

General Principles Of Eczema/Dermatitis

(General Principles Of Eczema/Dermatitis & Atopic
Dermatitis)

Objectives:

1. To know the definition & classification of Dermatitis/Eczema
2. To recognize the primary presentation of different types of eczema
3. To understand the possible pathogenesis of each type of eczema
4. To know the scheme of managements lines

Team leader:

Mohsen Almutairi

Done by:

Faisal ALsubaie

Mohammed Alhumud 

Lama Alyahya

Taibah Alzaid

Noura Alturki

Color index:

 Important

 Doctors Notes

 Extra

Contact us:

Dermatologyteam438@gmail.com



[Editing file](#)

Eczema & Dermatitis

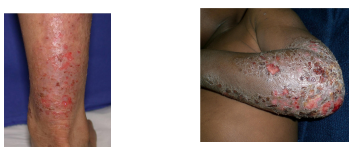



Eczema:

Definition:

- Inflammation of the skin.

Eczema VS Dermatitis:

- Eczema is a general word.
- Atopic dermatitis is a specific type of eczema and there are other types (contact dermatitis and others).

Eczema		
Acute eczema	Subacute eczema	Chronic eczema
<p>Erosion, oozing and vesicles.</p> <p>The primary lesion of eczema is vesicles</p> <ul style="list-style-type: none"> - Oozing = "Fluids"  <p>Acute red erosions on top of chronic with crust</p> 	<p>Redness + Swelling crust + Scale + infection</p> 	<p>Lichenification, dark pigmentation and thick papules and plaques.</p> <p>Lichenification: Thickening of skin due to chronic rubbing, increase skin marking</p>  <p>Child with adult pattern (neck)</p>

Dermatitis types:



Atopic	Seborrheic	Contact	
		Allergic	Irritant
Nummular	Asteatotic	Stasis	Neurodermatitis/ Lichen Simplex Chronicus

Atopic Dermatitis (AD):

Introduction

Definition: **chronic relapsing** itchy skin disease in genetically predisposed patients e.g: family Hx of asthma, nose or eye allergy.

Associated diseases: **bronchial asthma, allergic rhinitis, allergic conjunctivitis.**

Incidence: up to 15% in developed countries. **Up to 15% - 20% in early childhood.** More in males

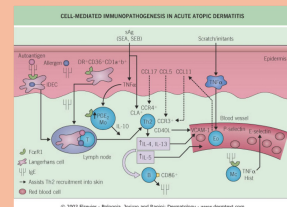
Age of onset: 60% within the first 2 months, 30% by the age of 5, 10% between age of 6-20

Grow out tendency!

Multifactorial;

- **“Atopy”:** genetic predisposition
- **Dry (atopic) skin** (decrease human B-defensin 3 predisposing patients to frequent skin infections : colonized by S aureus. infection with S aureus often causes a flare of AD) (decrease production of moisturizing lipids; sebum)
- **Allergy, increased tendency to certain allergens. AD and Food! minor role**
- **Mutation of FLG (encodes filaggrin) disturbed skin barrier function** dry skin, decrease moisturizing lipid production
- T-Cell (elevated Th2 cytokines and increased IgE production.
- Recent studies showed a potential role for the Th17 pathway, with increased circulating Th17 cells in atopic patients, & increased Th17 in acute eczematous lesions. A decreased Th17 in chronic eczema argues for a dynamic role for the Th17 pathway.

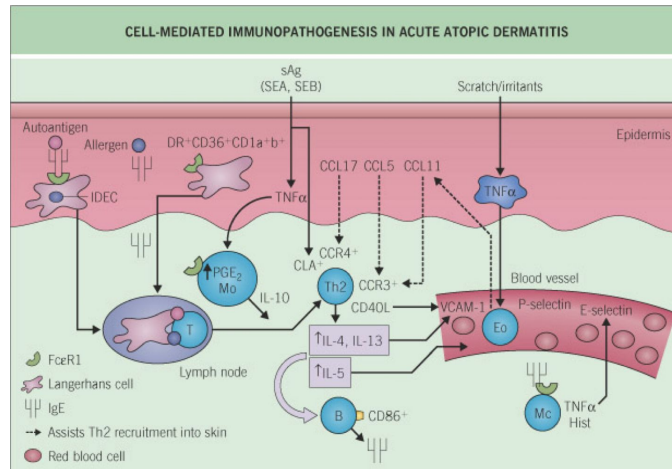
Pathogenesis



Prevalence and association with other atopic disorders

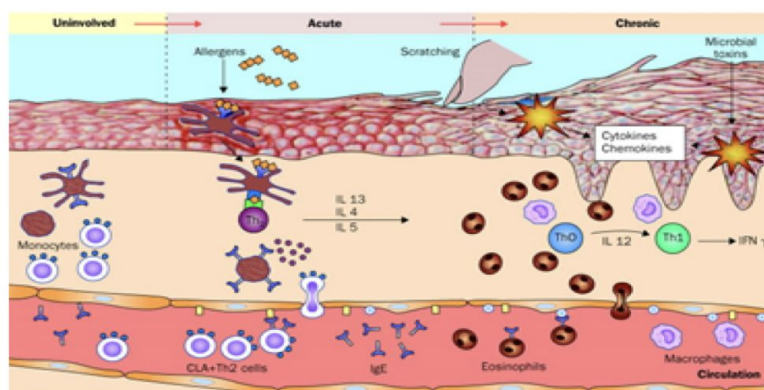
- Prevalence is almost 20% in US, representing a marked increase during the past several decade.
- Studies before 1960 estimated the prevalence to be up to 3%.
- **AD is often the 1st manifestation of the “atopic march”;**
 - **AD → Asthma → Allergic rhinitis**
 - Asthma occurs in up to 50% of children who develop AD during the first 2 years of life;
 - Allergic rhinitis develop in 43-80% of children with AD.
 - In general children showing more severe dermatitis have a higher risk of developing asthma, as well as sensitization to foods and environmental allergens.
- **AD occurs more frequently in urban areas than in rural areas, in smaller families, and in higher socioeconomic classes.**
- Ultimately 80% of patients will develop increased IgE levels.
- **Loss-of-function mutations in profilaggrin (FLG) (induce moisturizing of the skin) cause ichthyosis vulgaris, a common genetic disorder characterized by dry, scaling skin and hyperlinear palms that has long been known to be common in individuals with AD.**
- **Distinct mutations in FLG** have been discovered in the European and Japanese populations, but all are strongly linked with AD, particularly of early onset.

Notes on Pathogenesis



© 2003 Elsevier - Bologna, Jorizzo and Rapini: Dermatology - www.dermtext.com

- The immune system is divided into 2 parts: Innate (langerhan cells, antigen presenting cells(APC),etc..) & Acquired.
- Firstly the APC receives antigen “Recognition”(viral, bacterial,allergen, auto-antigen)
- The innate immune system is always first to react (through adhesion molecules, cytokines..) if not enough-> activation of the acquired immune system.
- APC and T Cells will meet in the **lymph node**-> **APC** presents the allergen antigen and **ACTIVATE T helper 2 (Th2)** (responsible for allergic reaction)
- Th2 will:
 1. Increases IL-5: Increases Eosinophils (migrate from blood to epidermis)
 2. Increases IL-4 & IL-13: Activate the Vascular-Endothelial system > Inflammation (WBC, Cytokines..etc).
- usually: IgM-> acute infection, IgG-> chronic infection, in this situation (allergy) the blood IgE will increase in the chronic phase
- persistence of inflammation is why we call it “ immunodysregulation”.



- as time passes the immune system will try to balance the high Th2 => Th1 will increase and Th2 will decrease.
- when Th1 appears (chronic eczema) it is often mixed with psoriasis (both have Th1 activation).

- Allergic reaction types:

- type 1 => urticaria (tested w skin prick test takes 15-30min)
- type 4 => delayed hypersensitivity reaction (skin patch test requires 48-72hr)




Atopic Dermatitis (AD):

Histology

- **Edema within the epidermis (spongiosis)** and infiltration with lymphocytes and macrophages in the superficial dermis (no need to take a biopsy in AD).

Clinical Variants of Atopic Dermatitis (AD) :

according to morphology when they first appeared

<p>Infantile Atopic Dermatitis</p>	<ul style="list-style-type: none"> ● 60% of case AD present in the first year of life, after 2 months of age. ● Begin as itchy erythema of the cheeks. ● Distribution: <ul style="list-style-type: none"> ○ Includes scalp, neck, forehead, wrist, and extensors. ○ Diaper area is usually spared ● Well demarcated, ill defined (eczema always ill defined, psoriasis well defined), erythematous plaques (raised cause of inflammation), crusty and non-scaley. ● Red skin, tiny vesicles on "puffy" surface. Scaling, exudate with wet crust and fissures. 	 <p>Baby looks well</p>
<p>Childhood Atopic Dermatitis</p>	<ul style="list-style-type: none"> ● Characterized by less acute lesions. ● Distribution: <ul style="list-style-type: none"> ○ Antecubital and popliteal fossae, flexor wrist, eyelids, and face. ● Severe atopic dermatitis involving more than 50% of body surface area is associated with growth retardation. ● May be generalized ● Papular, lichenified plaques, erosions, crusts. 	
<p>Adult Atopic Dermatitis Can be localized or generalized</p>	<ul style="list-style-type: none"> ● Distribution: <ul style="list-style-type: none"> ○ Antecubital and popliteal fossae, the front side of the neck, the forehead, and area around the eyes. ● Atopic individuals are at greater risk of developing hand dermatitis than are the rest of the population. ● 70% develop hand dermatitis some times in their lives. ● Ill demarcated hyperpigmentation with lichenification. 	 <p>This is Erythroderma, a very rare complication of atopic dermatitis.</p>

Atopic Dermatitis (AD):

Sequelae

- Atopic individuals have a distinct tendency toward an extra line or groove of the lower eyelid, so called "atopic pleat", is present at birth or shortly after and usually retained throughout life, referred to as "Dennie-Morgan fold".
- Another feature, an **exaggerated linear nasal crease**, caused by frequent rubbing of the nasal tip (allergic salute), although not a specific sign of AD.

Complications of Atopic Dermatitis:

Secondary infections



- **Impetigo**, a bacterial infection caused by *S. Aureus* or streptococcus.
- If seen assume infection over subacute state and take swab, start Abx

Eczema herpeticum

Eczema Herpeticum is a serious complication caused by herpes simplex virus that needs admission and systemic antiviral (IV acyclovir) analgesia and an ophthalmologist.



Post Inflammatory Hyper/Hypo-pigmentation (PIH)



Cellulitis



Growth retardation

Psychological

Table 5.1. Revised criteria for the diagnosis of atopic dermatitis⁴

- a. Must have:
- Pruritus
- b. Plus 3 or more of the following:
- History of involvement of skin creases (front of elbows, back of knees, front of ankles, neck, around the eyes)
 - History of a generally dry skin in the past year
 - Personal history of asthma or hay fever
 - Onset under the age of 2 years
 - Visible flexural dermatitis

The diagnosis of atopic dermatitis in adults is primarily clinical; special investigations only contribute in identifying external aggravating factors.

DIAGNOSTIC FEATURES OF ATOPIC DERMATITIS

Major features (3 of 4 present)

- Pruritus
- Typical morphology and distribution of skin lesions
- Chronic or chronically relapsing dermatitis
- Personal or family history of atopy

Minor features (3 of 23 present)

- Xerosis
- Ichthyosis/palmar hyperlinearity/keratosis
- Immediate (type I) skin test reactivity
- Elevated serum IgE
- Early age of onset
- Tendency towards cutaneous infections/impaired cell-mediated immunity
- Tendency towards non-specific hand or foot dermatitis
- Nipple eczema
- Cheilitis
- Recurrent conjunctivitis
- Dennie-Morgan infraorbital fold
- Keratoconus
- Anterior subcapsular cataract
- Orbital darkening
- Facial pallor/erythema
- Pityriasis alba
- Anterior neck folds
- Pruritus when sweating
- Intolerance to wool and lipid solvents
- Perifollicular accentuation
- Food intolerance
- Course influenced by environmental/emotional factors
- White dermographism/delayed blanch

جلد السمك

2 of 4

In hypothyroidism

التقرن (جلد الدجاج)، الفصام

مقاربة وقاسية

Atopic Dermatitis (AD):

Investigations

- Atopic dermatitis is purely clinical; No investigations are needed.

Management

Management of Atopic Dermatitis (AD):

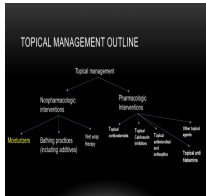
Education! Education! Education!

Psychological Support!

Skin care:

- Moisturizing the skin.

Topical therapy (topical steroids, Tacrolimus, Pimecrolimus)



Class	Potency	Examples (Not exclusive)	Caution
1	Superpotent	Clobetasol Propionate, Halobetasol Propionate	Avoid in children, face, folds, near genitals.
2	Potent	Betamethasone dipropionate, Halcinonide, Flucinonide	Avoid in children, face, folds, near genitals.
3	Upper Mid Strength	Betamethasone Valerate	Use cautiously in children. Avoid on face, folds, near genitals.
4	Mid Strength	Mometasone, Beclomethasone, Flucinolone 0.25%, Triamcinolone acetonide, Methyl Prednisolone acetonate	They may be used in children for a short duration. Not to be used on face and body folds.
5	Lower Mid Strength	Hydrocortisone butyrate, Fluticasone propionate	Can be used in all ages and on the face and folds for a short duration.
6	Mild	Desonide, Flucinolone 0.1%	Can be used in all ages and on the face and folds for a short duration.
7	Least Potent	Hydrocortisone 1%	This can be used in all, the only one that may be bought over the counter.

Local cutaneous side-effects

- Atrophy
- Striae
- Periorificial granulomatous dermatitis
- Acne
- Telangiectasia
- Erythema
- Hypopigmentation
- Ocular effects
 - Cataracts
 - Glaucoma
- Systemic side-effects
 - Hypothalamic-pituitary-adrenal axis suppression

Topical steroids:

Phototherapy

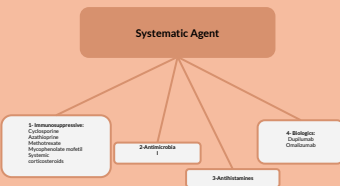
Sedative antihistamine (Oral H1 antihistamine) to control itching and help sleep

Antibiotics--- Antistaphylococcal drugs

Systemic therapy: Steroids, Cyclosporin, Methotrexate, Azathioprine

New biologics such as dupilumab

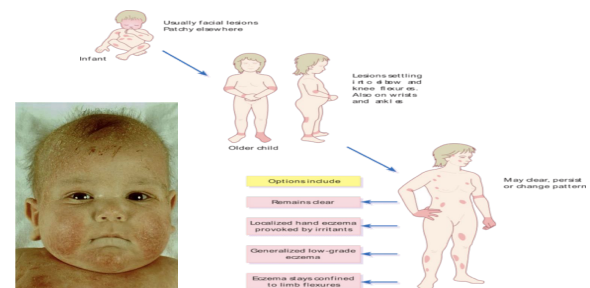
Pharmacological intervention



- Topical calcineurin inhibitors level of recommendations is A
- Efficacy:
 - Tacrolimus ointments(0.03% and 0.1% strengths)
 - Approved for moderate to severe disease
 - 0.1% as effective as the mid potency TCS hydrocortisone butyrate 0.1%.
 - 0.03% is less effective than hydrocortisone butyrate 0.1% but more effective than the low potency TCS hydrocortisone acetate 1%
 - Pimecrolimus cream (1% strength)
 - indicated for mild to moderate AD
 - Less effective than mid and high potency TCS direct compression made

Prognosis

- Half of the cases improve by 2 years of age. Most improve by teenage years
- <10% of patients have lifelong problems
- 30-50% will develop BA or hay fever



Nummular Dermatitis & Regional Eczema

Nummular dermatitis

- Coin shaped patches and plaques.
- Secondary to xerosis cutis.
- Primary symptom itch.



Notice the surrounding xerosis

Regional Eczema

Regional Eczema:

<p>Ear eczema</p>	<ul style="list-style-type: none"> • Most frequently caused by seborrheic or atopic dermatitis. • Staph, Strep, or Pseudomonas. • Earlobe is pathognomonic of nickel allergy. 	
<p>Nipple eczema</p>	<ul style="list-style-type: none"> • Painful fissuring, seen especially in nursing mothers • Maybe an isolated manifestation of atopic dermatitis. • If it persists more than 3 month, and/or unilateral, biopsy is mandatory to rule out Paget's disease. 	
<p>Hand Eczema</p>	<ul style="list-style-type: none"> • Spongiosis histologically. • Irritant hand dermatitis seen in homemakers, nurses. • Resulting from excessive exposure to soaps. • Pompholyx- tapioca vesicles, on sides of fingers, palms, and soles • Irritant vs allergic. 	
<p>Juvenile plantar dermatosis</p>	<ul style="list-style-type: none"> • Begins as a patchy symmetrical, smooth, red, glazed macules on the base of the great toes • Affect age 3 to puberty. • Symmetrical lesions on weight bearing area • Virtually always resolve after puberty <p>Bilateral III defined erythematous shiny plaques with scales and fissures More in summer, heat, sock wearing Can happen in palm too (palmo-plantar dermatosis); problem with sweat glands. Treat with proper ventilation and moisturizer</p>	<p>Ddx: scabies.</p>

Eyelid dermatitis

Diaper dermatitis

Xerotic eczema & Contact Dermatitis

Xerotic / Asteototic eczema

- Aka: winter itch, nummular eczema, eczema craquele, and asteototic eczema.
- Anterior shins, extensor arms, and flank
- **Elderly person predisposed.**
- Use of bath oils in bath water is recommended to prevent water loss
- Moisturizers – urea or lactic acid.



Contact Dermatitis

Definition: dermatitis results from contact with external materials.

Pathogenesis:

Irritant like detergent, chemicals, acid vs. allergic dye, henna, latex : (cytotoxic vs type IV)

Common irritants: detergent, acids, dust, burning chemicals, etc

Common allergens: perfumes, hair dyes, nickels, leathers, metals, rubbers, latex, cosmetics, etc

Allergic contact dermatitis

- **Type 4 Hypersensitivity Response.**
- Classically well demarcated/patterned.
- **First exposure does not cause a reaction**
- Begins 24 h after subsequent exposure if already allergic
- Exposure can be infrequent (once a month).
- **Commonest: Nickel**, chromates, rubber, preservatives, topical Abx, topical cs
- **Patch testing is gold standard for diagnosis** (read at 48, 96 h)

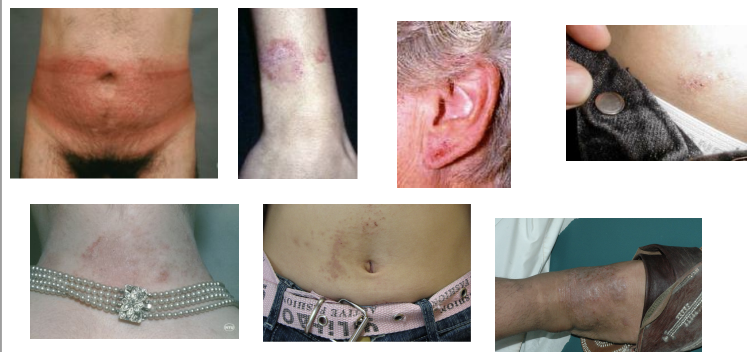
Management

- Identification removal of causes.
- **Patch testing:**

for allergic contact dermatitis not for irritant

Avoidance allergens

Topical corticosteroids



TOP TEN ALLERGENS AS IDENTIFIED BY THE NORTH AMERICAN CONTACT DERMATITIS GROUP		
Test substance	Allergic reactions (%)	Relevant reactions (%)
Nickel sulfate	14.2	49.1
Neomycin sulfate	13.1	46.2
Balsam of Peru	11.8	82.9
Fragrance mix	11.7	86.9
Thimerosal 10.9		16.8
Sodium gold thiosulfate 9.5		40.6
Formaldehyde 9.3		63.2
Quaternium-15 9.0		88.7
Cobalt chloride 9.0		55.1
Bacitracin 8.7		50.4

Xerotic eczema & Contact Dermatitis

Irritant contact dermatitis

- All people will react to an irritant if applied in a high enough concentration
- At 1st exposure
- **Most contact dermatitis is irritant in nature.**
- Occupational morbidity.
- Prevention is key!

Common causes:

- Hands repeatedly exposed to water, cleansers
- Lip-licking habit – wetting and drying caused by saliva
- Napkin dermatitis



IRRITANTS AND MECHANISMS OF TOXICITY	
Irritant	Mechanisms of toxicity
Detergents	Solubilization and/or disruption of barrier lipids and natural moisturizing factors in the stratum corneum Protein denaturation Membrane toxicity
Acids	Protein denaturation Cytotoxicity
Alkalis	Barrier lipid denaturation Cytotoxicity through cellular swelling
Oils	Disorganization of barrier lipids
Organic solvents	Solubilization of membrane lipids Membrane toxicity
Oxidants	Cytotoxicity
Reducing agents	Keratolysis
Water	If barrier is disrupted, cytotoxicity through swelling of viable epidermal cells

Shoe dermatitis

Causes:

- Rubber (most common)
- Chromates (in leather)
- Glutaraldehyde (in leather)
- Adhesives
- Dyes

Clinical features

- Predilection sites: site of contact
- Distribution & configuration



Dyshidrotic Eczema



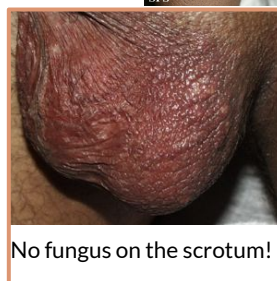
Stasis Eczema



Neurodermatitis/Lichen Simplex Chronicus & Seborrheic Dermatitis

Neurodermatitis/Lichen Simplex Chronicus

- Paroxysmal pruritus.
- Habitual excoriating or rubbing.
- Skin thickens to defend.
- Consider underlying disease.



Seborrheic Dermatitis

- **Definition:** redness and scaling in regions where the **sebaceous glands** are most active as the face, scalp, **presternal area and body folds**. **Very common chronic dermatosis**.
- **Age:** infancy, puberty, old age. More in **male**
- **Pathogenesis:**
- Increased Sebum! (seborrheic state), Tendency, **Pityrosporum ovale (Malassezia furfur)** over growth, More in Parkinson, HIV/AIDS patients.
- **Clinical features / Presentation:**
- Pruritus is variable. Gradual onset, worse in winter dry environment.. **Orange-red greasy scaling macules**, papules of varying size. **Trunk:** nummular, annular. **Scalp:** marked scaling, diffuse involvement
- **Distribution:**
- Hairy are of head, cradle cap
- Face: forehead, nasolabial folds, glabella and eyebrows.
- Trunk: DDx: PR vs pityriasis versicolor
- Body folds: axillae, groins, anogenital area, submammary areas, umbilicus and diaper area (infants)--- sharply marginated erythematous eruption, erosions and fissures
- Genitalia: with yellow crust and psoriasiform lesion
- **Management**
- Scalp :
- Zinc pyrithione Shampoo
- Selenium sulfide 2.5% shampoo
- 2% ketoconazole shampoo **anti fungal**
- Low - potency glucocorticoid solution, lotion or gels.
- Skin:
- Topical: antifungals, glucocorticoid, pimecrolimus
- Combined therapy
- Maintenance & recurrence

Cradle cap of oily scales on red scalp



Questions

1- One-year old boy known to have atopic dermatitis presented to the emergency department with 1 day history of eruptive painful vesicles and crusted erosions over face. What is the most likely diagnosis?

- A) Impetigo
- B) Eczema herpeticum
- C) Pityriasis versicolor
- D) Allergic contact dermatitis

2- A 6 months old infant had been very itchy, presented with Eczematous Eruption. He was diagnosed with Atopic Dermatitis. Which one of the following is the most common site distribution for the above patient of this disease?

- A) Diaper Area
- B) Scalp
- C) Neck
- D) Face

3- One-year-old boy known to have atopic dermatitis presented to the emergency department with a one-day history of painful vesicles and crusted erosions over his face associated with fever. How will you treat this patient?

- A) Topical steroid
- B) Oral antibiotics
- C) Oral Steroids
- D) Systemic antiviral

4- Infant with dermatitis, diarrhea & hair loss. management?

- A) Oral antibiotics
- B) Zinc supplement
- C) Topical steroids
- D) Systemic steroids

5- What would you recommend to the parents of a child who was diagnosed with atopic dermatitis ?

- A) Drinking fluids to prevent dehydration
- B) Using topical steroids every day
- C) Taking antibiotics to prevent infections
- D) Using moisturizers

6- A-55-years-old female who works as a hairdresser presented with hand eczema. Which of the following best describes allergic and irritant contact dermatitis?

- A) Patch test will be positive in irritant contact dermatitis
- B) Irritant contact dermatitis is caused by delayed type hypersensitivity reaction
- C) Allergic contact dermatitis occurs in previously sensitized individuals
- D) Allergic contact dermatitis is non-immunologically mediated

Answers:

1 : B, 2 : D 3 : D, 4 : B 5 : D, 6 : C