

Atopic dermatitis: an update review of clinical manifestations and management strategies in general practice

Sawsan Talib Jamal

Atopic dermatitis is the most common skin disease encountered in general practice. It is mostly prevalent in infancy and childhood, with a steady increase in incidence worldwide. The disease is typified by itching, dry, thickened skin: hence treatment is directed towards reducing itchiness and dryness using emollients, topical steroids and antihistamines. Patients with secondary bacterial or viral infections should be referred to a specialist as the condition is often serious and may even be fatal. Refractory cases need other modalities of drugs including systemic

steroids, phototherapy and immunomodulators. The treatment in such patients has to be initiated and monitored by the dermatologist. Exacerbation of attacks can be minimized through proper health education about avoidance of trigger factors, dietary manipulation and drug therapy.

Key words: dermatitis, xerosis, trigger factors, emollients, topical steroids

Bull Kuwait Inst Med Spec 2007;6:55-62

This article, *Atopic dermatitis: an update review of clinical manifestations and management strategies in general practice* is designated as CME/CPD. Readers who study it, answer the questions related to it on page 62, and send a copy of the Answer Sheet (page 92) to the CME Center of KIMS become eligible for 1 CME/CPD credit in Category 1 in the MPC Program of KIMS. To claim credit, the reader has to be Registered in the MPC Program, the answer sheet should be received by the CME Center before 31st May 2008, and all questions should have been attempted. Readers would then receive a certificate from the CME Center indicating the credit data.

Introduction

Atopic dermatitis (AD) is the most common chronic skin disease, affecting 15-20% of children in developed countries,¹ leading to a significant reduction in quality of life and a burden on health care resources.²⁻⁴ AD is defined as a chronic inflammatory condition of skin characterized by intense pruritis and a series of exacerbations and remissions.² Most of the patients could be managed by the general practitioner, with only a minority of 10-20% needing referral for secondary care by a specialist dermatologist.⁵ In one third of patients, the disease persists through adulthood.⁶ In most patients the condition is mild, but those with moderate to severe disease usually have intense itching and experience

loss of sleep. The social stigma of a visible skin disease can be soul destroying for both patient and family.¹

Pathogenesis

The pathogenesis of atopic dermatitis is unknown, but the disease seems to be the result of genetic susceptibility, immune dysfunction and epidermal barrier dysfunction.⁸ Recent studies impute a fundamental imbalance between the response of TH1 lymphocytes and TH2 lymphocytes. In response to antigen exposure TH1 lymphocytes activate interferon-g, interleukin-2 (IL-2) and tumor necrosis factor alpha. They also aid in recruitment and activation of monocytes, macrophages and cytotoxic T lymphocytes in the fight against intracellular pathogens. TH2 lymphocytes secrete interleukins IL-4, IL-5, IL-10. IL-4 stimulates the switching of B cells for IgE production, while IL-5 causes eosinophilia and IL-10 depresses T-cell mediated immune inflammation.⁸ It is the genetic predisposition of atopics to demonstrate systemic expansion of TH2 cells activity by a variety of immunological and non-immunological allergens. The most commonly reported trigger factors and allergens are heat, sweating, irritants (soap, harsh chemicals), humidity, stress and anxiety, certain foods, inhalant allergens and microbial agents such as Staphylococci, viruses, pityrosporum, Candida and dermatophytes.⁸ The exact role of

Head, Jaber Al-Ali Health Center, Ahmadi Health Region, Kuwait.

Correspondence: Dr. Sawsan Talib Jamal, Jaber Al-Ali Health Center, Ahmadi Health Region Primary Health Care, Ministry of Health, Kuwait.
Tel: (965) 3830402; Fax: (965) 3845446; email: Sjamal@yahoo.com

aeroallergens and food allergy is controversial due to limitations of the *in vitro* Radioallergosorbent Test (RAST) and Prick Skin Test. Both tests have a nearly 90% negative predictive value for atopic dermatitis, but their positive predictive value is less than 50% because of frequent false-positive results.¹⁰ Exposure to aeroallergens such as pollens, molds, mites and animal dander appears to be important in some patients. Clinical improvement may occur when these patients are removed from environments that contain the allergens to which they react.⁷

There is also increased evidence that neuropeptides may be involved in the pathogenesis of atopic dermatitis. Flushing of affected skin and pruritis are features of exacerbation. Several neuropeptides are identified as potent inducers of vasodilatation and pruritis. Scratching of skin releases substance P from cutaneous proprioceptor nerves, which induces the release of histamine from mast cells in the scratched area. Elevated concentrations of histamine are found in the skin and plasma of patients with atopic dermatitis.

Clinical Manifestations

The predominant symptom of AD is intense pruritis, usually with no fever or other constitutional symptoms.⁹ Pruritis can be so severe as to cause sleep disturbance, irritability and generalized stress for the affected patient and family.⁷ One of the most common presentations is a persistent rash in infants younger than 6 months. It is usually reported that there is dry skin since birth and the rash present on and off for a few months.³

The skin findings depend on the stage of disease:

- *Acute* – Erosions with serous exudate or intensely itchy papular rash and vesicles on an erythematous base.
- *Subacute* – Lesions characterized by scaling or plaques over erythematous skin.
- *Chronic* – Lesions recognized by the presence of lichenification and pigmentary changes⁷ with excoriated papules and nodules. Lesions may be secondarily infected due to scratching. Infected lesions present with yellow crusting or impetigo or the surrounding erythema characteristic of cellulitis.

Atopic dermatitis may present in other manifestations such as:

1. Ichthyosis vulgaris, which presents as hyperlinear palms and soles with polygonal fishlike scales, particularly on the lower legs;
2. Keratosis pilaris, asymptomatic horny follicular papules on the extensor surfaces of the upper arms, buttocks and anterior thighs;
3. Xerosis or dry skin, which leads to a tendency for cracks and fissuring with the resultant skin barrier breakdown increasing the susceptibility to irritation and infection;⁷
4. Keratoconus (a cone shaped cornea) in severe cases, which requires subsequent corneal transplant;
5. Periorbital findings, which include periorbital hyperpigmentation, prominent infraorbital fold (Dennie-Morgan folds), anterior subcapsular cataracts (in 4 to 12% of patients with AD, while posterior cataracts are usually a side effect of oral corticosteroids or of topical steroids used in the periorbital area).¹¹

Other associated features include facial erythema, perioral pallor, and pitriasis alba.

Table 1. Clinical features of atopic dermatitis

Major features

- Pruritis
- Chronic or relapsing dermatitis
- Facial and extensor involvement in infants and children
- Flexural and lichenification in older children and adults
- Personal or family history of atopy

Minor features

- Early age of onset after 2 months of age
- Xerosis
- Ichthyosis, hyperlinear palms, keratosis palmaris
- Anaerobic
 - Dennie-Morgan infraorbital folds
 - Anterior subcapsular cataracts
 - Keratoconus
- Nipple eczema
- Facial pallor or erythema
- Non specific dermatitis on hands and feet
- Cutaneous infection

DISTRIBUTION

In infants (3 months to 2 years), the cheeks, forehead, scalp, wrists and extensor aspects of the arms and legs are often involved. The

nappy area is usually spared. Scalp involvement may be severe enough to cause alopecia.¹² In children aged 2 – 12 years the flexor surfaces, neck, wrists and ankles are the usual sites. The lesion in teenagers and young adults is a rash on the flexor areas of arms (antecubital and popliteal), legs, face (especially periorbital region) and neck. The presence of extensor distribution in older children and adults indicates a poor prognosis for ultimate cure.¹³ Axillary, groin and intergluteal involvement is uncommon and should raise suspicion of some other causative factor. The diagnostic features are shown in Table 1. For the diagnosis to be established at least 3 major features and 3 minor features need to be present.¹⁴ The differential diagnoses are listed in Table 2.

Table 2. Differential diagnosis of atopic dermatitis

• Seborrheic dermatitis
• Psoriasis
• Neurodermatitis
• Contact dermatitis
• Ataxia-telangiectasia syndrome
• Histiocytosis –X
• Lichen simplex chronicus
• Photosensitivity rashes
• Icthyosis vulgaris
• Scabies
• Dermatitis herpetiformis
• Dermatophyte infection
• Systemic illnesses such as malignancies, thyroid disorders, hepatic or renal failure

Laboratory Findings

There are no specific laboratory findings or histologic features which can be relied on for diagnosis, hence the importance of history and physical examination.⁷ Elevated IgE levels are found in up to 80% of affected patients, but this result may be observed in other atopic disorders as well.¹⁵ Biopsy of skin shows a thickened and hyperkeratotic dermis with perivascular inflammation.²

Care and Management

Prevention is the mainstay of treatment of atopic dermatitis. This includes proper diagnosis, explanation and patient education about the natural history of the disease, the different types of drugs and the prognosis of the

condition. Given the chronic nature of the disease, emotional support and psychological counseling may be helpful. Patients should be informed that no cure exists, but that exacerbations can be minimized with optimal care.

ADVICE ON TRIGGER FACTORS

Patients and their parents should know the common trigger factors and try to avoid them.¹² The trigger factors include excessive bathing, hand washing, lip licking, sweating or swimming. Contact with solvents, deodorants, cosmetics and exposure to sunlight as well as wearing loose or poorly fitting clothes that constantly rub the skin can exacerbate the disease. Prolonged heat exposure, which includes hot showers, overdressing, use of hot pads and exposure to high humidity may lead to a flare up. Patients should be advised about the importance of showers or taking a short bath in lukewarm water with minimal or no use of soap on axilla, groin and feet. Moisturizing soft type soap is advised. Upon leaving the bath the patient should be patted dry with a soft towel, and within 2-3 minutes a copious amount of a moisturizer should be rubbed into the skin to seal in the water content.¹⁶ Rough towels should be avoided because they can provoke further irritation and itching. Intolerance to wool and other itchy fabrics is characteristic in atopic dermatitis, and these materials, therefore, should be avoided.¹⁶

Food can play a role in exacerbation of atopic dermatitis. A clear history of flare up after ingestion of certain foods warrants a trial of dietary manipulation under the guidance of a dietician.¹⁷ Examples of food types that induce allergic reactions are peanuts, eggs, fish and other sea food, milk and chocolate.

Eradication of house dust mite has shown to reduce disease severity under experimental conditions, but it is time consuming and no more effective than simple bed covers. Although the use of non-biological washing powder has been recommended by many, there is no evidence that it is better than its biological counterpart.¹⁷ Written information on AD and instructions on treatment should be given to patients or parents. Table 3 shows tips on key advice on eczema management. Table 4 gives general methods effective in reducing pruritis.

Table 3. Key advice on management of atopic dermatitis

- Protection against scratching
- Functions and importance of emollient
- Care of home environment to reduce flare-ups
- Appropriate clothing
- Use and effect of antihistamine
- Effect of sunlight on atopic dermatitis (holidays, swimming)
- Bathing, showering technique
- Psychological effect on patient and family
- Dietary manipulation (may be relevant in infants)

DRUG REGIMEN

The aim of drug treatment is to control skin abnormalities, xerosis, pruritis, superinfection and inflammation.⁷ There are several types of drugs available, ranging from simple emollients, topical steroids and antihistamines to the more potent remedies such as immunomodulators.

Table 4. General measures to reduce pruritis

- Finger nails trimmed short. Cotton gloves may be worn at night
- Bathe in tepid water, avoid soap and pat dry with soft absorbent towel
- Avoid woolen and acrylic clothing. Loose cotton clothes and blankets recommended
- Clothes should be washed with enzyme-free washing powders
- Bedroom kept cool

Emollients

Emollients soothe and relieve itch, producing an oily layer over the skin that traps water beneath it. This barrier restoration results in preventing the penetration of irritants, allergens and bacteria, thus reducing the development of atopic dermatitis. Soothing the skin also reduces the need for topical steroids. The liberal use of a good skin emollient is mandatory immediately after bathing and before skin is completely dry especially in winter.¹⁸ There are several types of emollients: lotions, creams and ointments. Greasy ones (baby oils) are preferred. Unfortunately, many people cannot tolerate ointments and prefer creams and lotions; the latter is least effective because of their alcohol content.⁷ Emollients can often be sufficient to provide relief in children with very mild eczema, especially when using greasier preparations such as soft paraffin or liquid paraffin.⁴ However, they are not effective during acute inflammatory flare ups when additional topical steroids will be needed. Severely affected skin can be optimally hydrated by occlusion in addition to applica-

tion of an emollient. Small areas can be occluded with a plastic free wrap, hands being covered with gloves. However, the physician must be careful because occlusion using topical steroids promotes systemic absorption of drugs, thus increasing the side effects.⁷

Sun Screen

Many people find that their dermatitis improves with exposure to sunlight while others have a worsening effect. Whatever the experience, the skin should have protection against the sun's harmful effects. A sun screen should be used prior to exposure to sun, and for patients with fair skin, a cream with sun protection factor (SPF) 15 or higher is suitable. An emollient has to be applied half an hour prior to application of the sun screen, and the patient should not remain in the sun for an excessive length of time.

Topical Steroids

Topical steroids are effective agents in managing atopic dermatitis but should not replace the use of frequent skin emollients. They are grouped into 7 potency groups, with Group 1 the most potent and Group 7 the least. A general rule in treatment with topical steroids is to use the least potent agents where possible and to limit the frequency of application. Compared with adults, children are at a high risk of the local and systemic side effects of topical steroids. The risk of side effects depends on steroid potency, amount used, concomitant use of occlusion, area covered and integrity of skin.

Table 5. Side effects of topical steroids

Local effects	Systemic effects
Skin atrophy	Adrenal suppression
Striae	Cataract
Telangiectasias	Glaucoma
Hypopigmentation	Growth retardation in children
Rosacea	Hyperglycemia
Perioral dermatitis	Hypertension
Acne	Osteoporosis

Skin penetration of steroid is greatest on groin and face, with it being the lowest on palms and soles.⁷ Table 5 lists the side effects of topical steroids.

It is reasonable to use mild steroids, e.g. Hydrocortisone 1% initially in infants and for intertriginous areas in patients of any age. In

severe cases a more potent type, e.g. Beta-methasone valerate is to be used with close monitoring and dosage reduced as skin improvement occurs. Strong agents should be reserved for areas with thickened plaques and for palms and soles. A current suggestion is to use short bursts of potent steroids followed by a holiday period of emollient only, which is as effective and safe as long treatment with low dose topical steroids.⁴

The different preparations of topical steroids include creams, ointments, lotions, gels and solutions. Thicker preparations (ointments) have better skin penetration. The potency of the steroid may differ depending on the form of preparation. Lotions and solutions are the most effective treatment form for scalp lesions. Topical steroid preparations containing antibiotics must be avoided because of the risk of development of bacterial resistance.

It is now routinely recommended that topical steroids should be used on a once daily basis⁴ as this will simplify plans for busy parents, halve the cost of treatment on health-care, and reduce the adverse effects of steroid therapy. Moreover, topical steroids and emollients should not be used simultaneously because the emollient may dilute or inactivate the therapeutic effect of the topical steroid, and possibly spread the steroid to non-affected areas: the preparations may be applied with a gap of one hour in between.

Patients with severe flare ups or weeping lesions may benefit from oral Prednisolone (40-60 mg/day for adults; 1 mg/kg/day for children). If a systemic steroid is used in the treatment of atopic dermatitis the potential of a rebound effect can be decreased by tapering its dosage while increasing the topical steroid and aggressively hydrating the skin.⁷

Antihistamines

Antihistamines are used to control itching. Their sedative effect is more beneficial than the antipruritic effect. This is mainly because the process in atopic dermatitis is histamine-mediated, which therefore would not respond well to histamine blockage. Additionally, patients tend to scratch while sleeping, and sedating them prevents scratching which plays a major role in the development of the rash. The older sedating agents are better than the newer non-sedating ones, but they can affect the child's ability to learn or the adult's ability

to drive and work.⁷ Antidepressants such as Doxepin and Amitriptyline have an antihistamine effect, inducing sleep and reducing pruritis.

Tar Preparations

Tar preparations have anti-inflammatory and antipruritic effects on the lesions of AD, but they should be used alone or with topical steroids.⁷ Some tar preparations (gels) may include alcohol, thus causing irritation. Shampoos, bathing solutions and creams are less irritating¹¹ and are available as over the counter preparations. Side effects include dark staining and strong odor, which may be dealt with by restricting their use to night and by covering the area of the skin.

Immunomodulators

Tacrolimus and Pimecrolimus are new topical preparations released in 2002 for use in the treatment of AD.⁵ These are similar to Macrolatum molecules in preparations that are currently used systemically to prevent graft rejection in liver and kidney transplant patients. They probably work by suppressing T lymphocyte responses by inhibiting calcineurin.¹ Unlike topical steroids they do not cause skin thinning, which is a major advantage in long-term use.¹

Tacrolimus seems to be equivalent in potency to topical steroid¹ as it is superior to weak preparations such as 1% Hydrocortisone. The current recommendation is for its use in the small proportion of patients with moderate to severe disease who have failed to respond adequately to conventional therapy. They are used mainly on the face or intertriginous areas where local side effects of topical steroids might be a problem.

The most common adverse effects of immunomodulators are burning sensation and pruritis following application. However, the frequency of such reactions seems to decrease after a few days as the lesions heal.

The concentration of the preparation for use in children is 0.03% although at present a 0.1% concentration, which was previously reserved for adults, is licensed. Treatment should be started as twice daily application and continued up to 3 weeks. This is followed by once a day application until clearance of the lesion, when it is discontinued. Improvement is usually seen within one week of treatment.

Emollients should not be applied to the same area within 2 hours of applying ointment. Additionally, Tacrolimus is not to be used in clinically infected areas.

Exposure to sunlight or other sources of UV light must be minimized or avoided as a precaution against the increased risk of skin cancers.⁵

As these drugs are immunosuppressive agents, they should be used only by a dermatologist and patients followed up with long-term surveillance for visceral and skin cancer.¹⁹

Other Immunomodulator Drugs

Cyclosporin is used in treatment of patients with severe disease in whom conventional therapy is ineffective. It works by inhibiting calcineurin. The dose is 2 mg/kg orally twice a day, to be increased gradually after one month but not to exceed 5 mg/kg/day. The drug has to be stopped if there is no response after 6 weeks. As the skin lesions improve, the dose is reduced by 0.5-1 mg/kg/day/month to the lowest effective dose for maintenance. PUVA or UV-B radiation should not be used with cyclosporine because of the risk of skin cancer.¹⁶ Monitoring renal and liver functions is mandatory.

Phototherapy

Phototherapy is beneficial in patients with extensive refractory disease.²⁰ UVA or UVB or combined psoralen with UVA is an option to be chosen by the dermatologist.

SECONDARY INFECTIONS

Bacterial infections are usually recognized by the presence of crusting of eczematous lesions, with pustules being less frequently encountered except in infections of the palms and soles.⁹ A subgroup may have infected lesions with diffused erythema of a characteristic beefy red color, which is suggestive of streptococcal infection. Such patients are at risk of developing acute glomerulonephritis. Any honey coloured crusting should be regarded as a clinically significant impetigo and treated with a systemic antibiotic that is effective against both staphylococci and group A beta hemolytic streptococci. Cephalexin (Keflex) or Cloxacillin can be used in the treatment of *Staphylococcus aureus*. For streptococcal infections Keflex is preferred in a dose of 1-4 g/day in divided doses in adults and 25-50 mg/

kg/day orally in 4 doses in children. Cloxacillin is given in a dose of 250-500 mg orally 6 hourly in adults and 50-100 mg/kg/day orally in 4 doses in children. Topical antibiotics are of no value in these patients. Topical therapy for secondarily infected skin can be accomplished by soaking or wrapping cloth saturated with aluminum acetate solution or saline solution.⁷

Viral infections are mainly herpetic in origin and present as vesicular lesions. Blisters are common, which often rupture leaving discrete punched out ulcers. Herpetic infections are much more widespread than typical herpes simplex in non atopic people. The lesions may be observed in flexures and genital areas.⁹ Infections with herpes simplex mandates the use of systemic antiviral agents because they are often severe and may result in disseminated disease that is life threatening.

Acyclovir (Zovirax) started within 24 hours of onset of rash and in a dose of 200-800 mg orally 4 times a day is recommended for adults and 5-20 mg/kg 4 times a day for children for 5-10 days.¹⁶ Referral to a hospital is considered in these patients irrespective of whether the infection is due to viral or bacterial agents.⁴

Prognosis

Most patients improve with a good therapeutic regimen. However patients and parents should both understand that no cure is available. Exacerbations are minimized by following the recommended prevention strategies. Approximately 90% of patients are completely cured by the time they reach puberty.¹² One third of patients may develop allergic rhinitis and another third asthma.¹⁶ It is rare for atopic dermatitis to continue into older ages, but when it occurs it is usually widespread and severe.⁹ Poor prognostic features include a family history of the disease, early disseminated infantile disease, female gender and coexisting allergic rhinitis and asthma.²⁰

Conclusion

Atopic dermatitis is a disease of children and young adults that is widely spread and increasing in its prevalence worldwide. The main feature is pruritis triggered by dry skin, with treatment aimed at reducing it by several management strategies. Management should start with education of patient and parents about trigger factors and the need of avoiding

them. Skin moisturization by emollients is an important procedure to control the condition. Topical steroids and oral antihistamines are of extreme importance as drug therapies.

Due to the chronic nature of the disease, patients are followed up by the family physician, with referral to a dermatologist considered for those with secondary infections that need hospital care or for refractory cases that require potent therapies such as immunomodulators and phototherapy.

References

1. Williams H. New treatments for atopic dermatitis. *BMJ* 2002;324:1533-4.
2. Williams HC. Epidemiology of atopic dermatitis. *Clin Exp Dermatol* 2000;25:522-9.
3. Herd RM, Tidman MJ, Prescott RJ, Hunter JA. The cost of atopic dermatitis. *Br J Dermatol* 1996;135:20-30.
4. Floh C, Williams HC. Evidence based management of atopic eczema. *Arch Dis Child Educ Pract Ed* 2004;89:ep35-9.
5. S Conroy. New products for eczema. *Arch Dis Child Educ Pract Ed* 2004; 89:ep23.
6. Wahn U, Bos JD, Goodfield M, Caputo R, Papp K, Manjra A, et al. Efficacy and safety of pimecrolimus cream in the long term management of atopic dermatitis in children. *Pediatrics* 2002;110:1-8.
7. Correale CE, Walker C, Murphy L, Craig TJ. Atopic dermatitis: A review of diagnosis and treatment. *Am Fam Physician* 1999;60:1191-210.
8. Leicht S, Hanggi M. Atopic dermatitis: How to incorporate advances in management. *Postgrad Med* 2001;109:119-27.
9. Motala C. Skin allergy Eczema and atopic dermatitis. In: The ALLSA Handbook of practical allergy. Available from: URL:<http://www.Allergysa.org/dermatitis.htm>.
10. Burks AW, James JM, Hiege IA, Wilson G, Wheeler JG, Jones SM, et al. Atopic dermatitis and food hypersensitivity reactions. *J Pediatr* 1998;132:1.
11. Boguniewicz M, Leung D. Atopic dermatitis: In: Middleton E Jr. *Allergy: principles and practice*. 5th ed. St. Louis: Mosby; 1998. p.1123-34.
12. Kass DA, Sinert R, Chidorzi AJ. Dermatitis, Atopic e-medicine. Available from: URL: <http://www.emedicine.com/emerg/topic130.htm>.
13. Lookingbill DP, Marks JG. Eczematous rashes. In: *Principles of dermatology*. Philadelphia: Saunders; 1993. p.127-30.
14. Barnetson RSC, Rogers M. Childhood eczema. *BMJ* 2002;324:1376-9.
15. Leung DY. Atopic dermatitis: the skin as a window into the pathogenesis of chronic allergic diseases. *J Allergy Clin Immunol* 1995;96:302-18.
16. Cooper KD. Atopic dermatitis; recent trends in pathogenesis and treatment. *Invest Dermatol* 1994;102:128-37.
17. Smethurst D, Macfarlane S. Atopic eczema. Clin Evidence 2002. Available from: URL: <http://www.Clinicalevidence.com>.
18. FDA Center for Drug Evaluation and Research. Elide (pimecrolimus) cream. Available from: URL: <http://www.Fda.gov/cder/foi/nda/2001/21-302Elidel.htm>.
19. Fitzpatrick TB. *Dermatology in general medicine*. 4th ed. New York: McGraw-Hill; 1993.

CME/CPD Questions

After you have completed reading the article *Atopic dermatitis: an update review of clinical manifestations and management strategies in general practice*, take the test given below. Circle T (True) or F (False) in the Answer Sheet on page 92 to show the correct answer to each question. Questions 1 to 10 are related to the content in this article.

1. Atopic dermatitis is the most common skin disease encountered in general practice.
2. The chronic form of atopic dermatitis is characterized by serous exudates with papular rash and vesicles on an erythematous base.
3. Posterior cataracts develop usually as a side effect of systemic steroids or use of topical steroids on periorbital area.
4. Axillary, groin, and intergluteal involvement is common in atopic dermatitis.
5. Dietary manipulation is indicated if there is a clear history of flare-up of rash after ingestion of specific types of food.
6. As a general rule in infants, treatment should start with the most potent topical steroid and changed to mild forms when the rash clears up.
7. Topical steroid antibiotic combinations must be avoided because of the risk of the development bacterial resistance.
8. Tacrolimus and Primecrolimus are safe to be used on infected areas in atopic dermatitis.
9. Poor prognostic factors include family history, early-disseminated infantile disease, female gender and coexisting allergic rhinitis and asthma.
10. The therapeutic effect of antihistamines in controlling pruritis in atopic dermatitis is primarily due to their sedative action and reduction in itching.