a loss of ECAD, may play a role in cancer as found in laryngeal squamous cell carcinoma.[30] Down-regulation or loss of cadherins correlates with an increased metastatic potential of the affected cells due to the loss of their adhesive properties.[31]
2) Renal fibrosis is associated with downregulation of ECAD in kidney fibrosis.[32]
3) Cerebral cavernous malformation may be accompanied by irregular distribution of vascular endothelial-cadherin (VE-cad) and upregulation of N-cadherin in endothelial cells.[33]
4) Pemphigus vulgaris is associated with autoimmune disease directed against DSG1 and DSG3.[34]
5) Arrhythmogenic cardiomyopathy is associated with mutations in DSG2 and DSC2 in humans.[35]
6) Cognitive disorders and neurosensory diseases may be associated with protocadherin dysfunction.[36]

Loss of Cell Cohesion in Eczematous Dermatitis

Recently, it has been found that T-cell-mediated KC apoptosis plays a key pathogenic role in the formation of eczematous dermatitis. Spongiosis, the histologic hallmark of eczematous dermatitis, is characterized by impairment of cohesion between epidermal KCs. It is conceivable that the intercellular junction of KCs is an early target of apoptosis-inducing T-cells. It has been demonstrated that the induction of KC apoptosis is accompanied by a rapid cleavage of E-cad and loss of β-catenin. In situ examination of ECAD expression and cellular distribution in acute eczematous dermatitis revealed a reduction in KC membrane ECAD in areas of spongiosis. In contrast, the *in vitro* and *in vivo* expression of desmosomes during early apoptosis remained unchanged. Therefore, induction of KC apoptosis by skin-infiltrating T-cells, subsequent cleavage of ECAD, and resisting desmosomes suggests a mechanism for spongiosis formation in eczematous dermatitis.[5]

The development of spongiosis is initiated by early KC apoptosis due to cell shrinkage and cleavage of ECAD, which is essential in mediating KC cohesion. It has been found that impairment and loss of KC cohesion constitute the primary event in spongiosis formation. Therefore, despite being the obvious driving force of spongiosis formation, fluid influx into the skin is apparently not the primary step, but rather the end result of a sequence of pathogenic events. Accordingly, dermal inflammation and intense fluid influx into the dermis in urticaria leave skin coherence totally intact.[37]

In contrast, in early lesions of bullous autoimmune skin diseases in which desmosomes are targeted by auto-antibodies, spongiosis is visible. It should be noted here that spongiosis is a nonspecific sign of cutaneous inflammation involving the epidermis. It is found in all kinds of eczemas, in bullous skin diseases, and in some viral and superficial fungal infections as well.[38]

Spongiosis takes place mainly in the spinous layer of the epidermis. The heterogeneous basal layer contains stem cells, transit amplifying cells, and postmitotic differentiating cells with high expression of integrins.[39] It seems that in the basal layer at least stem cells exhibit strong anti-apoptotic defenses.[40] In contrast to adherens junctions that may contain only ECAD, desmosomes always include cadherins from two subfamilies, Dsg and Dsc.[41]

It has been demonstrated that apoptosis-induced protein cleavage in KCs is selective for certain adherens junction and desmosomal proteins. E-cad was cleaved, whereas β-catenin and desmosomal cadherins were not. The functional properties of ECAD and desmosomal cadherins are distinct despite their overall structural homology.[5]

The most striking sequence difference between Dsc, Dsg, and ECAD lies in their cytoplasmic tails.[42] This may contribute to the selectivity of the cytoplasmic tails for different plaque proteins connecting them with different cytoskeletal filaments. These differences may also account for the differential behavior of desmosomal cadherins and E-cad in KC apoptosis. In the spongiotic epidermis of eczematous dermatitis, not all KCs go into apoptosis. Therefore, it is likely that in areas of intense spongiosis there is additional cleavage of cadherins on by standing KCs without ongoing apoptosis possibly due to proteinases released from secondary necrotic KCs.[5]

E-cad acts as a substrate for activated caspases during KC apoptosis and its cleavage was inhibited by caspase inhibi-

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tors. These caspase inhibitors were also able to abrogate T-cell-induced KC apoptosis at the same concentration that blocked caspase-mediated cleavage events. Because of high levels of glycosylation of cadherins, several potential caspase cleavage sites, and different antibody epitopes.[5]

It was demonstrated that the cleavage site of ECAD during apoptosis is proximal to the transmembrane domain in the cytoplasm. At the 24 h time point the 85 kDa cleavage product was not consistently detectable, suggesting that further degradation may also occur.[43]

CONCLUSION

To summarize; T-cells infiltrating the skin in eczematous dermatitis induce KC apoptosis.[44] The early apoptotic response of KCs is characterized by cleavage of ECAD, whereas desmosomal cadherins remain intact. Hydrostatic pressure, which is an important factor in the development of spongiosis, and the portions of the epidermal cell surface that still retain desmosomes may explain the elongation and distortion of remaining IC contacts observed in histopathology.[5]

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Research

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Pattern of Children Hospitalization in a Tropical Dermatology Department: Case of the Teaching Hospital of Conakry (Guinea)

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ABSTRACT

Background: Skin diseases are important morbidity factors in both resource-limited and developed countries.
Aim: The objective of this work was therefore to document the different reasons for hospitalization of children in the Dermatology-Venereology Department of the Teaching Hospital of Conakry.
Methods: Patients aged less than or equal to 18 years hospitalized in the Department of Dermatology and Venereology of the Teaching Hospital of Conakry (Guinea) from January 2000 through December 31, 2014 were included.
Results: During this period of 14 years, a total of 227 patients were recruited. There were 122 (54%) girls and 105 (46%) boys. The boy to girl ratio was 0.85. Bacterial skin diseases (44.5%), inflammatory skin diseases (27.7%), drug reactions (14.9%), viral skin diseases (9.2%) were the most frequent skin diseases. Skin infections, mostly erysipelas and necrotizing fasciitis, accounted for 58.4% of the bacterial skin diseases. Of the non-infectious diseases, toxic epidermal necrolysis (29.4%), acute urticaria (13.7%) and atopic dermatitis (11.7%) were the most common.
Conclusion: In the Dermatology Department of the Teaching Hospital of Conakry, the reason for hospitalization of children is dominated by skin infectious, in particular erysipelas and necrotizing fasciitis followed by toxic epidermal necrolyses, with significant morbidity. An improvement in the socio-economic conditions of the population could reduce the prevalence of skin infectious in specialized hospitals.

KEY WORDS: Hospitalization; Infectious skin diseases; Children; Dermatology; Guinea.

BACKGROUND

Skin diseases are important morbidity factors in both resource-limited and developed countries.[1] In children particularly, these dermatological diseases represent a major public health problem with an overall prevalence ranging from 21% to 87%.[2] This high prevalence is particularly influenced by geographical and socio-economic factors.[3]

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While the existence of specialized pediatric dermatology consultation has provided data on children in developed countries, the situation remains quite different in resource-limited countries where skin disorders are among the five most common causes of morbidity.[4]

In these countries, which have few dermatologists, there is a low coverage in dermatological care, especially in children with special characteristics in all medical specialties.[2]

These dermatologic conditions are in marked increase and remarkably varied since the advent of HIV infection. They may occur at all stages of the infection, with varying frequency depending on their nature and, for some of them, are more likely to indicate HIV infection.[5]

Kiprono et al reported in a study in northern Tanzania, an HIV prevalence of 5.8% in children with skin diseases.[6]

To date, no data concerning the reasons for hospitalization of children in dermatological settings are available in Guinea.

However, the existence of data on this subject can provide a basis for effective planning of preventive measures in a context of low socioeconomic level such as ours.

The objective of this work was therefore to document the different reasons for hospitalization of children in the Dermatology-Venereology Department of the Teaching Hospital of Conakry.

MATERIALS AND METHODS

This monocentric, retrospective study was conducted during the period of January 2000 to December 31, 2014 in the Dermatology and Venereology Department of the Teaching Hospital of Conakry (Guinea).

This was a of the records of patients aged less than or equal to 18 years hospitalized in the Department of Dermatology and Venereology.

The patients were grouped into 5 categories based on their age at the hospitalization: 0-12 months, 13 months-2 years, 25 months-6 years, 7-12 years and 13-18 years. For each patient, the variables studied were: age, sex, time of consultation, reason for hospitalization, duration of hospitalization and course of illness.

The different conditions have been grouped into infectious skin diseases (bacterial, viral, parasitic, fungal) and non-infectious skin diseases (immuno-allergic, drug reaction, tumor, congenital).

The diagnosis was made on the basis of detailed history and clinical examination. Complementary investigations (bacteriological, radiological and cutaneous biopsy) were performed when the clinic was not sufficient to establish a diagnosis.

All incomplete files (files not mentioning data such as age, sex, year of admission, diagnosis of hospitalization or evolution) were excluded.

Ethically, the study was approved by the ethics group of the Faculty of Medicine through the research department.

RESULTS

During this period of 14 years we hospitalized 4,437 patients, of all ages in the dermatology department. Of these, 227 were an age between 0 and 18 years, i.e., a hospitalization rate of 5%.

There were 122 girls and 105 boys with an average age of 11.36 years and extremes of 3 months and 18 years. The boy to girl ratio was 0.85. Twenty-five incomplete files were excluded.

Patients in the 13-18 years group were the most represented, with a proportion of 54.1% followed by the 7-12 years group with 19.8%. The distribution of the different patients by sex and age group is summarized in Table 1.

There were 125 cases (55.1%) of infectious skin diseases and 102 cases (54.9%) of non-infectious skin diseases.

Table 1: Distribution of Patients According to Different Diseases.

Diseases	Frequency (N=227)	%
	125	55.1
Infectious skin diseases bacterial	101	44.5
Viral	21	9.2
Fungal	1	0.4
Parasitic	2	0.8
Non infectious skin diseases	102	44.9
Inflammatory or immunologic allergic	63	27.7
Drug reactions	34	14.9
Tumor	3	1.3
Congenital	2	0.8
Total	227	100.0

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Infectious skin diseases were dominated by erysipelas, representing the first pattern of hospitalization with 37.6% followed by necrotizing fasciitis in 20.8% of patients, the majority of whom were between the ages of 13 and 18 years (Table 2).

The non-infectious skin diseases distributed in four entities summarized in Table 3 were largely represented by bullous drug reaction with a proportion of 29.4%.

Retroviral serology was positive for type 1 HIV in nine out of 195 patients who performed it, a prevalence of 4.5%. There were 5 cases of kaposi disease, by a case of profuse molluscum contagiosum and 2 cases of chicken pox.

The mean hospital stay was 13 days with extremes of 1 and 60 days. The trend was favorable in 87.2% of the cases. The mortality rate was 7.5%.

DISCUSSION

Our study is the first to document the reasons for hospitalization of children in dermatology department in Conakry.

Table 2: Distribution of Patients According to Different Infectious Skin Diseases and Age Groups.

Effect if (N=125)

Infectious skin diseases	0-12 months	13-24 months	25 months-6 years	7-12 years	13-18 years	Frequency N (%)
Bacterial						
Erysipela	0	2	9	8	28	47 (37.6)
Necrotizing fasciitis	0	1	2	7	16	26 (20.8)
Cutaneous tuberculosis	0	0	1	1	8	10 (8.0)
Ecthyma	2	2	0	1	5	10 (8.0)
Staphylococcal malignant of the face	0	0	1	3	4	8 (6.4)
Fungal						
Basidiobilomycosis	0	0	1	0	0	1 (0.8)
Parasitaires						
Profuse scabies	0	0	0	1	1	2 (1.6)
Viral						
Chicken pox	0	2	4	3	4	13 (10.4)
Kaposi disease	0	0	0	1	4	5 (4.0)
Profus Molluscum contagiosum	0	0	0	0	1	1 (0.8)
Zona femoral	0	0	0	1	1	2 (1.6)
Total	2	7	18	26	72	125 (100)

Table 3: Distribution of Patients According to Different Non-Infectious Pathologies and Age Groups.

Non Infectious skin diseases N=102	0-12 months	13-24 months	25 months - 6 years	7-12 years	13-18 years	Frequency N (%)
Inflammatory or immunologic allergic						
Erythrodermic eczema	0	0	0	5	7	12 (11.7)
Acute eczema	0	0	1	0	8	9 (8.8)
Pyodermaga gangrenosum	0	0	3	0	0	3 (2.9)
Atopic dermatitis	0	0	3	0	9	12 (11.7)
Erythrodermic syndrome	0	0	0	0	3	3 (2.9)
Acute urticaria	0	0	0	9	5	14 (13.7)
herpétiforme dermatitis	0	0	0	3	2	5 (4.9)
Psoriasis	0	0	0	0	2	2 (1.9)
Lichen planus	0	0	0	0	3	3 (2.9)
Congenital skin disease						
Ichtyosis	2	0	0	0	0	2 (1.9)
Tumor skin disease						
Hemangioma	3	0	0	0	0	3 (2.9)
Drug reactions						
Toxic epidermal necrolysis	0	0	9	9	12	30 (29.4)
Generalized bullous pigmented erythema	0	0	0	2	2	4 (3.9)
Total	5	0	16	28	53	102 (100)

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However, it has limitations inherent to its retrospective nature and to the fact that the majority of diagnoses have been essentially clinical due to lack of technical plateau.

Despite these limitations, it allowed us to increase the hospitalization rate for children by 5% over the period from January 1, 2000 to December 31, 2014, an average of 15 hospitalizations per year.

The main reasons for hospitalization were decreasing frequency of bacterial infections dominated by erysipelas followed by necrotizing fasciitis, inflammatory and immunoallergic skin diseases dominated by acute urticaria followed by erythrodermic eczema and atopic dermatitis and, severe drug reactions dominated by toxic epidermal necrolysis.

The hospitalization rate of 5% observed is relatively low compared to the prevalence of 9-37% and 40.9% respectively reported by Sayal et al[7] and Khalifa et al[8]. However, it is higher than that of 0.3% reported by Afsar et al.[9]

On the one hand, this difference in outcome could be related to the mode of recruitment, which in our study took into account only hospitalized cases. On the other hand, other services such as pediatrics share the same enclosure as our recruitment site. In addition to these aspects, cultural contingencies could be added, whereby the majority of patients consult traditional healers first or practice self-medication, consulting the specialist as a last resort, especially for chronic and most skin diseases. Indeed, in some cultures, the tendency to find a supernatural cause for phenomena extends far beyond the framework of medicine.[10]

Different age groups can be reached with unequal proportions. With an average age of 11 years and extremes of 3 months and 18 years; the age group between 13-18 years was the most represented in our study. This result goes in the same direction as that reported by Oninla et al[11] in Nigeria but differed from that of Marrone et al[12] in Ethiopia for whom the age range between 1 and 5.9 years was the most affected.

The type of skin disease in any geographical area is influenced directly or indirectly by various climatic, cultural and socio-economic factors.[3,13,14]

Due to the often unfavorable socio-economic conditions combined with the promiscuity in which most children live, infectious pathologies are considered by many authors to be the most frequent in children in tropical environments.[11,12,14,15] Contrary to the study by Adegbidi et al[16], for whom immunoallergic skin diseases dominated by eczema, was the leading pediatric dermatosis in hospital in Cotonou. Our study was mainly dominated by infectious pathologies (55.1%) dominated by erysipelas.

This result is similar to that of some authors in the subregion, where predominant infantile skin diseases were infectious.[17,18]

It is known that HIV infection makes the bed of several skin diseases especially infectious conditions. Only 197 patients out of 227 in our series performed retroviral serology. The breakdown of inputs for the realization of the retroviral serology linked in part to the socio-political disorders that occurred in Guinea during the period of 2007 is one of the justifications for the non-realization of the retroviral serology in all the patients. HIV prevalence among hospitalized children was 4.5%. In the Kiprono et al[6] study in Tanzania, HIV prevalence among children with cutaneous disease was 5.8. In their study, the majority of HIV positive children had inflammatory diseases and Kaposi disease.

In our study, skin diseases associated with HIV infection were dominated by kaposi disease (6 cases) followed by a case of profuse molluscum contagiosum and 2 cases of chicken pox.

These pathologies have a high positive predictive value for HIV infection.

This prevalence of HIV in children (national prevalence in the general population is 1.7) highlights the importance of intensifying HIV prevention campaigns in general and mother-to-child transmission in particular.

Erythrodermic eczema followed by atopic dermatitis were the most common among immunoallergic disorders with respective proportions of 5.3% and 3%. This proportion of immunoallergic skin diseases is different from those reported in developed countries where the prevalence of atopic dermatitis can reach 25.9%.[19]

Toxic epidermal necrolysis accounted for 29.4% of non-infectious diseases.

The hospital stay did not exceed two weeks (average duration 13.43 days) with extremes of 1 and 60 days. Most skin diseases are not life-threatening; however, some of these dermatological pathologies can lead to alterations in the general condition leading up to death. We found a favorable outcome in 87.2% and 7.5% of deaths. Epidemic Kaposi's disease and Toxic epidermal necrolysis were the tables in which the deaths were mainly observed. This is in line with the literature. Mortality due to severe drug reaction is 20-25% during hospitalization.[20]

In the Ivory Coast, Ecra et al[21] reported 10.26% mortality due to skin diseases. It was respectively due to the infectious skin diseases (41.57%), followed tumor dermatosis (29.77%) and drug reactions (16.85%).

CONCLUSION

With an average hospitalization rate of 5%, this retrospective study shows that the hospitalization of children represents a significant part of the activities of the Dermatology Department of the Teaching Hospital of Conakry.

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It also points out that skin infectious, in particular erysipelas and necrotizing fasciitis followed by toxic epidermal necrolysis, are the main reasons for hospitalization with significant morbidity. An improvement in the socio-economic conditions of the population could reduce the prevalence of skin infectious in specialized hospitals.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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