Eczema and dermatitis

Prof. Dr. Khudair K. Al.Kayalli
College of medicine Diyala University , 2016-2017

Eczema :-

Definition- eczema is an inflammatory skin reaction characterized histologically by spongiosis with varying degrees of acanthosis and superficial perivascular lymphohistiocytic infiltrate , with clinical features including : itching , scaling and clustered papulo-vesicles , with a wide range of external and internal factors acting singly or in combination , can induce the condition .

Although the terms dermatitis and eczema are nowadays generally regarded as synonymous , so that all eczema is regarded as dermatitis but not all dermatitis is eczema .

Classification- eczema is classified into groups :-

A. <u>Exogenous eczemas</u> – are related to clearly defined external triggering factors , although inherited tendencies can also play a part , this group include :

1. Irritant contact dermatitis (ICD) .
2. Allergic contact dermatitis (ACD) .
3. Photo-contact dermatitis .
4. Eczematous polymorphic light eruption .
5. Infective dermatitis .
6. Dermatophytide .
7. Post-traumatic eczema .
8. Exanthematous drug eruption .

B. <u>Endogenous eczema</u> – it implies that the condition is not a result of exogenous or external environmental factors , i.e. is mediated by processes originating within the body , include :

1. Atopic dermatitis (AD) .
2. Seborrhoeic dermatitis .
3. Asteatotic eczema .
4. Discoid eczema .
5. Exudative discoid and lichenoid dermatitis .
6. Chronic superficial scaly dermatitis .
7. Pityriasis alba .
8. Hand eczema and pompholyx .
9. Gravitational eczema .
10. Juvenile planter dermatitis .
11. Metabolic or eczema associated systemic diseases .
12. Chronic hyperkeratotic palmer eczema .

In some types of eczema , both exogenous and endogenous are important in the aetiology .

Prevalence- in UK in Belfast 25% of patients with dermatoses had dermatitis , (15% hand eczema , 12%CD , 11%seborrhoeic , 7%discoid , 6%lichen simplex , 5%AD , 4%gravitational , 40%others) . In Iraq (Diyala Province) , the prevalence of dermatitis was 20% out of all dermatoses , (49% had hand eczema and CD , 21%AD , 10.5%seborrhoeic , 4%lichen simplex , 3%napkin dermatitis and 8.5%others) .

Certain patterns of eczema can be seen more commonly in particular age group , e.g. AD mostly in infancy and young children , discoid astestotic eczemas occurs particularly in elderly .

***Histopathology*-** is according to the stage of eczema :

a. **Acute form –** shows , spongiosis with formation of vesicles , acanthosis , variable infiltration of the epidermis by lymphocyte , with dense superficial lymphocytic dermal infiltration with histiocytes .
b. **Subacute form –** spongiosis diminished , increasing acanthosis , with formation of a parakeratotic horny layer , moderate dermal lymphocytic infiltrate with histiocytes and decrease epidermal infiltrate .
c. **Chronic form –** there is arythrokeratotic hyperkeratosis with areas of parakeratosis , marked acanthosis , elongation and broadening of the rete ridges , mild chronic dermal lymphocytic-histiocytic infiltrate and absence of epidermal infiltrate e.g. lichenification .

***Pathogenesis*-** it involves the interaction between three things : **a. Triggering factor , b. Keratinocytes , c. T-lymphocytes ,** which seems particularly important in most eczema types , e.g. in **ACD** , cell mediated immune response (type-4) is enhanced after exposure to trigger factor (haptens) , which induce epidermal damage (T-cell-mediated cytotoxicity) , in particular phase –induced keratinocyte apoptosis , which may be a final common path way in many types of eczema . **In ICD** , the three predominant processes are : ***disturbed barrier function , *epidermal cell damage and *release of inflammatory mediators and cytokines** , which induce morphological changes apparent histologically and clinically as eczema .

Secondary dissemination :

A very characteristic feature of eczema is it's tendency to spread far from it's point of origin . This is especially likely when the primary site of the eczema on the legs or the feet , the eczema may have been present for only a few days , or for many years , before dissemination occurs . The dissemination which is often preceded by an exacerbation at the primary site , usually occurs explosively . The secondary eruption may at first consist of small oedematous papules , but these soon become obviously eczematous and grouped papulo-vesicles may become confluent in small plaques , occasionally the lesions take the form of red macules or weals , the distribution is usually symmetrical .

The **course** of the secondary eruption depends largely on the progress of the primary lesion , if the primary lesion remains acutely inflamed , the eruption increases in severity and may become generalized . If the patient is rested and the local lesion allowed to settle , the secondary eruption will subside , but will often recur very readily if the local lesion relapses , in small proportion of patients , the generalized secondary eruption evolves into an erythroderma , which may become self-perpetuating .

There are four main mechanisms of dissemination :

1. **Spread by contact with external allergen –** i.e. as a result of contact of new areas of the skin with a specific external allergen , the eczematous response is a symmetrical and it's progress is irregular , e.g. a contact dermatitis of the lower leg induced by lanolin ointment , may spread to the hand and the face as a result of casual contact during application of the ointment , other e.g. use of topical medication in chronic venous eczema of the lower leg lead to secondary dissemination , with positive patch test in over 90% of cases (allergy to topical therapy) .

2. **Spread by ingestion or injection of an allergen –** i.e. in other cases , an eruption originally induced by sensitivity to topical allergen (e.g. ACD) may relapse after ingestion or injection of the same chemical , e.g. medication that can be used topically or systemically . The eruption tends to be wide spread and more or less symmetrical and is usually of sudden onset , the diagnosis may be suspected when the eczematous eruption dose not conform to the recognized pattern of endogenous eczema , yet can not be related to external contact or to dissemination from a primary focus .
3. **Conditional hyperirritability ('angry back' syndrome) –** this term refers to the phenomenon where by an area of inflamed skin on one part of the body results in generalized hyperirritability of the skin sites , that are distant from the primary site of inflammation . There is considerable evidence that eczematous patients are more vulnerable to mild primary irritants than normal people , but the increased reactivity dose not persist after the eczema subside . The 'angry back' syndrome is clinical phenomena in which a strongly positive patch –test response can increase the percentage of false –positive reactions on the back at the same time , this suggests that an abnormal cell-mediated immune response could be occurring .
4. **Bacterial hypersensitivity –** heavily infected eczema will some times disseminate in the absence of demonstrable allergic sensitivity to topical medication , it is probably that allergy to bacteria or their products is some times a factor in the dissemination .

Infective dermatitis (microbial eczema):

Definition- eczema that is caused by microorganisms or their products , which clears when the organisms are eradicated . This should be distinguished from infected eczema , in which eczema resulting from some other cause is complicated by secondary infection (bacterial or viral) .

Pathology- is generally that of subacute or chronic eczema , in which spongiosis is combined with acanthosis , hyperkeratosis and patchy parakeratosis , dermis shows inflammatory infiltrate of polymorphnuclear and lymphocytic cells that invade the epidermis to a variable extent .

Pathogenesis- the mechanism by which the microorganisms causes eczema is not understood , but the possibility that bacterial antigens may play a role .

Clinical features- the distinction between infective and infected eczema may be difficult .

Infected eczema – shows erythema , exudation and crusting , the exudation may be profuse with crusting , or slight , with accumulation of layers of somewhat greasy moist scale beneath which the surface is raw and red , with sharply defined margin and may be small pustule in the advancing edge , in flexures a deep and persistent fissures .

Infective eczema- usually presents as an area of advancing erythema , some times with micro-vesicles , seen predominantly around discharging wounds or ulcers , or moist skin lesions of other types . It is relatively common in patients with venous leg ulcer , which must be distinguished from contact dermatitis caused by topical therapy , other type of infective eczema , this that occasionally develop around lesions of molluscum contagiosum , interdigital spaces on the dorsum of the medial toes , tinea pedis may also become eczematous because of the over growth of Gram-negative organisms , chronic thread worm infestation , pediculosis or scabies .

Treatment- factors predisposing to infection should be sought and when possible eliminated . Mild forms are treated by topical antibacterial agents , in sever or wide spread infections systemic antibiotics are used , with topical wet compresses (e.g. K. permanganate solution) .

Dermatophytide :

Definition- it is an allergic reaction (response) to a dermatophyte infection elsewhere on the skin .

Irritant contact dermatitis (ICD):

Definition- ICD in a broad sense represents the Cutaneous response to the physical , toxic effects of a wide range of environmental exposures . The following types of irritant contact reaction may be distinguished :-

1. **Burns .**
2. **Irritant contact dermatitis : a. acute (toxic) ICD , b. Cumulative irritant /insult CD .**
3. **Transient or immediate –type , non-immune contact urticaria .**
4. **Symptomatic (subjective) irritant response .**
5. **Others : pimentary and granulomatous response and those localized to appendageal structures (folliculitis by tar and oil , acne –halogenated , miliria –ALCL –occlusion , hyperpigmentation –phototoxic agents , hypopigmentation –phenols , granulomatous –silica-talc –beryllium , alopecia –borax –chloroprene dimmers .**

History and epidemiology:

CD was first described to plants as long ago as 2000BC , when extract of the caster oil bean was rubbed into the scalp , as an irritant to promote hair growth . The prevalence of hand eczema in Sweden per one year period is 11% , female : male is 2:1 . 35%ICD , 22%AD, 19%ACD . In Iraq (Diyala province) the prevalence of CD is 10% of all skin diseases and 49% of all types of dermatitis , 34% was ICD and 15% was ACD out of all dermatitis .

Pathogenesis- the following factors are important :

1. **The skin barrier –** the skin provides the first and most important line of defense against exogenous noxious agents , **a. The surface film ,** is the first part of the line of defense , which has a negligible influence on percutaneous absorption , stratum corneum hydration and on the barrier function of the skin . **b. Stratum corneum represented the** entire principle epidermal barrier , which is normally renewed every 17-27days , but barrier function can be restored in 2-5days following stripping or superficial injury , damage to stratum corneum is normally followed by an increase in percutaneous absorption and in transepidermal water loss , including irritant substances .
2. **Mechanism of action of irritant –** an irritant is any agent physical or chemical , which is capable of producing cellular perturbation if applied for sufficient time and in sufficient concentration . Dermatitis arise when the defense or repair capacity of the skin is exhausted , or when the penetration of chemicals will alter or damage skin cells excites an inflammatory response . Strong irritants will induce a clinical reaction in almost all individuals , whereas with less potent irritant , only the most susceptible individuals , and those with repeated contact with irritants will develop dermatitis .

Pathology – is variable according to the stage of the disease i.e. acute or chronic . Acute irritant form shows , spongiosis , intracytoplasmic vaculation , vesicular

changes , sever forms shows necrolysis of epidermal cells with intra-or-sub epidermal vesicles and bulla(burn) . Chronic form (cumulative) shows hyperkeratosis with areas of parakeratosis , moderate to marked acanthosis and elongation of rete ridges .

Predisposing factors :

A. Individual factors :

1. Genetic /racial background – those with fair skin and Japanese are more susceptible to UVB and chemicals , then ICD , than those with black skin (thicker skin more protection) .

2. Age- skin of the very young , neonate or premature is more vulnerable to some irritants than in older individuals , due to often a reduced inflammatory response to irritant substance .

3. Sex- women more frequently report skin diseases than men , this is persumablly relate to more exposure to irritants in women than men .

4. Neuro-psychological factors – e.g. sleep deprivation , the stress and decrease activity , may resulted in delayed barrier recovery .

5. Site – the effects of irritant contact varies from region to region on the body , thickness or type of stratum corneum , hair follicles , sweat ducts , potential for occlusion by body folds and inherent differences in keratinization and intercellular lipids are important in the barrier function of the skin e.g. skin of the face , scrotum and back of the hands is more permeable than other sites .

6. Skin diseases and atopy – inflammatory changes in epidermis facilitate percutaneous absorption e.g. ACD can promote penetration of irritants and Vera-versa , atopic patients and those with AD are more liable to skin irritation than normal individuals .

B. Environmental factors :

1. Temperature – higher temperature leads to a reduction in barrier function and increases the penetration of detergent through the skin , so the hot detergent appears more irritant than cold one . Cold alone will reduce the water content and plasticity of stratum corneum and lead to cracking .

2. Climate- low humidity (ambient) is the single most important factor with regard to the water content of the stratum corneum , a change to a low dew point can occur suddenly during winter and can cause chapping even in normal persons . There is interaction between temperature and humidity .

3. Occlusion – promotes percutaneous absorption and facilitate ski irritation and enhance the effects of irritants to which an individual has already been exposed , by increasing the water content of the SC e.g. rubber and plastic gloves , rings water proof adhesives , shoes , boots , clothes and the natural folds of the skin , soft paraffin itself has an occlusive effect .

4. Mechanical irritation – e.g. friction may enhance penetration of chemical and toxic substances .

C. Chemical and physical events or substance – there are different groups of chemical and physical agents , which shows significant differences in absorption and diffusion characteristics , e.g. alkaline solutions have a deleterious action on the horny layer and promote percutaneous absorption , physical injury e.g. rough sheets produced facial dermatitis in neonates and frictional factors in cases of hand dermatitis .

Clinical features- irritant contact reactions are inflammatory reactions of the skin to an external agent or agents in which , although inflammatory and immunological mediators may be activated . No memory T-cell function or antigen specific immunoglobulin are involved . Irritants produce a wide range of response on the skin , which may range from purely subjective sensation such as stinging , smarting , burning , sensation of dryness and tightness , transient urticarial reactions to more persistent irritant reactions or irritant contact dermatitis . ICD has a spectrum of clinical features , ranging from a little dryness , redness or chapping through various types of eczematous dermatitis to an acute caustic burn .

1. **Chemical burn –** it results when there is irreversible cell damage and necrosis occurs , there is usually rapid onset of painful erythema , often with in minutes , at the site of exposure , followed by blistering and the development of necrotic ulcers , wheals may be seen as a result of toxic degranulation of mast cells . Symptoms coincide with the exposure , but with some chemicals , including phenols and weak hydrofluoric acid , the onset may be delayed . Most acids coagulate skin proteins and as a result form a barrier which impedes further penetration . **Alkalis** e.g. NaOH , CaOH , KOH , Na & K cyanides , degrade lipids and saponification of the resulting fatty acids form soaps , which aid penetration deeper into the skin , as a consequence damage is more sever than with most acids and pain is also a feature , the dead skin turns brown and later black , usually without blistering and forms a hard Escher .
2. **Irritant contact dermatitis (ICD) :** The clinical appearance is essentially not differ from that due to other causes , allergic or endogenous .

a. **Acute irritant contact dermatitis (AICD) :** It is often the result of a single overwhelming exposure to an irritant or caustic chemical or a series of brief chemical or physical contacts , which results in acute inflammation of the skin and usually associated with an immediate sensation of burning or stinging . The initial reaction is usually strictly limited to the site of application or contact , which may be enhanced by occlusion and prolonged duration of application . **Clinically** AICD presented in spectrum , ranging from a mild irritant reaction with transient erythema or chapping to a much more florid dermatitis with edema , inflammation , pain and vesiculation , in more sever cases there may be exudation , bulla formation and tissue necrosis indistiguifable from a chemical burn e.g. cement dermatitis . In patients with accidental or sporadic exposure , the dermatitis usually heals quickly , unless there is skin necrosis .
b. **Delayed irritancy :** It is a delayed time course of irritation , which sometimes cause problems in the interpretation of patch test reactions , because the inflammatory response occurs later (at48h) and may there for simulate a ACD , e.g. propylene glycol .
c. **Cumulative irritant contact dermatitis (CICA) chronic ICD /Wear and Tear dermatitis :** It develops as a result of a series of repeated and damaging insults to the skin , which results in break down of stratum cornium barrier and a great number of normally innocuous substance can perpetuate an irritant contact dermatitis .CICD may therefore be due to the summation of various adverse factors, many of which would not in themselves be strong enough to cause ICD . Once CICD develops , any of these minor irritants may also act as perpetuating factors . CICD most commonly affects thin or exposed skin for e.g. the dorsa of the hands , fingertips , the finger's webs , the face and the

eyelids in those with cosmetic intolerance or low humidity dermatosis . ICD often begins with a few localized patches of dry , slightly inflamed or chapped skin and the tendency to disseminate is normally less than with constitutional or ACD . Nearly 80% of those with chronic disability dermatitis were found to be atopic . Occupations associated with ICD are hair dressing , medical , dental , veterinary , cleaning , agriculture , horticulture , forestry , food preparation and catering , printing and painting , mental work , mechanical engineering , construction and fishing .

d. Hand dermatitis (eczema) : The pathogenesis of hand eczema is often complex . Constitutional irritant and allergic factors frequently coexist , more common in women , as a result of increased irritant exposure rather than an inherent susceptibility , usually there is no pathognomonic single cause . Hand dermatitis that are suggestive of ICD include : ***patchy house –wife type eczema – which affecting principally the dorsa , sides , finger's webs , a ring eczema** , due to wet work and exposure to detergent , usually start as dryness and developing into patchy or diffuse erythema with scaling , fissuring and even visculation . However vesicles are less commonly seen in irritant than allergic or constitutional eczema (principally shows dryness or chapping). ***Another common pattern of irritant hand eczema is the "apron"** or extended finger tip eczema , with dryness , redness and fissuring affecting principally the palmer aspects of fingers and distal palm . ***Another form is discoid or nummular hand eczema ,** which is rare form of ICD affecting especially the dorsa of the hands or fingers . **D.D. fungal infections , psoriasis and scabies in the interdigital spaces .**

e. Cosmetic dermatitis : cosmetics , toiletries and skin care products , including sunscreens , quite frequently cause adverse reactions , in most cases these are only mild or transient and most consumers simply change to an alternative products . In minority , reactions may be more sever , with redness , edema , dryness and scaling , particularly the eyelids , most commonly seen in premenopausal women using many cosmetic products , which is a form of cumulative cosmetic ICD , allergic contact dermatitis is only excluded by patch test to both products and ingredients .

f. Volatile air borne ICD – both irritant as well as allergens may cause volatile CD , not infrequently cause eyelids dermatitis , for e.g. fumes of acids , alkalis , solvents , resins or any other irritant chemicals , such as ammonia or formaldehyde , irritant dusts of woods , cement , fiberglass or rook wool ,, metal and metal salts .

g. Cheilitis – is a common problem , atopic eczema , lip licking ICD , cosmetic and medication ACD .

h. Napkin (diaper) – peristomal and perianal dermatitis : It is an ICD results from prolonged or too frequent contact with degraded urine or faeces /faecal residues , sweat , occlusion , irritant cleansers , secondary infection and secondary medicament allergy are all additional complicating factors . It occurs in infancy and in elderly in situations of urinary or facial incontinence , and may be complicated by secondary bacterial or candidal infection . Perianal dermatitis may occur as a result of mucus or faecal leakage , that occurs in association with haemorrhoids and /or poor sphincter function , a similar condition with peristomal dermatitis .

3. Non-immune contact urticaria : it is an immediate contact reaction occurs without prior sensitization , the reaction remain localized and may present as a

transient erythema or as an urticarial wheal and flare , e.g. benzoic , sorbic , cinnamic acids , nicotine acid esters , alcohols , arthropods .

4. **Symptomatic (subjective) irritant responses :** With some irritants the individuals complain of a subjective sensation of stinging , burning or smarting , these sensory symptoms are not limited to **chemical exposure ,** woolen **garments , mechanical irritation** , also may cause these symptoms . It is divided Into :

a. Immediate –type stinging – it is a painful sensation occurs within seconds of contact with the chemicals , like acids , which may be a prodrome to the development of more sever Cutaneous damage . Other substances like chloroform and methanol causes only stinging with out skin damage , this sensation abates quickly following the removal of irritant substance .

b. Delayed-type stinging – typically there is no immediate stinging , but discomfort develops within 1-2min. , reaches a maximum in 5-10min. and fading slowly over the next half hour e.g. sunscreen and insect repellent . The sensation dose not correlate with a predisposing to irritant dermatitis or non-immune contact urticaria .

Common and specific irritants :

*Water an wet work , e.g. sweating under occlusion .
*Household cleaners , e.g. detergents , soap , shampoos and disinfectant .
*Industrial cleaning agents , including solvents and abrasives .
*Alkalis , including cement .
*Acids – pesticides , raw food , animal enzymes and secretions .
*Cutting oils – e.g. desiccant powders , dust , soil .
*Organic solvents .
*Oxidizing agents .
*Reducing agents .
*Certain plants .

Diagnosis- ICD is essentially a clinical diagnosis based on knowledge of the nature and condition of an individuals exposure in the context of their dermatitis . ACD always needs to be excluded by patch test .

Management :

1. Chemical burns – initially it requires irrigation with large volume of lukewarm water , if the chemical is insoluble in water a soap solution may be used instead . High pressures should not be used , to avoid splashing other areas of the body , specific antidotes can be used e.g. 2.5% calcium gluconate gel for hydrofluoric acid , ulcerated areas should be managed by topical antibacterial agents to prevent secondary infections . Don't forget the risk of systemic toxicity from absorption as with chromic acid .

2. ICD – the successful management requires both : a. **Prevention and b. Treatment of dermatitis if developed .**

a. Prevention – includes :

A. Avoidance – e.g. mechaninization is used to avoid exposure to wet or irritant work will help to reduce the incidence of ICD .

B. Protection – which includes :

1. Personal protective equipment e.g. gloves in hair dressers (unprotected wet work for greater than 25days was the most significant risk factor for the development of dermatitis .
2. Topical preparations e.g. **barrier creams (W/O as ointment against aqueous irritant or O/W as cream against lipophilic materials) , emollients or**

hand creams (to prevent dryness or chapping of the skin and then subsequent dermatitis) , soap substitutes , avoidance of brushing of hands for surgical procedures .

b. Treatment – once dermatitis present , it requires palliation of symptoms with topical steroids and emollients . It has been suggested that conditions in which the lamellar body secretary system is impaired or immature e.g. radiation dermatitis , sunburn , ICD due to some surfactants and retinoid and premature infants of less than 33weeks gestation) , should be treated with non-physiological lipids (damaged lipid metabolism) e.g. **petrolatum . Whereas** most other causes of ICD , where lipid metabolism has not been damaged (e.g. diaper dermatitis) , should be treated with a mixture of cholesterol : ceramides : free fatty acids , in a 3:1:1 ratio , to achieve most rapid return to normal barrier function .

In sever cases , phototherapy or systemic drugs such as **azathioprine and cyclosporine** , may be required . Where there is secondary infection , topical or systemic antimicrobial agents may be necessary .

3. Non-immune contact urticaria – is treated by non-steroidal anti-inflammatory drugs and UV light , but not by antihistamines .

Prognosis- the previously irritated skin returned to normal after 5weeks , prolonged duration of exposure to irritant substance results in prolonged recovery (e.g. 3weeks induration requires 10weeks for recovery) . **Atopic individuals** have a worse prognosis , a change of job may be helpful if under taken early , **delay in diagnosis** and assessment worsens the prognosis . **The use of** inappropriate cleansers will also affect a patients overall prognosis and outcome .

11% of individuals with ICD developed what is called persistent post occupational dermatitis , which even if the original cause has been eliminated , this condition will persist .

<u>Allergic contact dermatitis (ACD)</u>:-

Definition- it is one type of exogenous eczema , caused by exposure to an sensitizing substance (allergen) , may be acute , sub acute or chronic on clinical base .

Prevalence- of ACD through the world accounts for 4-7% of all dermatological consultations (skin diseases) , In Iraq (Diyala) , CD accounts for 10% of all skin diseases and 49% of all dermatitis , 15% of which is ACD and 34% is ICD . **Dermatitis** accounts for almost half of all reported cases of occupational diseases . Over 20% of females will suffer from hand eczema at some stage in their lives . The prevalence of ACD in the European countries is variable from 1.7%in Sweden to 28%in Germany and 15.2% in Denmark .

Pathogenesis- there are two main processes :

1. **Sensitization (induction , or afferent limb of sensitivity) –** the immunology of ACD is a delayed –type or cell –mediated immunity . It is the primary event which take place before clinical expression of dermatitis . This process is initiated by binding of allergen to skin associated with major histocompatibility complex (MHC) class-2 molecules , either directly via **antigen-binding sites** , in groove of the MHC class-2 molecule on antigen-presenting cells (APCs) , these MHC class-2 molecules are coded on the human leukocyte antigen (HLA) –D region genes and are present on epidermal dendritic cells and Langerhans cells . This process is called **cell stress (develop within 6ho)** , which result in release of co-factors (IL-1B, TNF-alpha ,

GM-CSF) , which are required for the activation , maturation and migration of Langerhans cells , travel via the afferent lymphatic to the Para cortical areas of the regional lymph nodes , which result in activation of CD4 (T-helper cells) and CD2 (T-effecter lymphocyte) , with release of many mediators or cytokines (e.g. IL-1 by APCs and IL-2 **by T-lymphocytes)** . These cytokines cause **proliferation and dissemination of sensitized T-lymphocytes , with blast formation in the lymph node** and proliferation of antigen-specific cytotoxic CD8(Tc1) and also CD4(Th1) lymphocytes . The type of T-cell response generated is dependent on the pathway by which the antigen is processed : **small lipid-soluble molecules** such as **urushiol** enter the cytoplasm and presented on MHC class -1 as an endogenous antigen , **polar haptens** are more likely to be presented on MHC class-2 as an exogenous antigen . The process of sensitization in most subjects developed and accomplished with in 5-25days of exposure to allergen (called 'late' reaction) .

2. **Elicitation –** if a sensitized person is re-exposed to a specific allergen in sufficient concentration , the clinical reaction subsequently develops much more quickly , usually within 24-48ho , however depending on the *degree of sensitivity ,*penetration and *other factors , this may vary from a few hours to many days . Some times a sensitive person might react to substances of related chemical structure , a phenomenon later termed **'cross-sensitization'** , some sensitizer only provoke a reaction if activated by light (photo allergic dermatitis) .

Predisposing factors:

1. **Individual factors** :
   a. **Constitutional –** individual susceptibility , siblings and children of patients suffering of ACD have an increased incidence of positive patch test and first degree relatives of nickel allergic subjects have increased prevalence of the same disorder .
   b. **Sex –** women have stronger cell-mediated immune responses than men and yet , at least experimentally women do not appear to be more susceptible to sensitization , but with female preponderance in clinical patch test , is largely the result of **metal-sensitive females** , especially the nickel .
   c. **Hormones-** e.g. oral contraceptive enhanced ACD by DNCB in women , also pregnancy and the use of gestagens may unpredictably **either** improve **or** aggravate ACD , also the premenstrual flare .
   d. **Race-** Afro-Caribbeans are generally more resistant to sensitization than white people , most probably a reflection of exposure rather than predisposition .
   e. **Age-** has little influence on capacity for sensitization , but the number of positive patch test reactions tends to increase with age , most probably due to the accumulation of allergens acquired over a life time .
   f. **Medication-** e.g. antihistamines and sodium cromoglicate have little effect , where as steroids like prednisolone (dose >15mg/day) and potent topical steroids , both suppress ACD , similarly , other immunomodulaters such as cyclosporine and azathioprine may reduce the intensity of allergic contact reactions , aspirin , UVB and PUVA may also temporarily reduce contact allergic reactions .

g. **Coincidental diseases-** acute or debilitating diseases such as cancers , Hodgkin's disease and mycosis fungoides have impaired capacity for contact sensitization , also sarcoidosis and lepromatous leprosy , parasitosis with impaired T-lymphocyte functions .

h. **Local factors- e.g. skin damage** and irritancy (ICD, eczema) effect the barrier function of the skin and may increase the allergen absorption and secondary sensitization (ACD) , acanthosis also facilitated sensitization .

2. Environmental factors :

a. Climate- UV exposure , heat and relative humidity may play a part in liability to contact allergy . **UVB** exposure has been shown to diminish the skin immune response to contact allergens , but **UVA** have transient effect on immune response , **chapping** of the skin during winter predispose to ICD and ACD , **occlusion** and increased sweating may increase allergy from shoes and clothing .

b. Flora and founa – e.g. plants dermatitis commonly shows a distinct seasonal pattern , cold , warm , light , dryness, wind and some geographical location may show a very important influence . Fishermen are liable to ACA of exposed skin during summer when handling nets containing **marine organisms .**

c. Socio-economic and cultural- e.g. exposure to cheap (nickel releasing) metals used as jewelry , perfume and cosmetic use , might vary according to social class . Hair dyes are used much more commonly by men in the Middle East . Sunscreens and cosmetic use are commonly seen in Western culture , other cultural factors e.g. use of topical or oral medication in the Middle and Far-East .

3. Chemical factors – skin cells , especially their nucleic acids and proteins are composed of molecules that contain nucleophilic atoms , i.e. negatively charged and electron rich , while most allergens (haptens) are 'simple' chemicals of low molecular weight (less than 500-1000Da) , that contain electrophilic atoms , i.e. positively charged and electron deficient . **Interaction** between these two types of atoms leads to strong covalent bonding to form a hapten-protein complex (complete antigen) . Metal and metal salt can bond to electron-rich atoms (ligands) by taking some of the electrons and forming coordinate bond . Some molecules (pro-haptens , not allergens) are converted into electrophilic molecules by skin hydroxylating systems , monoamine oxidase , peroxidase , atmospheric O2 and UV irritation into complete haptens , so become allergenic .

Pathology: is similar to that of ICD , but electron microscope examination suggests that **langerhan's** cells play an important in ACD .

Clinical features: ACD can mimic or be associated with any type of eczematous eruption . The diagnosis is based on ***a carful history ,** combined with ***a sound knowledge of common allergens and irritants** in the environment .

1. History – a comprehensive history to elicit potential allergens is essential and some knowledge of chemistry and industrial processes is of value . Sensitization and subsequent ACD may result from a single exposure to a strong allergen , although usually several or many exposures are necessary before sensitization and dermatitis occur .

a. Primary site – must be ascertained by questioning the patient carefully , it is the beginning site of ACD , which may give an important clue to the cause .

b. Duration and behavior- once the date of onset and the primary sites have been identified , it is necessary to establish the *subsequent behavior of the disorder , e.g. improvement of ACD during weekends or holidays favors an occupational origin , while relapse suggests a hobby of non-occupational allergen . *Seasonal variation

suggests a plant allergen (recur in atypical patterns) . *Dermatitis around a wound suggests sensitization to medicaments .

c. Previous history – of dermatitis may provide a clue to the origin of a relapse e.g. earring dermatitis may precede nickel dermatitis of the hand by several years , leg dermatitis due to topical antibiotic , lanolin and preservatives in creams . history of atopy (hay fever , asthma and atopic eczema) .

2. sources of allergens – a search for possible sources of allergens in ACD should concentrate on :-

a. Occupation- a precise history by a through knowledge of the materials handled at work , machinery operated and personal protection employed will be necessary when occupational ACD is suspected , also housework should not be overlooked and personal second jobs .

b. Hobbies- common sensitizers will known as industrial allergens , are introduced into most homes for de-it-yourself work . Cement , glues , paint, wood and wood preservatives are handled by many householders , gardening , sports , cookery and photography should be considered .

c. Personal objects- cosmetics and clothing , textiles , footwear , gloves , protective clothing , jewellery , spectacle , hearing aids , medical appliances , toiletries , fragrances and medicaments , all may be a cause of ACD .

d. Home environment .

e. Current and previous topically applied medicaments .

On examination the clinical features are :-

***Eczematous responses (dermatitis):**

The primary signs of CD are erythema , swelling , papules and papulovesicles and blisters , which reflect the sequence of inflammatory changes in the dermis and the intra- and intercellular edema in the epidermis , rupture of vesicles and blisters results in weeping dermatitis , itching is the dominant symptom . If CD persists , the skin becomes dry , scaly , thick , lichenification and fissuring may develop later , this chronic ACD cannot always be distinguished from constitutional or ICD and the etiology is indeed often mixed , but the distribution of dermatitis is of diagnostic importance and its morphology is usually of no help in tracing the cause .

1. Primary patterns (sites): anatomical patterns of dermatitis often suggests a specific cause , but in other cases the pattern merely indicates a range of possible allergens , such as in shoes dermatitis , some times the dermatitis is sharply limited to the usual site of contact , but some allergens may be spread locally by the fingers or be carried to distant body regions foe e.g. from the fingertips to the genital area , eyes , face and neck . Once the primary site has been established , questioning should focus on those allergens that are particularly frequent causes of dermatitis in that region .

***Hands and arms:** it is usually multifactorial , represent about 2/3 of all cases of CD involve the hands (ICD&ACD) , also the hands are a common site for constitutional patterns such as pompholyx , hyperkeratotic eczema an atopic dermatitis , which represent a diagnostic difficulties for ACD . The majority of housewives and occupational dermatitis are of ICD and remain confined to the hands (e.g. rubber gloves dermatitis) . Vesicular palmer ACD may mimic constitutional eczema and may also result from contact with or ingestion of an allergen to which the person is already sensitized (e.g. nickel , chromate , balsams and garlic) . The ICD affect mainly the dorsa of the hands , webs , backs and sides of the fingers , but ACD usually affect the finger webs and extending to the front of the wrists and up the forearms , also in domestic workers begins under the ring and spread to the neighboring webs and adjacent part of the palm (i.e. there is overlap between both

ICD &ACD) , may be due to exposure to plants , animal products and agricultural substances . **The arms** are affected by the same allergens as the hands but usually later e.g. nickel , chromate , formaldehyde resin , leather and glue respectively in watchstraps and wood dust .

*__Face:__ ACD of the face may occur alone or in association with hand eczema , as a result of exposure to fragrances , preservatives and other constituents of skin-care products and cosmetics , including nail varnish , it is a common type of ACD which may involve the face , eyelids , neck and chest . The other allergens are acrylic nails , rubber sponge , cosmetic applicators , hair dyes , chromate in leather hatbands , spectacle frames , also air born or volatile(epoxy) allergens and photosensitizes . **Clinically** the cosmetic dermatitis may start with dryness , tightness and itching . In cases of eyelids dermatitis ACD , nail varnish , fragrances sprays , volatile substance(epoxy resin) , eye creams , eye shadows , mascara , eye make up removers , hair dyes and eye drops and ointment medications e.g. neomycin gentamycin , sulphaonamides , anesthetics , antihistamines and contact lens solution .

*__Lips or perioral area :__ lipstick is the common cause of ACD , usually limited to the vermilion border , which appears dry , scaling or cracked , occasionally the perioral area is also affected (eosin , ricinolic acid , caster oil , gallates and UV filters present in lipsticks are the cause of ACD . Other sensitizers presented in tooth paste and dentures are a possible causes of ACD (chelitis) , also badly fitting dentures , nail varnish and nickel plated objects e.g. keys , pins or musical instruments) are considered .

*__Ears:__ ACD her are due to medications , hairpins (nickel), matches (chromate) , nail varnish , hearing aids (acrylates and chemicals) , head sets (urea , phenol , formaldehyde resins) and rubber in earphone . Also spectacle-frame (nickel , palladium or titanium) , hair dyes , plastic components (epoxy resins) earplugs .

Other skin areas: including scalp , neck , axillae , trunk , anogenital region , thighs , lower legs and feet may be involved by ACD , as a result of exposure to a different allergens . Some time the ACD may be generalized called erythroderma .

2. Secondary patterns (sites) : ACD may start at one site , but commonly other sites are subsequently involved , and sometimes several regions simultaneously . This phenomenon has been termed an **id-like** spread (**id reaction)** or secondary contact dermatitis .

3. Mucous membrane : mouth , nose , eyes and genital mucosa may be involved by a low degree of contact sensitivity , which develop as a result of exposure to allergens e.g. nickel , chromate , toothpaste , dentures and medications , but most commonly it is **a secondary** contact sensitivity , **clinically** the mucosa shows erythema and swelling but vesicles are rarely seen except on the vermilion border , the symptoms are soreness , burning and itching is uncommon and the eczematous reaction of the adjacent skin may occur , which may be the only signs of an ACD .

Systemically reactivated contact dermatitis :

ACD may be reactivated by ingestion or other systemic exposure to a contact allergen , takes place in an already sensitized person . The threshold of reaction varies in each individual case and depends on the dose given and the level of sensitivity , and the reaction may be enhanced by ingestion of the primary allergen or secondary (close related) allergen . **Clinically** , the most frequent types of reaction are focal flares of previous patch test , vesicular hand eczema or much more widespread eczema and erythema , sometimes with additional urticarial features , less commonly in severs cases vasculitis , EM and systemic upset may occur . The ingested allergens includes

nickel , medication (neomycin , streptomycin , sulfa, antihistamine, anesthetics , steroid , Balsam of Peru) , garlic , food colors , preservatives and antioxidants may be ingested orally , parenterally , rectally , intravesically or as an inhalant .

Photo contact dermatitis : Toxic (ICD) and allergic (ACD) .

It is a type of CD (ICD, or ACD) , which is enhanced by exposure to a certain substances called photosensitizers , in the presence of irradiation with UV or short – wave visible light (280-600nm) , so the irradiation transformed these inert substances in to either irritant or sensitizers , by a physical phenomenon , called photoactivation and may occur in vitro , when it occurs in vivo the activation may have a phototoxic (non-immunological) or a photoallergic (immunological) action . The phototoxic reaction is similar to ICD and the photoallergic reaction is similar to ACD by pathogenesis and clinical features , but both the phototoxic and photoallergic substances required the light to become active .

The allergens may involve the following :

*UV filters (sunscreens) paraaminobenzoic acid , cinnamates .
*Perfumes e.g. coumarin .
*Halogenated salicylamides , used as antibacterial and antifungal soaps .
*Topical non-steroidal anti-inflammatory agents e.g. ketobrofen .
*Phenothiazines e.g. tranquillizers , topical antihistamines and insecticides .
*Sulphonamides , used for topical treatment .
*Bithionol and hexachlorophene in toilet soap , shampoos .
*N-Butyl-4-chlorsalicylamide in Jadit antifungal .
*Eosin –used in lipstick .
*Quinines in hair tonic , quinine , quinidoxin .
*Thiourea .

Clinical features : Photosensitive reaction can resemble sunburn , but usually shows the same spectrum of features seen with ACD (erythema , papules, vesicles, scaling and fissuring) , her the dermatitis is localized to the exposed areas of the skin , usually with well demarcated margins , where the skin is uncovered by clothing e.g. at the collar and 'V' area of the neck and other exposed areas , the areas below the chin , triangle behind the earlobe are usually spared . The **disorder** usually subside when the exposure to photo allergen is discontinued , but some times and in particularly when exposure is continue , the persistent (chronic) light reactivity may be developed and chronic photoallergic CD occur , which may progress to chronic actinic dermatitis .

The **intensity of** response to phototoxic and photoallergic agents depends upon a number of factors :-

1. Nature and concentration of the substance applied .
2. Duration of exposure to the substance.
3. Percutaneous absorption .
4. Intensity and the wave length of the radiation .
5. Duration of exposure to the radiation .
6. Radiation absorption in the skin , depending on the thickness of stratum corneum as well as the amount and distribution of melanin .
7. Extraneous matter and secretions on the skin .
8. Humidity .

Avoidance: by avoiding the exposure to photosensitizer and using physical sunscreens e.g. tatenium dioxide and zinc oxide .

Diagnosis: is confirmed by photo patch test .

Non-eczematous response to contact allergens :
A group of skin disorders are result for exposure to certain substances (allergens) , without the development of eczematous rash . These are include ;- **contact urticaria, E.M. like reaction , purpuric reactions ,** lichen planus and lichenoid reactions , lymphamatoid eruptions , pigmented dermatitis, depigmented lesions, onycolysis, systemic non-eczematous reaction (intense edema , general malaise, metabolic effects) and granulomatous reactions .

Differential diagnosis of ACD:
The diagnostic problems differs according to the site of the dermatitis and patch test will often required .
Face- A.D., seb. D., psoriasis, cellulites, erysipelas, angioedema, dermatomyositis, herpes simplex, by clinical criteria and history .
Hands and arms: ICD , pompholyx, constitutional eczema, AD, psoriasis, hyperkeratotic eczema, tinea manuum, LP, porphyria cutanea tarda .
Flexures & anogenital region: seb. D, psoriasis, tinea and erythrasma .
Legs & feet: stasis eczema, tinea pedis, scabies .
Trunk: popular drug eruptions, scabies, dermatomyositis, mycosis fungoids .
Generalized: erythroderma, , psoriasis, drug rash, bullous diseases e.g. pemphigus folacious, scabies .

Patch test: is used to confirm the diagnosis of ACD and **photo patch test** for photoA**CD** . It is **type 4 immune reaction** (delayed hypersensitivity) . It is performed by using **an adhesive tap** contains chambers or discs , which are used to ensure occluded contact with the skin (the fixing tap should be non-occlusive, non-allergenic, non-irritant . **Patch test** should not be carried in patients with **:*active eczema, *following sun bathing, *steroid & immunosuppressive therapy .**
Patch test tap is applied to the back (trunk) , the ideal regimen to read patch testing is a 48ho. Application time, with readings taken 1ho. after removal , than after 2days and after 4days . The recording of patch test reactions according to ICDRG (**International Contact Dermatitis Research group) are :**
***- Negative .**
***?+Doubtful reaction – faint erythema only .**
***+Week positive reaction –**palpable erythema , infiltration, possibly papules .
***++Strong positive reaction** –erythema , infiltration, papules, vesicles .
***+++Extensive positive reaction** –intense erythema and infiltration and coalescing vesicles .
IR-Irritant reaction of different type .
NT-Not tested .
****In photo patch testing –** a source of UVA is required as well as to the suggested photo allergen .
****Causes of false positive reactions:**

1. High concentration .
2. Impure substance (contaminants) .
3. Irritant vehicle .
4. Excess allergen applied .
5. Uneven dispersion .
6. Current or recent dermatitis at patch test site .
7. Pressure effect of hard materials .
8. Adhesive tap reactions .

9. Angry back reaction .
10. Artifact .

Management of ACD:- Allergy when develop it persist through out the life .
a. Prevention of ACD involve :*Avoidance of exposure & *protective advice , which include:- *work preventive measures, *domestic equipment, *education . Once diagnosis of ACD has been made , possible sources of exposure to the causative allergen(s) should be identified and protective advice given , e.g. use of plastic instead of rubber gloves , cosmetic and medicaments free of an identified allergen and clothing free of nickel-containing studs , zips, etc--- . In some patients continued exposure is unavoidable , but can be reduced to a sufficient degree to keep the dermatitis at an acceptable level .
Principles of prevention can be related to two categories , *individual and *collective. **Further divided in to:** ***primary** prevention focuses on the induction of contact sensitization and control of exposure , ***secondary** prevention related to elucidation of ACD , ***tertiary** prevention include measures for established and continuing dermatitis .
b. Treatment :
1. **Active treatment -** *T**opical** corticosteroid is the **most important** step in the treatment of ACD . In acute , sever , localized ACD , a potent topical corticosteroid should be used . In more chronic or wide spread ACD , the potency may need to be reduced and for long term use in certain sites (face, genitalia &flexures) , mild topical corticosteroids are indicated . On the palms and soles , the long term intermittent use of a potent topical corticosteroid preparation is usually used .
***For acute weeping forms of ACD ,** wet dressing with saline , aluminum acetate or silver nitrate (0.5%stain black) may be of benefit , potassium permanganate solution 1/8000 in warm water is helpful when used four time/day as a soak for vesiculobullous eruption till become dry .
***New topical ascomycin derivatives (immunomodulater), topical tacrolimus .**
***General hydrating emollients** and soap substitutes , antibiotic for secondary infection , antihistamines for pruritus .
***For sever or wide spread ACD** , systemic steroids may be necessary .
***For recalcitrant disabling cases,** immunosuppressive therapy such as azathioprine and cyclosporine may be required .
***Phototherapy** by both PUVA and **UVB** are helpful in some subjects .
***Superficial X-rays** and Grenz-rays to suppress experimental CD .
2. **Hyposensitization –** an attempts to down-regulate the immune response to allergens in an already sensitized individual , by oral or parental rout hyposensitization .
Prognosis: The prognosis of ACD depends on it's cause and the feasibility of avoiding repeated or continued exposure to the causative allergen . **Older atopic individuals** carry a poor prognosis , also those allergic to nickel and chromate . Once acquired ACD tend to persist , chronic and relapses due to :- ***impairment of barrier function, *inappropriate treatment, *ingestion of allergens, *secondary infections, *auto sensitization, *stress, *constitutional factors predispose to chronicity,*inherent tendency in almost any eczema to become chronic and continuous .**

Atopic dermatitis:

Definition- It is an itchy, chronic or chronically relapsing , inflammatory skin condition with itchy papules , occasionally vesicles in infants , which become excoriated and lichenified , typically has a flexural distribution and frequently associated with other atopic conditions in the same individual or other family members .

Atopy- Is a convenient collective term for group of diseases , chief among which are ***asthma ,** ***hay fever** and ***atopic dermatitis ,** which occurs spontaneously in individuals who have a family history of susceptibility . Sixty-80% of atopic individuals has elevated IgE level and could be transferred to normal individuals and may have others IgE elevated diseases (e.g. anaphylaxis) . The others 20-40% of atopic individuals , who have normal IgE level can be distinguished clinically , immunologically and prognostically and termed **intrinsic non-atopic infantile eczema or atopiform dermatitis .**

Prevalence- AD is most prevalent in the most developed Westernized countries and least prevalent in the most non-Westernized and under developed countries , e.g. in Norwegian children from 7-13years , the prevalence was 19.7% , Danish children up to 7years , the prevalence was 22.9% , in Germany 13.1%, Sweden 15.5%, UK children from 3-11years , the prevalence was 11.5-14% , Japanese children of 3years old , the prevalence was 20% and for school children was 9.5%, Tanzanian children 7-8years the prevalence was 0.7% , in Iraq (Baquba) , the prevalence in children from 6months -10years was 21% of all types of dermatitis and 4% of all skin diseases . There are a consequence rising in the prevalence of atopic dermatitis all over the world from 1960 till now , which may be due to environmental factors .

Etiology- The following factors are important :

1. **Genetic factors-** the importance of genetic factors is reflected in data from twin studies , thus monozygotic twins have a concordance rate of 0.72, where as dizygotic twins have a concordance rate of only 0.23 . A gene predisposing to atopy was found on chromosome 11q13 . **Maternal factors and inheritance** also important , that the risk of children to develop atopy is significantly greater with an atopic mother than with atopic father .
2. **Pregnancy & intrauterine factors-** *there is a positive correlation between increasing birth weight and prevalence of AD , this is called **intrauterine programming mechanism ,** *other possible mechanism is called **genetic imprinting** (in which paternal genomic effects are suppressed), *a third possible factor is the onset of immunological sensitization through intrauterine exposure to food and environmental allergens .
3. **Environmental factors –** these factors are the most likely modulating influences , that increase the prevalence of atopic eczema , two principle factors are important the, *the pollution of CO2 , SO2 and *microbes (bacteria) .
4. **Immune dysregulation –** the defining characteristic of the atopic immune system is the capacity to generate IgE antibodies in response to allergens . The **dendritic cells (APC) in atopic individuals bear antigen-specific IgE** on their surface , which allow them to present AGs to T-cells much efficiently , which result in activation of Th0 to Th2 cells by production of IL4,10 and

PGE2, this Th2 release IL4,5&13 , which help or control the type of Ig that produced by B-lymphocytes , result in induction of IgE synthesis , mast cells degranulation and eosinophils activation .

Pathogenesis of AD:

There are many factors , including allergies , infections, emotional, climatic and other environmental influences that contribute to the causation of AD .

1. **In early life** , atopic infants develop eczema due to immune sensitization , as the first of the possible atopic syndrome (eczema, asthma and rhinitis**) .** There is evidence that immune sensitization occurs to food-derived allergens as well as aeroallergens . These occurs because the infantile intestine shows increased permeability to macromolecules and this is greater in atopic infants , this may be due to inherent leakiness and also because of the transient deficiency of IgA, so the IgA mediated clearance mechanisms are less effective , allowing greater entry of food-derived macromolecules into the systemic circulation , where they may induce immunological sensitization .
2. **The role of airborne environmental allergens (aeroallergens e.g. HDM, pollens, malassezia yeast) ,** both the initial sensitization of atopic individuals and the subsequent elicitation of the clinical features is become clear , in genetically predisposed individuals .
3. **Once eczema become evident ,** the epidermal permeability barrier is disrupted , allowing penetration of environmental aeroallergens , during the first few years of life , which result in progressive development of immune reactivity to these aeroallergens , reflected by the presence of specific IgE and T-cell response .

Pharmacological and vascular abnormalities in AD:

The small blood vessels in atopic dermatitis shows a tendency to vasoconstriction responses , which manifested by:

1. Pallor of the skin after stroking – whit dermographism .
2. Delayed blanch with acetylcholine .
3. White reaction to nicotinic acid esters .
4. Abnormal reactions to histamine in affected skin .
5. Low finger temperature .
6. Pronounced vasoconstriction on exposure to cold .

Non of these findings is pathognomonic , but they are a characteristic of AD. These vascular changes are due to **neuropeptides** , which mediate vasodilatation , edema, itch, pain, axon-reflex flare, sweat gland secretion and have some probably minor ability to regulate T-cell activation (e.g. substance P SP, calcitonin gene-related peptide CGRP), often present together in unmyelinated C sensory fibers and (somatostatin, vasoactive intestinal polypeptide) , which coexists with acetylcholine in postganglionic sympathetic fibers and neuropeptide Y in adrenergic fibers . Theoretically neuropeptides could have significant role in many of the features of AD, including vascular changes , itch, the symptoms associated with sweating and leukocyte infiltration .

Pruritus: is the major symptom that accompanies the rash of AD , which are induced by stimuli that induce sweating , both thermal and emotional , as well as contact with fibers especially wool . Antihistamines generally have a little effect on pruritus of AD , but *sedating H1 antagonists may improve the subjective comfort through sedation and deep sleep . *Cyclosporine causes very rapid cessation of the pruritus , but effects other cells other than lymphocytes .*Capsaicin also reduce pruritus in AD by

depleting CGRP and SP . *Opioids may play apart in controlling pruritus of AD . **Sweating** induce pruritus by either *direct stimulation through ACH or by IgE mediated allergic reaction .

Psychological factors : AD is exacerbated by episodes of psychological stress , because P. stress modulates immune/inflammatory processes by increasing production of epinephrine , releasing factor and cortisol (ACTH, corticotrophin CRF) and reduced production of growth hormone , prolactin and progesterone . The rise in free cortisol is lower in patients with AD , increased lymphocyte, monocyte, neutrophil and basophile number with 10min. after the stress provocation test and were equal in both atopic and normal individuals . The significant **premenstrual flare** of AD and in 52%of **pregnant women is due to the effect of sex steroids** , which modify and reduce sensitivity to the anti-inflammatory effects of glucocorticoids (cortisol) .

Pathology: is that of sub acute (infantile phase) or chronic(adult phase) eczema .

Clinical features: AD is an itchy , chronic, fluctuating disease that is more common in boys than girls . The age of onset is between 2-6mon. in the majority of cases , but it may start at any age , even before the age of 2mon. in some cases . **The clinical features include:-**

1. **Itching .**
2. **Macular erythema, papules or papulo-vesicles .**
3. **Eczematous areas with crusting .**
4. **Lichenification & excoriation .**
5. **Dryness of the skin .**
6. **Secondary infections .**

The distribution of the eruption varies with ages as described :

a. **Infantile phase-** the lesions most frequently start on the face but may occur anywhere on the skin surface , often the napkin area is relatively spared . When the child begins to crawl the exposed surfaces , especially the extensor aspect of the knees are mostly involved . The lesions consists of erythema and discrete or confluent edematous papules , which are intensely itchy and may become oxidative and crusted as a result of rubbing , secondary infection and lymphadenopathy are common . The disease runs a chronic , fluctuating course varying with such factors as teething , respiratory infections , emotional upsets and climatic changes .

b. **Childhood phase-** from 18-24mon. on wards , the sites most characteristically involved are elbow and knee flexures , sides of the neck , wrists and ankles . The sides of the neck may show a striking reticulate pigmentation , some times referred to as atopic dirty neck . The anatomical bases for this distribution is unknown . The erythematous and edematous papules tend to be replaced by lichenfication, extensor distribution in later childhood are uncommon and may take longer to remit . As well as the typical mixture of papules and lichenification , true eczematous lesions with vesiculation may occur , often in discoid patches . Hands involvement , often with oxidative lesions and some times with nail changes is common . Secondary bacterial or viral infection may cause acute generalized or localized vesiculation .

c. **Adult phase-** the picture is essentially similar to that in later childhood , with lichenification especially of the flexures and hands . Localized patches can occur on the nipples especially in adolescent and young women , involvement of the vermilion of the lips and the adjacent skin , follicular papules (keratosis

pilaris) are frequent in black people and the Japanese . A distribution on the face , upper arms and back may correlate with areas of maximal thermal sweating or *malassezia* sensitivity . Photosensitivity is not uncommon especially in adults .

Atopic hand eczema: a patchy , discoid , some what vesicular and lichenifid eczema of the hands is a common manifestation of AD in childhood and adult , the nails are often involved , resulting in coarse pitting and ridging . The picture may closely resemble the discoid eczema of young adults . A more diffuse , chronic lichenifid eczema of the hands is frequently found in cases of extensive AD , which persist into adult life (called constitutional hand eczema) . Involvement of the feet is also common and almost half the patients with atopic hand eczema will have eczema on the feet . AD may be a factor in the development of occupational eczema .

Other manifestations of atopy (associated disorders):

*A bout 30-50% of cases of AD have allergic rhinitis (hay fever) and asthma with later age of onset . *Dry skin is a common feature of AD , due to increased transepidermal water loss through abnormal stratum corneum , *ichthyosis and keratosis pilaris also common .

*__Other patterns of eczema__- *infantile seborrhoeic dermatitis , normally starts earlier than AD, clinically different and may progress to typical A**D** , *ACD and ICD incidence is high in patients with AD , *lip-licking cheilitis (perioral eczema), is also common in children with AD, presented as moist or fissured eczema around the mouth , also can occur as a result of food allergy and in children with no known atopy or allergy , frequently it spread some distance around the mouth and may become secondarily infected and crusted . Its persistence and perhaps its origin is attributable to habits of lip-licking , thumb sucking, dribbling or chapping , it is easily transformed into a true perioral dermatitis by application of potent corticosteroids, also toothpaste contact sensitivity can occasionally be demonstrated , 1%hydrocortisone ointment is usually helpful . *Discoid eczema, *pityriasis alba , *juvenile planter dermatitis , *nodular prurigo, *infraauricular dermatitis and fissures (infra-auricular fissures are specific of AD) .

*__Drug sensitivity__- e.g. anaphylactic type are more common in atopic persons , because of the increased propensity to produce IgE after natural exposure to antigens .

*__Alopecia areata__- there is a statistically significant association between AA and AD .

*__Urticaria__- contact urticaria for e.g. on the hands occurs not infrequently in AD and may present as an acute exacerbation of the AD .

*__Insect tings and bites- is similar in both atopic and non-atopic .__

Complications:

1. **Impact on quality of life**- AD disturbed the **psychosocial** aspects of patients lives and the lives of their families by disturbing the sleep pattern , behavior, also lead to delay growth .
2. **Bacterial infections**- 2ry bacterial infection by staph. or strep. which contributes to many exacerbations of the disease, e.g. impetigo .
3. **Viral infection**- both active and quiescent AD patients liable to develop acute generalized infection with herpes simple (eczema herpticum) and vaccinia (eczema vaccinatum) viruses , to produce the clinical picture of **Kaposi's varicelliform eruption** , which may present as a sever systemic illness with

high fever and a **widespread** eruption of papulovesicles , which rapidly ruptured to superficial scattered erosions or as extensive purulent exudates , which similar to impetigo and chicken pox **, other** viral infections like warts , MC. Are more common in AD patients **.**

4. **Ocular abnormalities-** Dennine-Morgan fold , conjunctival irritation, keratoconjunctivities , keratoconus (conical cornea), cataract .

Prognosis: the age of onset is less than 6mons**.** in 75% of cases and before the age of 5years in 80-90% . There is a general tendency towards spontaneous improvement through out children and often some slight relapse during adolescence . Relatively few typical **ca**ses persist over the age of 30years , perhaps 1/2 of all cases clear by the age of 13years , 40-60% clears with in 10-20% years , 84% with in 5-20years and 50%at 10years . The prognosis is worse if both parents are affected .

Diagnosis: the diagnostic criteria , the child must have : an itchy skin c**ondition (**or parental report of scratching or rubbing in a child) , plus 3 or more of the following :

1. Onset below age of 2years .
2. History of skin creases involvement (including cheeks in children under 10years .
3. History of generally dry skin .
4. Personal or first –degree relative history (children under 4years) of other atopic diseases .
5. **Visible** flexural dermatitis (or dermatitis of cheeks / forehead and outer limbs in children under 4years) .

Deferential diagnosis: scabies, infantile seborrheic dermatitis, immunodeficiency diseases . In adults : ACD, ICD, stasis dermatitis, pompholyx .

Investigation: IgE level, RASTS (Radioallergosorbent test) and prick test , bacteriological and virological tests for exacerbation .

Treatment: Nevertheless treatment should be tailored to an individual's needs, bearing in mind the age, sex, social conditions, sites of involvement and severity .

***First –line treatment:**

1. General advices- education about AD, types of trigger factors, treatment options, soaps , detergents, clothes, use central heating, wool, avoidance of aggravating food, stress, risk of viral infections (H.s.)and vaccinations .

2. Topical therapies: chronic lesions and lichenification are treated by ointments and acute oxidative lesions are treated by creams and lotions .

a. Bathing and emollients- bathing is soothing for the majority of patients and is helpful as long as the skin is moisturized immediately after words and avoidance of foaming detergents and soaps , ceramide-rich emollients are used which reduce the irritation and increasing the benefit of topical steroids .

b. Topical corticosteroids- are predominant treatment for the inflammation of AD and if not abused are very safe . The strength and mode of application of the topical steroids depends on the severity of the AD , sites and age of the patient . **A general principle** is to use topical steroid strong enough to settle sever dermatitis by twice daily application for 3-7days and then to reduce either in frequency of application or the steroid potency for maintenance therapy . **For resistant or infected or crusted AD** , combination of steroid/antibiotic or steroid/antiseptic is more effective , **tack care when steroid and antiseptic in children** , to avoid the side effects of abuse , which include systemic toxicity due to clioquinol antiseptic , steroids telangiectasis on cheeks in babies , striae of the breast , abdomen and thighs in adolescents and around the eyes may cause glaucoma .

c. Ichthamol and tar: preparations containing ichthamol and coal tar may be helpful as maintenance treatment in patients with lichenification, 1-10% coal- tar solution in an appropriate cream or ointment is preferred to crude coal tar .

3. Oral therapies:

a. Antihistamines – are used to control itching , H1-reseptors antagonists are used predominantly for their sedative effect , the non-sedating antihistamines are of little value e.g. promethazine or trimeprazine given 1ho. before bedtime for 10-14days to avoid tachyphylaxis .

b. Antibiotics- when there is exudation and pustules formation (means staph. infection) , an oral antibiotics such as flucloxacilin or erythromycin are indicated , also combination of topical steroid and antibiotics are of some benefit . H.S. is treated by acyclovir .

*__Second-line treatment :__

Patients who fail to respond to first line treatment should be reviewed to check, ***compliance ,** to exclude ***antibiotic resistant infections ,** or **herpes simplex infection** and to consider second-line treatment .

1. **Intensive topical treatment-** increase the strength of topical steroid for a short period as an outpatient .
2. **Wet-wrap technique-** it is used for the control of severe AD in younger children . Two layers of absorbent tubular bandage are applied to the skin , the inner layer is presoaked in warm water and outer layer is dry . A generous quantity of low-potency topical steroid is applied to the skin before the dressing , which can be used overnight or changed every 12ho. **(may suppress hypothalamopituitary gland) .**
3. **Immunomodulatory creams-** non-steroidal anti-inflammatory topical compounds e.g. tacrolimus , pimecrolimus . Tacrolimus used as 0.1% ointment in adults and 0.03%ointment in children is indicated for moderate to sever AD that has failed to respond to conventional therapy . Pimecrolimus used as 1% cream , is selective inhibitor of inflammatory cytokine . Both are safe and effective .
4. **Allergy management –** 80% of patients with AD show IgE hyperreactivity to common allergens . Foods allergens may aggravate the AD in about15-35% of children , but not adolescent and adults , so advice breast feeding for at least 6months in infants , with AD , with avoidance of cow's milk . Also avoidance of inhalants is of some value (e.g. HDM) and contact allergen also should be avoided (contact urticaria) .

*__Third- line treatment :__

1. Phototherapy – these include UVB, narrow-band UVB , medium and high dose UVA1 , and PUVA, avoid combination with immunosuppressant .

2. Oral immunosuppressants- it is used for recalcitrant cases, for short time e.g. *****cyclosporine for adult and childhood phase of AD , side effects are hypertension , renal toxicity and risk or skin cancer .

*****Oral corticosteroid , have a limited but definite role in the management of sever exacerbation of AD , used as short course .

*****Azathioprine , is an other immunosuppressive but slow onset of action .

3. Other therapies- IFN-gamma , type-4 phosphodiesterase inhibitors . IV immunoglobulin , and oral ketakonazole .

4. Desensitization and immunotherapy- desensitization plays a very limited part (role) in the management of AD , even when an allergic factor has been firmly

established clinically , but immunotherapy give an encouraging results e.g. Mycobacterium vaccine (killed) .

*__Diseases prevention and occupational advices:__

*****Breast feeding or diet to avoid potential allergens during the first 6months of the infant's life .

*****Avoidance of exposure to chemical irritant and physical trauma during adolescent and adult life .

Seborrhoeic dermatitis-SD *(dermatitis of sebaceous areas).*

Definition: It is a chronic type of dermatitis with distinctive morphology , consisted of red, sharply marginated lesions covered with greasy-looking scales and distinctive distribution in areas with a rich supply of sebaceous glands , namely the scalp , face and upper trunk . **Dandruff** (scaly scalp) appears to be the precursor of seborrhoeic dermatitis .

Incidence: The prevalence of SD is approximately 1-3% in the general population of USA , 3-5% in young adults and 36% of HIV patients . **In Iraq** (Baquba) , the prevalence is 8% of all types of dermatitis and 1.6% of all skin diseases .

Etiology: It is not well known , but the following factors are of some importance :-

1. ***Malassezia yeast*-**is increased in the scaly epidermis of dandruff and SD , so suggested as the cause of this disease (most important factor) , also response of SD to treatment with antiyeast therapy .
2. **Sebaceous glands-** SD is the disease of young adult e.g. it's peak between 18-40years , during which the sebaceous glands become mature and active , so play a role in the pathogenesis of SD .
3. **Sebum-** the pooling of sebum in immobile skin may important in the SD e.g. in parkinsonism , SD responded well to treatment with levodopa , which reduce sebum excretion , also facial nerve palsy cause unilateral SD of the face , paraplasia and hemiplasia of the trunk .
4. **Increased susceptibility-** of seborrhoeic skin to bacterial infection and HIV infection , suggested some exogenous role in the SD , also physical and chemical injury may play a role .
5. **General medical disorders e.**g. MI, malabsorption , epilepsy , obesity and alcoholism , also are suggested .

Pathology: It is not diagnostic generally shows features of both psoriasis and chronic dermatitis , parakeratosis , slight to moderate acanthosis with slight spongiosis and mild chronic inflammatory infiltrate .

Clinical features:

The disease is more common in males than females , they commonly originate in hairy skin and involve the scalp, face, presternal , interscapular region and the flexures , the lesions tend to be dull or yellowish red in color and covered with greasy scales .

*__Scalp-__ SD is usually manifested as dandruff and at later stage , perifollicular redness and scaling gradually extend to form sharply marginated patches that may remain discrete or coalesce to involve the greater part of the scalp and extend beyond the frontal hairline as the **corona seborrhoeicca .** Chronic cases may be associated with reversible hair loss . The SD may involve the postauricular fold , give a red, greasy scaling and crusted fissure and may involve both sides of the pinna, periacuricular region and the sides of the neck .

*__Face:__ SD involves the medial part of the eye brows, glabella and the naso-labial folds, as an areas of erythema and scaling , usually in association with scalp

involvement , blepharitis is common , the margins of the lids are red and covered by small white scales , yellow crust may form and separate to leave small ulcers which heals with scars and destruction of lash follicles .

*__Trunk:__ The most common form is the pataloid form (petal-shape) , often seen in man on the front of the chest and interscapular region , the initial lesion is a small red-brown follicular papules , covered by a greasy scale , which may remain as such with widespread eruption or more often extension and confluence of the follicular papules give rise to a figured eruption, consisting of multiple circinate patches with a fine branny scaling in their centers and with dark-red papules with larger greasy scales at their margin . A rare form involving the trunk and the limbs is the so called pityriasiform type which is a generalized erythema to squamous eruption , some what similar to but more extensive than pityriasis rosea , involving the neck up to the hair margin and usually non-pruritic and resolve spontaneously .

*__Flexures:__ Notably the axillae , the groins , the anogenital , submammary regions and umbilicus , SD presented as intertrigo with diffuse , sharply marginated erythema and greasy scaling , crusted fissures and with sweating , secondary infections and inappropriate treatment , a weeping dermatitis may extend far beyond them , the genitilia of both sex may be involved .

All forms of SD shows a tendency to chronicity and recurrence occasionally may become generalized , resulting in erythroderma .

Diagnosis: by clinical features and excluding of the following :- Scalp- psoriasis , lichen simplex chronicus, infective dermatitis (pediculosis) , CD, tinea capitis, PR, candidiasis, erythrasma . Flexures- pemphigus erythematousus , pemphigus foliaceous, Hayli-Hayli disease , T.V. , Darier's disease , and acrodermatitis enteropathica .

Treatment: there is no permanent cure , what ever the treatment :

*__Dandruff__ –is usually treated by the frequent and regular use of medicated shampoos , which act against malassezia yeasts , e.g. selenium sulphide , ketoconazole, tar shampoos , 1%terbinafite solution , for sever dandruff with persistent scaling or crusting, 5%salicylic acid ointment may be useful , treatment of secondary bacterial infection by erythromycin or flucloxacillin .

*__Acute forms of SD:__ on the face and trunk usually responded to mild steroid ointment e.g. 0.5%hydrocortison with 0.5%sulphur , but 2%ketoconazol cream is possibly a more logical therapy , combination therapy of both mild topical and ketoconazol is more effective for control of disease and ketoconazol cream is used for maintenance therapy .

*__For unresponsive cases: a course of UVB__ therapy may be helpful or even a short course of oral ketoconazol (200mg/day for 14days) , oral itraconazol 9100mg/day for up to 21days) is also effective as is oral terbinafine .

*__In generalized SD usually__ responds to the to the above medications , but for __recalcitrant__ cases systemic steroids may be required (30mg/day prednisolone, isotretinion may also be helpful .

*__Flexural SD :__ is treated in the same way as intertrigo .

Infantile seborrhoeic dermatitis.

Definition- it is a distinctive type of eczematous or psoriasiform eruption seen in infants, having a predilection for the scalp and the proximal flexures and a favorable prognosis compared with atopic dermatitis , it has no relationship with seborrhea and even sebum .

Etiology- not well established , but pityrosporum ovale (normal skin flora) has a possible etiological role (so responded to topical ketaconazol) .
Clinical features- the eruption generally first appears between second week of life and sixth month , but perhaps most frequently between the third and eight weeks . It may start (often) on the face , scalp, napkin area and occasionally on the trunk simultaneously , so all these areas may be involved rapidly and even other flexures like axillae may be involved . On scalp it involve the vertex and frontal head , on the face it involve the forehead , eyebrows, eyelids and nasolabial folds , become confluent around the neck up the sides of the face to the temples , behind the ears , napkin folds and trunk (umbilical area) are also involved . The rash consists of well-defined areas of erythema and scaling with tiny vesicles, the scales are yellow-brown in color, large and greasy in the scalp , but smaller , whiter and drier in other areas . Typically the infant is well with relatively mild pruritus .
Diagnosis- on clinical criteria , in differential diagnosis : psoriasis, cradle cap, intertrigo, primary irritant napkin dermatitis , AD, zinc deficiency , primary immunodeficiencies , Langerhan's histocytosis and multiple carboxylase deficiency .
Prognosis- even without treatment is good and clearance can be anticipated with in few weeks in the majority of cases , relapses are unusual after clearance , persistence suggests that the correct diagnosis is AD , psoriasis, zinc deficiency , histocytosis or immunodeficencies .
Treatment- as in AD , bath at least once daily and bath oil may added to the water , avoid soap and after bathing , a topical antiyeast (2%ketaconazol cream) , once or twice daily for 10-14days , scalp treated by 2%ketaconazol shampoo , avoid use of salicylic acid or corticosteroids .

Asteatotic eczema.
Definition- is eczema associated with a decrease in skin surface lipids , it is called **senile eczema , eczema craquele .**
Etiology- although the condition is thought to be caused by a decrease in skin surface lipid , the exact pathogenesis of the skin changes is obscure . But a relevant factors can be considered , which include :-

1. A naturally dry skin and a life long tendency to chapping .
2. A further reduction in lipid with age, illness, malnutrition or hormonal decline.
3. Increased transpiration relative to the environmental water content .
4. Loss of integrity of the water reservoir of the horny layer .
5. Chapping and degreasing by industrial or domestic cleansers or solvents .
6. Low environmental humidity and dry , cold winds increasing convection loss .
7. Repeated minor trauma leading to inflammation and further disorganization of the surface aqueous-lipid balance .
8. The other possible factors are diuretics , myxoedema , zinc deficiency , cimetidine and topical steroid .

These factors increased percutaneous absorption and epidermal damage and development of eczema .
Clinical features- It is a mild sub acute eczema , occurs in elderly (after age of 50years) , particularly on the legs , arms and hands, more marked in the winter . The skin is dry and slightly scaly , the dorsal surface of the hands shows marked criss-cross fashion , the finger pulps are dry and cracked producing distorted prints , on the legs pattern of superficial markings is more marked and deeper (crazy-paving pattern or eczema craquele) , some times the fissures may become hemorrhagic . The borders of this irregular reticulation become erythematous and slightly raised and frank

eczematous changes finally develop . The condition can remain in this state for months , relapsing each winter and clearing in summer , but eventually becoming permanent . Scratching , rubbing or contact irritants and sensitizers may aggravate the condition and more diffuse vesiculosquamous eruption occurs with intense irritation , which become worse with changes of temperature, particularly on undressing at night.
Treatment- *immediate adjustment of the patients environment .
*Central heating should be humidified where possible and abrupt temperature changes should be avoided .
*Wool also should be avoided .
*Baths are best restricted and should not be hot , bath oils or oatmeal packs are helpful .
*Emollients should be used after bathing or daily (e.g. creams based on lanolin or mixtures of lanolin and paraffin .
*Weak topical steroids and those contained in a urea base are very appropriate (urea encourages hydration) .
*Avoidance of soap and detergent cleansers , so emulsifying ointment BP (hydrophilic ointment), or oatmeal or bran can be substituted .
An extensive or generalized forms of eczema craqule involving the trunk as well as the legs are rare , but should be raise the suspicion of internal malignancy (lymphoma, angioimmunoblastic lymphadenopathy , anaplastic gastric adenocarcinoma and spheroidal cell carcinoma of the breast .

Discoid eczema (nummular eczema):
Definition- it is a type of endogenous eczema , characterized by a single , non-specific morphological features , namely circular or oral plaques of eczema with a clearly demarcated edge . It's prevalence in Iraq 3%of all type of eczema .
Etiology- in most cases the cause is unknown, but many factors are implicated :-
*Atopy may increase the incidence .
*Staphylococcal infection may increase the severity .
*Local physical or chemical trauma .
*Dry skin and low environmental humidity .
*alcohol intake .
*Allergy to (aloe, depilating creams, mercury and methyldopa) .
*Emotional stress may have a role in some cases .
Discoid eczema is relatively rare in children .
Clinical features- it is a sub acute dermatitis , the diagnostic lesion of discoid eczema is a **coin-shaped** plaque of closely set, thin-walled vesicles on an erythematous base , with arises quite rapidly from the confluence of tiny papules and papulovesicles . **Discoid eczema** may occur in two phases :- **Either** a very acute dissemination as individual lesions on the trunk or limbs , which are dull red , oozy, crusted and highly irritable , **or localized plaques .** The acute phase progress towards a less vesicular and more scaly stage , often with central clearing and peripheral extension causing ring shaped or annular lesions , as they fade they leave dry scaly patches .
After any period between 10days and several months , secondary lesions occurs on the opposite side of the body . A characteristic feature of this disease is that the patches which have apparently become dormant may become active again , particularly if treatment is discontinued prematurely . The following patterns are recognized : *discoid eczema of the hands and forearms, *discoid eczema of legs and trunk , ***Dry discoid eczema-** it is an uncommon variant , consisting of multiple dry, scaly, round, or oval discs on the arms or legs , but also with scattered micro vesicles

on an erythematous base on the palms and soles , itching is minimal , in contrast with other forms of discoid eczema and the condition persist for several years , with fluctuation or remission , it is notably resistant to treatment .
Diagnosis- D.D. ring worm, ACD, ICD, psoriasis, superficial scaly dermatitis .
Treatment- *general considerations (avoidance of irritants) .
*Emollient and topical corticosteroids with added cliquinol or antibiotic are useful .
In the early stages a potent steroid may be needed , **coal-tar pastes** or ointment may be added in the less acute stages , some times combination of both steroid and tar . **A course of a broad-spectrum systemic antibiotic** (e.g. erythromycin) is used in sever oxidative cases , **oral steroids** may occasionally be required for sever and extensive cases .

Hand eczema:
It is dermatitis largely confined to the hands, if it is a part of wide spread eczema , it is preferable to speak of hand involvement .
Etiology:
1. exogenous causes:-
a. Contact irritants –ICD due to soap, detergents, solvents --- .
b. Contact allergens-ACD due to type-4 immune reaction e.g. chromium, rubber---and immediate hypersensitivity due to type-1 e.g. seafood .
c. Ingested allergens - e.g. drugs, nickel, chromium .
d. Infective – e.g. following bacterial infection of the hand wounds .
e. Secondary dissemination –e.g. dermatophytide .
2. Endogenous causes:-
a. Idiopathic – e.g. discoid eczema, hyperkeratotic palmer dermatitis .
b. Immunological or metabolic defect -e.g. AD .
c. Psychosomatic- stress aggravates , but may not be causative .
d. Dyshidrosis- increased sweating aggravates , but may not be a cause .
Morphological patterns of hand eczema :
Pompholx , recurrent focal palmer peeling, hyperkeratotic palm eczema , ring eczema, Wear and tear dermatitis (dry housewives palmer eczema) , finger tip eczema, Apron eczema , discoid eczema, chronic acral dermatitis , gut eczema , other e.g. patchy vesiculosquamous .

Pompholyx (Dyshidrotic eczema , vesicular eczema of palms and soles):
Definition- it is a form of eczema of palms and soles in which fluid accumulates to form visible vesicles or bullae and because of thick epidermis the blisters tend to become larger before they burst and appears as deep seated , it's incidence is about 5-20% of all cases of hand eczema .
Etiology- the cause remain obscure , hereditary predisposition is suggested , nickel sensitivity , atopy , primary irritants , allergens both topically and orally , dermatophytide, bacteride , stress, drug eruption and cigarette smoking .
Clinical features- it may occur at any age , but it is more common before the age of 40 years , unusual before 10 years . An attack of pompholyx is characterized by the sudden onset of crops of clear vesicles , which appears deeply seated and sago-like , no erythema , but sensation of heat and prickling of the palms may precede attacks . Vesicles may become confluent and present as large bullae , especially on the feet , itching may be sever preceding the eruption of vesicles . The attack subsides spontaneously and resolution with desquamation occurs in 2-3weeks in most cases , but recurrent attacks in this period may cause a wave-like continuation of symptoms

in a majority of cases . In mild cases only the sides of the fingers may be affected , but in typical cases the vesicles develops symmetrically on the palms and /or soles . In 80% of cases only the hands are involved , hand and feet accounts 10% , feet alone 10% . Secondary bacterial infection lead to pustule formation and lyphangitis (not-uncommon) . Recurrent attacks may cause nail dystrophy , transverse ridging , pitting thickening and discoloration , the recurrence may occur at intervals of 3or 4 weeks to months or years and common in warm weather .

Diagnosis- D.D. dermatophytosis , psoriasis, ICD, ACD, pustular bacteride .

Treatment – any obvious cause of the eruption should be eliminated .

*In ac**ute** phase – rest wet soaks (3-4 times/day , soaked the hands and the feet in burrow's solution (1%Al-acetate) or potassium permanganate sol. (dilution 1:80000) . Large bullae may be aspirated by using sterile syringe , systemic antibiotic for secondary bacterial infection . As the eruption subsides the soaks should be discontinued and zinc cream or oily calamine lotion can be substituted , topical **steroids are useful in the sub acute and chronic phase and in sever cases oral steroid** may be used . For chronic cases with hyperkeratosis 2-3%crude coal tar alone or with steroid , low dose methotraxat and radiation therapy for refractory cases .

Recurrent focal palmer peeling:

It is a mild form of pompholyx , occurs during summer months , as small areas of superficial white desquamation develop on the sides of the fingers , palms and /or the feet . They appears abruptly and extend before peeling off , there is little or no irritation, no vesicles as such , it is not rare condition , asymptomatic , may subsequently progress to true pompholyx .

Hyperkeratotic palmer eczema:

It is a highly irritant , scaly fissured, hyperkeratotic patches on the palms and palmer surfaces of fingers , of unknown etiology , most frequent in men of middle age of over and is extremely refractory to treatment . PUVA , steroid ointment , crude coal tar , salicylic acid and Grenz rays and oral retinoid tablets , as tretinate .

Venous eczema (Gravitational eczema , stasis eczema):

It is eczema secondary to venous hypertension (varicose) .

Pathogenesis- 1. The oxygen content in the femoral venous blood of the leg affected by venous hypertension is increased and venous blood in such limbs has a faster circulation time than normal , which explain the development of arteriovenous shunts and development of dermatitis .

2. An alternative explanation suggested that the high ambulatory venous pressure with in the calf muscle pump is transmitted to the capillary circulation in the skin and subcutaneous tissues of the calf , which widens the endothelial pores , thus allowing fibrinogen molecules to escape into the interstitial fluid , which form a fibrin sheath around the capillaries , which acts as pericapillary barrier to the diffusion of oxygen and other nutrients that are essential for the normal vitality of the skin , so results in dermatitis .
3. Other suggestion is that cutaneous inflammation in venous hypertension may result from increased sequestration of WBC in the venules , with consequent release of proteolytic enzymes and free radicals which produce tissue damage and dermatitis .

Clinical features- the lesion consists of an erythematous , scaly and often oxidative eruption usually seen around the ankle and lower leg . It often occurs as a late result

of deep-vein thrombosis , the patient is usually middle-aged or elderly and most often female , obese, presumably a result of hormonal effects and the tendency for deep-vein thrombosis to occur during pregnancy . The eczema is often accompanied by other manifestation of venous hypertension , including dilatation or varicosity of the superficial veins , edema , purpura , haemosiderrosis , ulceration or small patches of white atrophic telangiectatic scarring (atrophic blanche) . Secondary patches of eczema may develop on the other leg , even with out obvious venous insufficiency and generalized secondary dissemination may occur and on occasions this can progress to erythroderma . Secondary contact dermatitis , infection and rubbing may be modified the picture .

Diagnosis- deferential diagnosis – atopic dermatitis, ACD , infective eczema, discoid eczema, asteatotic eczema , psoriasis , hypertrophic lichen planus , dermatophytosis, actinic keratosis .

Treatment – control of underlying hypertension by decreasing of weight in obese , well –fitted support socking or firm bandage , elevation of the leg during the rest , mild topical steroids to relive irritation , but potent steroids should be used for short periods (few days) , antibiotics for secondary infection and controlling of itching .

Juvenile planter dermatosis (Forefoot eczema , atopic winter feet):

It is a condition characterized by shiny , dry, fissured dermatitis of the planter surface of the forefoot , occurs mainly in children aged 3-14years , with possible association with atopy .

Etiology- not well known , but use of synthetic materials (nylon, plastics) in socks and shoes , enhance sweating and maceration , which end in this type of eczema .

Clinical features- it is the disease of childhood , rarely seen in infants and adults , with slight predominance in male patients . The presenting features are redness and sourness on the planter surface of the forefoot , which assumes a shiny glazed and cracked appearance , which is most sever on the ball of the foot and toe pads and tends to spare the non-weight bearing instep, the toe clefts are normal , the lesions are symmetrical and occasionally the hands are affected similarly , especially in atopic subjects .

Diagnosis- in D.D. tinea pedis , ACD , psoriasis .

Treatment – most of cases will clear spontaneously during childhood or adolescence, but the condition may persist into adulthood . **Advice** the patients to change the non-porous footwear to 100%cotton socks and leather shoes or sandals , **in sever cases** with cracking and exudation , bed rest may be needed , a variety of topical preparations may help , including urea preparations, Lassar's paste , white soft paraffin or tar , but no single preparation is always effective .

Chronic persistent superficial scaly dermatitis (benign form of parapsoriasis en plaque).

Definition- it is a chronic condition characterized by the presence of round or oval erythematous , slightly scaly patches on the limbs and trunk , which histologically shows mild eczematous changes with little or no dermal infiltrate . Although the condition is clinically benign by definition , but may represent a clinical presentation of mycosis fungoides (T-cell lymphoma) .

Prelymphomatous eruption	**CPSSD**
Bizarre or angulated shape	Regular-round or oval shape
Fine scales	Coarser scale
May be irritable	Little or no irritation

Progress to cutaneous lymphoma	Dose not become malignant
Histology- absence of epidermal eczema with dermal infiltrate of atypical T-cells	Histology may be eczematous changes ,little or no dermal infiltrate

Clinical features- the cause is unknown with histological changes of very mild eczematous eruption (patchy parakeratosis , mild spongiosis, slight perivascular dermal infiltrate , chiefly lymphocyte) .

The disease occurs in all races , but it is probably rare in dark-skinned people , in most of cases the onset is in middle life , much more common in men than in women . The disease begins insidiously with one or more erythematous , slightly scaly patches, predominantly on the legs , trunk and arms , seldom involves the face , palms or soles. The patches are generally round or oval, but finger-like processes are common , the patches are usually about 2-5cm cross , pink , brown or slightly yellow , often slightly wrinkled , resembling cigarette paper . Symptoms are usually minimal , but some itching may occur , patches are more prominent in winter than summer , usually the condition is persistent through the life , but may be clears permanently .

Diagnosis- in D.D. discoid eczema , eczamatide , poikiloderma , early eczematous stage of mycosis fungoides .

Treatment- only symptomatic , to suppress irritation , mild steroid ointment , natural sunlight , broad-band UVB and PUVA , Narrow-band UVB .

Pityriasis alba:

Definition-it is a type of mild sub acute endogenous eczema characterized by hypopigmentation , which is usually preceded by some erythema and scaling . It is of unknown etiology , but some times it is a manifestation of atopic dermatitis . Iraqi prevalence 4%of all types of dermatitis .

Clinical features- it occurs predominantly in children between the age of 3-16years , both sexes are equally affected . The individual lesion is a round , oval or irregular plaque , red, pink or skin colored and has fine lamellar or branny scaling . Initially the erythema may be conspicuous and there may even be minimal serous crusting , later the erythema subsides completely and the lesion shows only persistent fine scaling and hypopigmentation , that commonly induces the patient to seek advice . There are usually several patches ranging from 0.5-2cm in diameter , but may be larger especially on the trunk . In children the lesions are often confined to the face and are most common around mouth , chin and cheeks , 20% of affected children the neck , arms and shoulders are involved as well as the face . The course is extremely variable , most cases persist for some months and some may still show hypopigmentation for a year or more after all scaling subsides .

Diagnosis- in D.D. vitiligo, discoid eczema, psoriasis (trunk) , mycosis fungoides (trunk) .

Treatment- response to treatment is often disappointing , mainly because the pigmentation takes a long time to recover . The scaling can be reduced by a bland emollient cream and for chronic lesions on the trunk mild tar paste may be helpful , mild topical corticosteroids are helpful if inflammation persists .

Lichenification .

Lichen simplex chronicus (Circumscribed neurodermatitis) .

Definition- lichenification is a pattern of cutaneous response to repeated rubbing or scratching , characterized histologically by a canthosis and hyperkeratosis and clinically by a thickened appearance of the skin , with accentuation of the surface marking . It is common in patients with atopic dermatitis , but may also be secondary

to other irritant dermatoses . The term **lichen simplex** is used where there is no known predisposing skin disorder and the term **secondary lichenfication** is applied where the excoriation is initiated by a pruritic dermatosis .

Etiology- the prevalence in Iraq was 4%out of all dermatoses , of not well known etiology , there are racial predisposition for the development of lichenfication and emotional tensions (stress) may perpetuate the disease in the predisposed subject .

Clinical features- the pruritus is the predominant symptom in all forms of lichenification and is often out of proportion to the extent of the objective changes , paroxysmal , out of great intensity and scratching tends to give great satisfaction initially . During the early stages the skin is reddened and slightly edematous and normal markings are exaggerated , the redness and edema subside and the central area becomes scaly , thickened and some times pigmented , surrounding this central plaque is a zone of lichenoid papules and beyond this an indefinite zone of slight thickening and pigmentation merges with normal skin . Lichen simplex is uncommon in childhood , the peak incidence is between 30-40years , but it is seen at any age from adolescence on ward , more common in women then men , single or multiple sites are involved , most commonly those sites that are conveniently and easily reached , which include the nape of the neck , lower legs, ankles, side of the neck , the scalp, upper thighs , vulva, pubis , scrotum , the extensor forearms , the external auditory canal and post auricular folds . Scaling , crusting , fissuring and secondary bacterial infection are frequent complications of lichen simplex . The secondary lichenification complicates persistent skin lesions of many type .

Diagnosis- D.D. lichen planus, lichen amyloidosus, psoriasis, chronic Trichofyton rubrum ring worm and contact dermatitis .

Treatment- control of psychological tension , sedation by sedative antihistamine , antibiotic secondary infection and potent steroid ointment under occlusion , tar past for chronic lesions . intralesional triamcinilone asetonid 10mg/ml .

Prurigo :

Definition- it is a group of skin diseases characterized by intensely pruritic papules or nodules . The main types are prurigo simplex and nodular type .

Prurigo simplex – is more common type and usually result from insects bite for e.g. popular urticaria .

Nodular prurigo:

It is characterized clinically by chronic , intensely pruritic nodules and histologically by marked hyperkeratosis and acanthosis with downward projections of the epidermis.

Etiology- the cause is unknown , but emotional stress seems to be a contributory factor in some cases . Approximately 65-80% of patients are atopic , in them , the age of onset may be earlier . In 20% the condition starts after an insect bite . The pathology is similar to that of lichen simplex , with greater hyperkeratosis and the downward projections of the epidermis so marked .

Clinical features- cases occurs at all ages, but mainly from 20-60years , both sexes are equally affected . The individual lesions range from small papules to hard globular nodules , 1-3cm in diameter , with raised warty surface , the early lesion is red and may show a variable urticarial component , pigmentary changes are common , crust and scale may cover recently excoriated lesions . The intervening skin often shows slight xerederma and there is often an irregular ring of hyperpigmentation immediately around the nodules . The number of lesions varies greatly and may be

very large and the nodules may be arranged in groups , they usually develop initially on the distal parts of the limbs and are worse on the extensor surfaces , trunk, face and even the palms can be affected . The disease runs a very protracted course .
Diagnosis- in D.D. hypertrophic lichen planus , bullous pemphigoid nodularis, ACD, porokeratosis , renal failure skin lesions , liver diseases , lymphoma , HIV infection .
Treatment- measures used to reduce excoriation (cutting the nails very short , wearing gloves and occlusion) , sedative antihistamines at night , topical potent steroid , but intralesional triamcinolone can be more beneficial , topical capsaicin and cryotherapy , systemic thalidomide , cyclosporine, azathioprine, PUVA, UVB , narrowband UVB , antidepressants and tranquillizers .

Erythroderma.
Definition- it is the term applied to any inflammatory skin disease that affects more than 90% of the body surface . It's annual incidence is 0.9/100000 population , male : female ratio 2-3:1 , age of onset , most of cases over 45years .
Etiology:

1. **Hereditary disorders** (ichthyosiform erythroderma, PRP) about 1% .
2. **Psoriasis , about 25%** of cases .
3. **Eczema,** of various types , about 40% .
4. **drugs, (e.g.** organic arsenic , gold, mercury, occasionally penicillin, barbiturates) , about 10% .
5. **Pemphigus foliage's about 0.**5% .
6. **Lymphomas and leukemia** about 15% .
7. **Other skin diseases (** lichen planus, dermatophytosis, crusted scabies , dermatomyositis, sarcoidosis, Haily-Haily disease , pemphigoid, toxic shock syndrome , DLE, Graft-versus-host disease , HIV infection .
8. **Unknown etiology** , about 8% .

Clinical features- histopathology of erythroderma is that of the underlying cause , which is diagnostic in about 50% of cases and that of non-specific dermatitis . Clinically erythroderma is usually of sudden onset , as well as to clinical features of the underlying disease , it start as patchy erythema which extends rapidly to become generalized and universal in about 12-48hours and may be accompanied by fever, shivering and malaise, sometimes hypothermia may develop . Scaling appears after 2-6days , often first in the flexures , than become generalized , either as large or fine and bran-like , at this stage the skin is bright red , hot, and dry palpably thickened , some times sever irritation , but sensation of tightness is more characteristic and many patients complain of feeling cold . When erythroderma has been present for some weeks , the scalp and body hair may be shed and the nails become ridged and thickened and may be also shed . Edema of the periorbital skin results in ectroption , widespread hypopigmentation may occur . There are usually slight to moderate lymphadenopathy with rubbery consistency (dermatopathic lymphadenopathy) .
Secondary hemodynamic and metabolic disturbances – chronic generalized erythroderma is associated with profound metabolic disturbances which includes :-

1. Marked increase in blood flow through the skin , which may result in high-out put cardiac failure , especially in elderly .
2. The increased skin perfusion may lead to hypothermia .
3. Disturbance of the body temperature regulation , with excessive loss of heat leads to compensatory hyper metabolism and raised basal metabolic rate .
4. Increased fluid loss by transpiration lead to dehydration .
5. The loss of exfoliated scale may reach 99/sequir meter body surface/day .

6. Hypoalbuminaemia is common , due to dilution by increased plasma volume, decrease synthesis , increased metabolism, increased protein loss via scaling and exudation .
7. Immune responses may become altered , reflected by an increase in Igs .

Prognosis and complications- erythroderma is a serious condition in it self , quite a part from hazards of the underlying disease and some times fatal, particularly in elderly people (mortality rate 18-64%) . The more common forms of erythroderma (eczematous, psoriatic, or of unknown origin) , may continue for months or years and tend readily to relapse . The causes of death are the above hemodynamic and metabolic disturbances , as well as cutaneous , subcutaneous and respiratory infections .

Diagnosis- is easy ,but the diagnosis of underlying diseases may be very difficult .

Treatment- *Generalized measures , which includes admission to hospital , correction of protein and electrolyte balance , circulatory status and body temperature , the environmental temperature must be carefully regulated , cooling and over heating must be avoided , blood urea and serum electrolyte and fluid balance should be monitored and treatment of complications and the underlying diseases .

***Specific measures-** includes firstly treatment of skin inflammation by soothing emollient creams or mild topical steroids . In sever persistent cases , systemic steroids are indicated , antibiotic to control secondary infections , **extracorporeal photophoresis has been advocated .**