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UVA1 Phototherapy: A Concise and Practical Review

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ABSTRACT

High intensity long-wavelength ultraviolet A (340-400 nm; UVA1) lamps were initially developed as skin research tools; over time they have proven to be useful for treating a number of chronic dermatoses. UVA1 units and dosimetry are strikingly different from conventional UV phototherapy. The therapeutic effect of UVA1 is related to the fact that its long wavelength penetrates the dermis more deeply than UVB. UVA1 radiation induces collagenase (matrix metalloproteinase-1) expression, T-cell apoptosis, and depletes Langerhans and mast cells in the dermis. UVA1 exposure stimulates endothelial cells to undergo neovascularization. Ultraviolet A1 exerts significant therapeutic effects in atopic dermatitis and morphea; there is also evidence for its use in other skin diseases, including cutaneous T-cell lymphoma and mastocytosis.

Key words: phototherapy, skin diseases, ultraviolet A1, UVA1

Introduction

The roots of ultraviolet A1 (UVA1) phototherapy can be traced to the development of a relatively "pure", high-intensity UVA light source that was originally meant to be used for studying the physiologic cutaneous effects of UVA alone. UVA photons are approximately 1000 times less potent than UVB photons in eliciting photobiological responses, and thus, the technological challenge was to develop an artificial lamp that could deliver a biologically relevant UVA dose within a practical time frame and a sufficiently large irradiation field.[1] Using a specially filtered metal halide lamp, the spectral output was weighted towards longer, more penetrating UVA wavelengths (340-400 nm), and was distinctly different from that of fluorescent UVA tubes used for psoralen + UVA (PUVA) therapy. Over time UVA1 came to be used diagnostically for photoprovocation of conditions such as polymorphous light eruption and then as a novel treatment modality for certain inflammatory dermatoses.

UVA1 induces T-cell apoptosis, which is one of its proposed mechanisms for improving atopic dermatitis (AD), mycosis fungoides (MF), and localized scleroderma.[2] Consistent efficacious results with UVA1 have been observed with a variety of inflammatory, sclerosing, and neoplastic skin diseases that are characterized by dermal infiltrates rich in T lymphocytes.[3] UVA1 is one of the most recent advances in phototherapy for localized scleroderma and systemic sclerosis, and has been used more extensively in Europe than North America or Asia.

UVA1 Phototherapy in Practice

UVA1 treatment units typically consist of metal halide lamps equipped with a series of special optical filters. Smaller units provide localized therapy, whereas whole-body treatment is best carried out using lie-down or standing UVA1 cabinets. Standing cabinets are more practical for whole-body treatment since lie-down units can only expose one side of the body (i.e., anterior or posterior) at a time. Fluorescent tubes emitting predominantly in the UVA1 range have also been used for long wave UVA therapy, but these are not as powerful and efficient as filtered metal halide lamps. In North America UVA1 availability is limited, perhaps due to the relatively high equipment costs; UVA1 units are usually two to three times more expensive than conventional whole body UV treatment units.

UVA1 dosimetry has been categorized into low (≤40 J/cm^2), medium (40-80 J/cm^2), and high (80-130 J/cm^2) dose regimens,[4] and depending on the desired fluence and the irradiance of the UVA1 phototherapy unit, treatment times can range between 10 minutes and 1 hour per treatment session. Like other forms of UV phototherapy UVA1 requires a series of repeated exposures. However, with UVA1 the treatment fluence is usually held constant for a given course of therapy in contrast to UVB and PUVA, where the dosing is increased incrementally with each successive exposure. Prior to initiating treatment it may be appropriate to phototest the patient's normal skin to screen for unusual UVA1 reactivity (e.g., occult polymorphous light eruption or UVA photosensitivity). The number of treatment

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sessions recommended for atopic dermatitis is usually 15 and for morphea or systemic scleroderma 20-40 treatments are given. Patients are treated daily with a break on the weekends. Continued improvement is often observed for up to several months after a treatment course; therefore, therapy is usually limited to 1-2 courses per year. In addition, since there is no data on the remission potential of UVA1, maintenance phototherapy is not routinely recommended. As the long-term side-effects of UVA1 are not well established, patients younger than 18 years should be treated judiciously.

Biologic and Mechanistic Effects of UVA1

UVA1 induces immediate tanning through oxidation of pre-existing melanin and also causes delayed pigment darkening by an increase in melanin content.

Studies have shown that the mechanism of apoptosis with UVA1 differs from UVB and PUVA.[5] UVA1 induces early apoptosis or preprogrammed cell death through two apoptotic pathways in lymphocytes and immature proliferating mast cells.[6] The first pathway involves the production of superoxide anions and the second apoptotic pathway produces singlet oxygen species, which depolarize mitochondrial membranes.[5] Apoptosis of T-cells underlies UVA1's therapeutic effects in atopic dermatitis, mycosis fungoides (MF), and inflammatory scleroderma. Studies have been shown that UVA1 suppresses TNF-α, IL-12, IFN-γ, and ICAM-1.[7-10] IL-6 and IL-8, cytokines with pivotal importance in sclerotic skin diseases, are down regulated by UVA1 in localized scleroderma lesions.[11]

Irradiation with UVA1 increases collagenase synthesis, as demonstrated by increased levels of collagenase mRNA and protein in cultured fibroblasts from morphea patients.[12] Recently, it has been revealed that UVA1 radiation suppresses calcineurin activity, both *in vivo* and *in vitro*. This loss in activity is due to singlet oxygen and superoxide generated by photosensitization. These findings provide a mechanistic basis for the hypothesis that UVA1 and calcineurin inhibitors both affect the same signal transduction pathway in the skin.[13]

Indications

Although the use of UVA1 has been reported in a range of conditions, the main indications of UVA1 phototherapy are atopic dermatitis, cutaneous T-cell lymphoma, sclerosing skin diseases, and mastocytosis (Table 1).

Atopic Dermatitis

In 1992 Krutmann et al showed that UVA1 improved patients with atopic dermatitis, thus becoming the first skin disease to be effectively treated by UVA1.[2] The main mechanisms by which UVA1 phototherapy induces remission in atopic dermatitis involves a range of immunomodulating effects that include apoptosis of infiltrating T-cells, suppression of cytokine levels, and reduction in Langerhans cell numbers.

UVA1 has been proved to be superior to combined UVA/UVB therapy in several studies.[14] Narrowband (NB) UVB and medium-dose UVA1 are equally effective in the treatment of patients with moderate to severe AD.[15] Tzaneva et al showed that high-dose and medium-dose UVA1 therapies have comparable efficacy in severe atopic dermatitis. Both high- and medium-dose regimens achieved comparable results as demonstrated by similar reductions in clinical scores.[16] Low-dose UVA1 phototherapy did not reduce severity of atopic dermatitis.[17] Several controlled studies indicate that UVA1 is effective in acute, severe AD and superior to broadband UV regimens, and that a course of medium-dose UVA1 may be a safer modality than low-dose UVA1. Due to practical considerations (i.e., availability, exposure times, and clinical experience) conventional UV therapy remains the first treatment of choice for phototherapy in atopic dermatitis, with UVA1 being reserved for acute severe exacerbations.

Sclerotic Skin Diseases

Phototherapy is an effective therapeutic option in scleroderma and should be considered among the first approaches in the management of localized scleroderma or morphea. Collagen metabolism disturbance, autoimmune activity, and vascular dysregulation are the main pathways that lead to the development of scleroderma.[18] UVA1 photons are the most deeply penetrating form of UV therapy and they appear to exhibit their effects in all three of the above pathways by induction of collagenase messenger RNA expression, depletion of skin T-cells and cytokines (IL-1, IL-6), and neovascularization.[19-21]

High-dose UVA1 treatment for scleroderma was first conducted by Stege et al in 1997, who revealed that high-dose UVA1 phototherapy reduced sclerotic plaque thickness while increasing their elasticity.[22] Andres et al in 2010 also showed that UVA1 phototherapy had a significant effect on collagen metabolism by reducing sclerotic plaque and lesional skin thickness, and improving skin elasticity.[23]

Kreuter et al in a comparative study demonstrated that medium-dose UVA1 was superior to both low-dose UVA1 therapy and NB-UVB therapy with no significant difference between low-dose UVA1 and NB-UVB.[21] UVA1 phototherapy has also been used for patients with limited and diffuse systemic sclerosis. Morita et al treated sclerotic skin on the forearms of four patients with systemic sclerosis. Sclerotic skin lesions were softened after 10-30 exposures of medium-dose (60 J/cm^2) UVA1 therapy, resulting in increased passive joint mobility and cutaneous elasticity in patients with sclerosis.[24] A case report documented

Indication	Strength of Evidence	Recommended Dosing Regimen*	Treatment Course (# of exposures)
Atopic dermatitis	Randomized controlled trials	Medium-dose	15
Sclerosing skin diseases	Randomized controlled trials	Medium- and/or high-dose	20-40
Mycosis fungoides	Open studies	Medium- and/or high-dose	10-35
Urticaria pigmentosa	Open studies	Medium- and/or high-dose	10-15

Table 1: Main indications and recommendations for UVA1 phototherapy
* Medium-dose = 40-80 J/cm^2, high dose = 80-130 J/cm^2

UVA1's effectiveness in softening sclerotic perioral skin and improving symptoms related to microstomia in systemic sclerosis.[25]

Cutaneous T-cell Lymphoma (Mycosis Fungoides)

UVA1 phototherapy was used by Plettenber et al in three patients with stage IA and IB mycosis fungoides (MF). Complete clearance was achieved after 16 to 20 exposures, with a high- or medium-dose regimen.[26] In another study, 13 patients with widespread plaque-type, nodular and erythrodermic MF were given 100 J/cm^2 UVA1 phototherapy 5 times/week. Eleven patients showed complete response and partial improvement was observed in two patients. Circulating CD4$^+$/CD45RO$^+$ and CD4$^+$/CD95$^+$ lymphocytes were significantly reduced with therapy.[27] Suh et al treated 15 MF patients with UVA1, with 13 and 2 patients showing complete and partial remissions, respectively. This study reported that UVA1 therapy induced excellent therapeutic efficacy in patients with MF, delivering a quick response, and is safe in early and advanced stages of MF.[28]

Mastocytosis

UVA1 phototherapy reduces the density of dermal mast cells and has been reported to be effective for patients with urticaria pigmentosa. Four adult patients with generalized urticaria pigmentosa were treated with 130 J/cm^2 UVA1 for 2 weeks. Pruritus improved after three treatment sessions, and two patients with diarrhea and migraine experienced relief of these symptoms as well. None of these patients had relapsed after at least 10 months follow-up, although the authors did not specifically report on the response of the skin lesions to UVA1.[29] Gobello et al treated patients suffering from cutaneous mastocytosis with high- and medium-dose UVA1. In the majority of patients, the number of visible skin lesions was not significantly reduced; however, the number of mast cells in lesional skin decreased markedly in most patients. Pruritus and quality of life improved by the end of treatment and during the 6-month follow-up. No significant differences were observed between patients receiving high- or medium-dose UVA1.[30]

Other Skin Conditions

Other diseases treated with UVA1 with varying degrees of response include lichen sclerosus et atrophicus, dyshydrotic hand eczema, scleredema, necrobiosis lipoidica, granuloma annulare, pityriasis lichenoides chronica, systemic lupus erythematosus, sarcoidosis, granulomatous slack skin, and graft-versus-host disease. There are no controlled clinical trials investigating the efficacy of UVA1 on psoriasis. Because of its cost, longer exposure time, and limited availability, UVA1 is not used for psoriasis.

Side-effects of UVA1

Side-effects of UVA1 are usually fewer than with other types of phototherapy and most studies have reported no serious adverse effects. Most notably, the frequency of UV-induced burning seems to be lower for UVA1 than for conventional UVB or PUVA. In our experience, the minimal erythema dose for UVA1 is typically greater than 130 J/cm^2. Side-effects with UVA1 phototherapy have been classified as acute or chronic. The most common acute side-effects are hyperpigmentation, redness, dryness, and pruritus. Hyperpigmentation or tanning are virtually universal side-effects and can be striking, particularly within the active affected skin sites. Other observed side-effects include herpes simplex virus reactivation and polymorphic light eruption induction.[31] Chronic side-effects in patients who receive UVA1 phototherapy include photoaging and possible photocarcinogenesis. Reports of skin cancer in patients treated with UVA1 are usually confounded with the use of other therapies known to also increase the risk of cutaneous malignancies. For instance, a case of melanoma was reported in a patient with mastocytosis after receiving UVA1 treatment, however, this individual had also received PUVA bath therapy in the past.[32] As well, there are two cases of Merkel cell carcinoma after UVA1 phototherapy, but both patients had blood dyscrasias and were treated with immunosuppressants.[33]

Contraindications to UVA1 therapy include photosensitivity disorders such as xeroderma pigmentosum and porphyria disorders. Relative contraindications also include a history of melanoma or nonmelanoma skin cancers, immunosuppressed individuals following organ transplant, and patients who have received prior radiation treatment, which potentially predisposes them to skin tumor development.[4]

Conclusion

UVA1 is a relatively new, unique, and possibly underutilized therapeutic modality available in photodermatology that has shown relatively good evidence for treating atopic dermatitis and sclerotic skin diseases. Overall, the side-effects from therapy are well tolerated by patients, with the long-term adverse effects and relative utility for other dermatoses still remaining to be better elucidated.

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ABSTRACT

Several variants of psoriasis are seen in children, the most prevalent types include plaque, guttate, and psoriatic diaper rash; pustular and erythrodermic psoriasis are less frequently observed. Genetic susceptibility and environmental triggers are both involved in the development of this autoimmune disease. As well as improving symptoms, a treatment plan should strive to identify and eliminate precipitating factors. Topical medications are the first choice therapy for children with psoriasis. Systemic agents are used to treat more severe cases. Patient education and supportive care should be incorporated into the treatment plan.

Key words: adolescent, children, psoriasis

Introduction

Psoriasis is a common condition that affects about 3.5% of the population.[1] In greater than 33% of patients, the initial presentation of psoriasis occurs within the first two decades of life.[2-5] It is estimated that 10% of patients develop psoriasis before the age of 10.[6] In a review of 1262 cases of psoriasis, initial disease onset occurring before the age of 2 years was found in 14-27%.[7] Children present with the same clinical variants of psoriasis seen in adults, though they may differ in distribution, morphology, and natural history.[5]

Etiology

Psoriasis is a T-cell mediated chronic inflammatory condition characterized by keratinocyte hyperproliferation, vascular endothelial proliferation, and inflammatory cell infiltration.[8,9] The exact cause and pathogenesis of psoriasis are not well understood, but are known to be multifactorial, having both genetic and environmental influences.[9] Seventy-one percent of children with psoriasis have a positive history for psoriasis in a first degree relative.[7] The PSORS1 gene has been shown to be a major genetic determinant of Type 1 early onset non-pustular psoriasis.[5,10] HLA-Cw6 is the major disease allele at the PSORS1 locus that confers susceptibility to early onset disease.[2,5,11,12]

Exogenous and endogenous factors, such as upper respiratory infection, emotional stress, skin injury, and drugs, can precipitate and exacerbate psoriasis in children.[2,6,8,13] Streptococcal pharyngitis and perianal streptococcal dermatitis are common causes of guttate psoriasis in children.[2,7,8] Frequency of sore throat and skin trauma leading to an exacerbation of psoriasis is greater in pediatric onset psoriasis than adult onset.[5,13] The appearance of new lesions in times of emotional stress is also more common in pediatric patients.[13] Injury or irritation of normal skin can induce new psoriatic lesions at the site, known as the Koebner phenomenon. Antimalarials and the withdrawal of corticosteroids play a significant role in rebound psoriasis and the induction of childhood psoriasis, whereas β-blocking agents and lithium are recognized triggers for psoriasis in adult patients.[11,14]

Presentation

Classic psoriasis presents as sharply demarcated, deep red plaques with silvery scales.[9] The presentation in children may be atypical, thus making a diagnosis difficult in such cases; however, there are a few clinical features that can aid in identification. The Auspitz sign, which is pinpoint bleeding upon removal of scales, is characteristic of psoriasis.[2,15] Nail changes, such as oil spots, onycholysis, subungual hyperkeratosis and pitting (the most common finding), are frequently observed in adolescents with psoriasis and are valuable clues in establishing diagnosis.[2,8,9,16,17]

Psoriasis often presents differently in children than in adults. Involvement of the face and flexural regions are more common in children than adults, and psoriatic lesions in the diaper area are prevalent during infancy.[8] Plaque-type psoriasis is the most common variant in both adults and children, however, lesions in children are smaller, thinner, and less scaly than those seen in adults.[2,5,7] Pustular and erythrodermic psoriasis are less frequently seen in pediatric than adult patients. Though rare in occurrence, there are also reports of congenital[6,18] and naevoid[19] forms of psoriasis.

Plaque-type psoriasis is the most prevalent variant that affects children. Plaque psoriasis routinely affects the scalp. Scalp involvement characterized by pityriasis amiantacea (thick, adherent white scales that encase the hair shaft) may lead to temporary hair loss and visible psoriatic alopecia.[2,8] Plaque psoriasis can also affect the face, as well as extensor and flexor surfaces of the knees and elbows.[2,9]

Psoriatic diaper rash is the next most common variant, with highest prevalence in children under the age of 2 years.[5,7] Psoriatic diaper rash features a bright red, well-demarcated, glazed, diaper rash that may be followed by widespread dissemination of small psoriasis-like lesions.[2] This clinical variant can be differentiated from irritant diaper dermatitis by its unique presentation and poor response to conventional treatment for diaper dermatitis.[2]

Guttate type psoriasis presents as annular, localized, red to salmon colored plaques with hyperkeratosis, commonly located on the trunk, abdomen, and back.[8] Streptococcal pharyngitis and perianal dermatitis frequently precede abrupt appearances of guttate psoriasis.[20] Acute guttate psoriasis that is preceded by an upper respiratory infection may resolve spontaneously after 3-4 months; however, a significant portion of patients eventually develop chronic plaque disease.[2,9]

Pustular and erythrodermic psoriasis are less frequently seen in children than adults.[2,9] Pustular psoriasis is distinguished by the

presence of sterile pustules on erythematous skin; the pustules may be either localized or generalized.[2] Generalized pustular psoriasis in children has a more benign course than in adults.[21] Annular pustular psoriasis, a manifestation of generalized pustular psoriasis, occurs more frequently in children than in adults.[2,22] It is characterized by annular lesions with erythematous, scaly, and pustular margins. Erythrodermic psoriasis presents as erythema on >90% of the body surface area with less scales than plaque psoriasis.[21]

Treatment

When treating children with psoriasis, it is important to educate both patients and parents about the nature of the disease. It must be made clear that psoriasis is a chronic skin disorder without a permanent cure and, therefore, the goal of treatment is to establish disease control and prolong periods between flares.[23] Treatment results may vary from flattened plaques and reduced visibility of lesions (e.g., less redness and scale) to complete remission.[2] Proper education about the disease and treatment options often enhances the compliance of patients and their parents.[2,9]

The patient's age, quality of life factors, Psoriasis Area and Severity Index (PASI) score, and therapeutic preferences should all be considered when determining treatment selection.[5,8] The majority of children have mild disease that can be successfully treated with topical agents. Systemic drug therapy in children is generally reserved for severe disease that is resistant to other treatments.[5] A prevention strategy should aim to control and reduce known exogenous and endogenous factors that trigger or contribute to disease exacerbation, like skin trauma, emotional stress, aggravating drugs, and upper respiratory infections.[13]

A chronic, visible condition like psoriasis can have a significant impact on children's psychosocial development.[24] Through school years and adolescence, children may require substantial family and professional support to cope with the psychological and social sequelae of psoriasis, particularly the negative reactions of other children.[25]

Topical Medications

Corticosteroids

Corticosteroids have anti-inflammatory and antiproliferative properties that reduce erythema, scaling, and pruritus.[5,9] Corticosteroids have high acceptability among patients because they do not stain and are almost odorless. This acceptance combined with wide availability, ease of use, and faster onset of action make corticosteroids the first choice treatment of childhood psoriasis, especially in flexural disease.[2] Very high potency corticosteroids should be used only sparingly in combination or rotation with steroid sparing alternatives, such as coal tar, liquor carbonis detergens, anthralin, calcipotriene (calcipotriol), and topical calcineurin inhibitors.[5] Combination therapy can help reduce side-effects caused by topical steroids without reducing the efficacy of the treatment.[9] Side-effects of topical steroids include skin atrophy, striae, telangiectasia, acneiform eruptions, and in rare cases, suppression of the hypothalamic-pituitary-adrenal axis may occur after prolonged widespread application or overuse, especially of potent preparations.[2,5] There are reports of tachyphylaxis associated with prolonged corticosteroid use in the treatment of psoriasis. However, some attribute this phenomenon to decreased adherence to long-term therapeutic regimens.[5,26,27] Treatment with corticosteroids should be gradually withdrawn to prevent rebound flares.[9]

Coal Tar

The use of coal tar, which is both antiproliferative and antipruritic, is limited by its strong odor and ability to stain. A modified coal tar preparation, liquor carbonis detergens (LCD), has largely replaced crude coal tar in outpatient settings because of its superior cosmetic acceptability.[5] Coal tar is less irritating than calcipotriene and anthralin on the face and flexures, sites commonly affected in children.[25]

Anthralin

Anthralin (dithranol) is a potent anti-inflammatory and antiproliferative agent. Its negligible systemic absorption makes it a safe and easy treatment option for children.[5] Anthralin's use is limited due to its tendency to stain skin and clothing and irritate healthy skin. It is not recommended for application on the face, flexures and genitalia, and should not be used in erythrodermic or pustular psoriasis.[9] In an open study of 58 children ages 5-10 years, remission was achieved in 47 patients (81%) using dithranol at concentrations up to 1%.[28]

Calcipotriene

Calcipotriene (calcipotriol) is a vitamin D analogue that stimulates keratinocyte differentiation and inhibits DNA synthesis and proliferation.[23] It is considered to be a successful and safe treatment for children with mild to moderate plaque psoriasis involving <30% of the body surface.[2] Calcipotriene is non-staining and odorless.[9] Potential side-effects include local intolerance or irritation.[8]

Topical Calcineurin Inhibitors

Tacrolimus and pimecrolimus are non-steroidal immuno-modulating macrolactams that inhibit the production and release of interleukin-2 (IL-2) and subsequent T-cell activation and proliferation, through blockade of the enzyme calcineurin.[5] They are particularly useful for treating pediatric psoriasis in areas where atrophy is a risk, such as the face, intertriginous regions, and the groin.[9]

Salicylic Acid

Salicylic acid is recommended for use on thick localized plaques.[2,5] However, salicylic acid should be avoided in infants and children less than 6 years of age, or otherwise used with caution, as there is a risk of percutaneous absorption and salicylate intoxication.[2,5]

Phototherapy

Phototherapy is extensively used in adults and is a treatment option for children with widespread plaques.[2] Narrowband UVB (NB-UVB) phototherapy may be combined with topical therapies to enhance efficacy of both modalities and to reduce the NB-UVB dose and carcinogenic risk.[2,5] Psoralen + UVA (PUVA) therapy is not generally recommended in young children, but may be used in adolescents with caution.[5,9,25] When PUVA is administered, topical psoralens are chosen preferentially over oral psoralens to avoid gastrointestinal side-effects and the necessity to wear protective eye gear for 24 hours.[2,8] NB-UVB is considered the first-line phototherapy because it is as effective as PUVA, more convenient, and less carcinogenic.[5,29]

Systemic Medications

Acitretin

Acitretin, a retinoid, is an effective treatment for severe plaque, pustular, and erythrodermic psoriasis in adolescents.[5] It can be used as monotherapy or in combination with topical agents and NB-UVB phototherapy. Side-effects include cheilitis, pruritus, and hair loss.[2] Because of its high teratogenic risk, acitretin should be used with caution in girls of childbearing age and must be accompanied by oral contraceptive therapy, as well as counseling, to avoid pregnancy during and 3 years after the completion of treatment.[30] Long-term use can lead to premature epiphyseal closure and radiologic bone evaluations may be required.[30]

Methotrexate

Methotrexate, a folic acid antagonist, is rarely used in children and reserved for severe psoriasis unresponsive to other treatments.[30,31] Side-effects include nausea, headache and gastrointestinal upset, which can be minimized with folic acid supplements.[9] Regular screening of the patient's blood count, liver enzymes, and renal function is necessary to monitor for potential development of acute hematotoxicity and hepatotoxicity.[2,9]

Cyclosporine

Cyclosporine is an immunosuppressant that can be used to treat extremely severe cases of pediatric psoriasis. The initial dose of cyclosporine is 3 mg to 5 mg/kg per day and should be gradually tapered to the lowest dose that can maintain disease control.[2,5] Major risks of hypertension and renal dysfunction necessitate close monitoring.

Biologics

Biologics are a class of drugs that include antibodies and fusion proteins targeting cytokines. Etanercept and infliximab are tumor necrosis factor-alpha inhibitors that are used for the treatment of pediatric autoimmune diseases. Etanercept is an effective method of treatment for moderate to severe plaque-type childhood psoriasis.[31,32] In a double-blind trial designed to assess the efficacy and safety of etanercept in children with plaque-type psoriasis, both non-infectious and infectious adverse effects from treatment were observed, the most serious of which were gastroenteritis and pneumonia.[32] All adverse affects were resolved without sequelae.[32]

Antibiotics and Tonsillectomy

Pharyngeal and perianal streptococcal infections may precipitate or exacerbate acute guttate and pustular psoriasis.[20] Antibiotics may be prescribed to treat patients with recurrence or flare of guttate psoriasis, and tonsillectomy may be considered for refractory psoriasis and recurrent tonsillitis.[30] However, these treatments are controversial, as there is a lack of controlled studies to support their efficacy.

Conclusion

Psoriasis is a life-long disease that often begins during childhood. In order to correctly diagnose and treat children and adolescents, it is important to recognize the different presentations of the disease in this cohort. Children with psoriasis, including their parents and caregivers, should be educated about the natural history and exogenous and endogenous factors responsible for increased disease morbidity, as well as receive support and counseling to help cope with their condition.

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Update on Drugs

Name/Company	Approval Dates/Comments
Dimethicone 50% topical solution *Nyda®* G. Pohl-Boskamp GmbH Pediapharm Inc.	Health Canada approved this new OTC pesticide-free pediculicide in September 2011 for the physical treatment of head lice infestation. The active ingredient, dimethicone, works by suffocating the lice, nymphs, and eggs. Due to its physical mode of action (interruption of the lice's oxygen supply of the central nervous system) development of resistance appears unlikely.
Radiofrequency aesthetic device *EXILIS Focused RF™* BTL Industries	Health Canada approved the marketing and distribution of this radiofrequency device in September 2011 for use in non-invasive body contouring, fat removal, wrinkle reduction, and skin tightening. It is the first focused radiofrequency technology in North America to receive approval for four aesthetic indications. This device has also received US FDA clearance for use in non-invasive dermatologic and aesthetic procedures with indications for non-invasive treatment of wrinkles and rhytids.
Skin barrier protectant *Neosalus® Lotion* Quinnova Pharmaceuticals	Neosalus® Lotion was launched in the US in November 2011 as a non-steroidal anti-inflammatory prescription product for the topical management of skin disorders, such as atopic dermatitis and allergic contact dermatitis. The key ingredients (dimethicone, glycerin, and stearic/palmitic acids) are formulated for skin barrier repair, protection, and hydration, as well as restoration of ceramides, cholesterol, and other free fatty acids.

Drug News

In October 2011, Centers for Disease Control and Prevention's Advisory Committee on Immunization Practices (ACIP) voted to recommend the routine use of the human papillomavirus (HPV) quadrivalent vaccine (Gardasil®) for boys aged 11 and 12 years. The ACIP also recommended vaccination for males aged 13 to 21 years who have not yet been immunized or have not completed the 3-dose series. Vaccinating males can confer protection from genital warts and HPV-related cancer, such as throat and anal cancers. This wider use of the vaccine may also prevent transmission of the HPV virus to females.
Available at: http://www.cdc.gov/media/releases/2011/t1025_hpv_12yroldvaccine.html

A recent article in the *Canadian Medical Association Journal** reports that a commonly prescribed antimicrobial compound containing trimethoprim and sulfamethoxazole (e.g., Septra®, Bactrim®, and generics) can potentially cause serious adverse reactions and calls for physician vigilance when prescribing. The most common adverse effects found were rashes and fixed drug eruptions, drug-drug interactions, and hyperkalemia. Rashes and fixed drug eruptions occurred in about 3% of patients. Serious and/or life-threatening adverse reactions (e.g., hyperkalemia, hypoglycaemia, and liver damage) have been reported and are usually the result of drug interactions.
*Ho JM, et al. Considerations when prescribing trimethoprim-sulfamethoxazole. *CMAJ* 2011 Nov 8; 183(16):1851-8.

In August 2011, the US FDA issued a Warning Letter citing Brazilian Blowout for safety and labeling violations. Formaldeyde, a known carcinogen, has been commonly found in a number of Brazilian style keratin-based hair straightening products tested. The letter lists health risks associated with inhaling formaldehyde when the product is used as directed. Reported adverse reactions include irritation/problems of the eyes and respiratory tract, headaches and dizziness, nausea, chest pain, vomiting, and rash. The letter also states that the labeling was misleading because the product is incorrectly labeled as "formaldehyde free" and neglects to warn of possible consequences from product usage under the conditions prescribed in the labeling.
Available at: http://www.fda.gov/ICECI/EnforcementActions/WarningLetters/ucm270809.htm

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