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Essence of Practice of Medicine

JV'n Dr. Ravi Jain

JAYOTI VIDYAPEETH WOMEN'S UNIVERSITY, JAIPUR

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Essence of Practice of Medicine

Part 1

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Foreward

First of I would like to give my gratitude towards our Honourable Chairperson Mam Jv'nVidushiGarg and our Honourable Founder and Advisor sir JV'nPanckajGarg sir for providing me an opportunity to write this book and publish in University press for the need of our students at JayotiVidyapeeth Women's University Jaipur.

This book is dedicated to the Students of BHMS, BAMS, BNYS.The book will be published in 5 volumes. This part covers diseases ofUrogenital system and Dermatology in a short and easier way. Dermatology is difficult to understand even for an expert physician. Proper care has been taken to include pictures of the various diseases and have been placed with the diseases to get clear and easy understanding of the disease. Diseases of urogenital tract are very common these days and proper understanding of these diseases are very important for treatment of the patients. Hence these two topics are included in this first volume. The topics are explained in short and only points are given for a quick review. In further volumes more chapters will be included for the benefit of the students.

The matter is collected from very authentic sources in order to avoid any sort of controversy.

This provides a readymade instrument for quick review for many competitive exams and for the quick review during the theory main exams.

Although an attempt has been done to keep the accuracy. Although if any issue is found feel free to contact the author for changes in the subsequent editions.

Jv'nDr Ravi Jain

Author

Index

Chapter 1.1

Introduction to Diseases of Urogenital Tract

The Genitourinary system, or Urogenital system, is the organ system of the reproductive organs and the urinary system.

We study them together because :

Proximity to each other.

Their common embryological origin.

Use of common pathways.

Kidney and Urinary Tract Diseases: We will study the diseases under following headings

Anatomy of Kidney and Urinary Tract

Physiology (Renal Functions)

Renal Disorders

Assessment of Renal Disorders.

Examinations in cases of Renal disorders

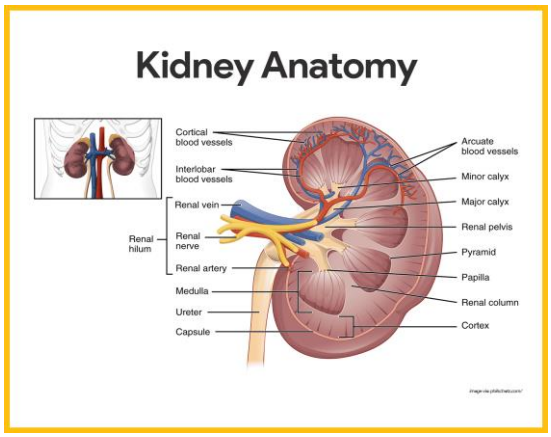


Fig1.1 Anatomy of Kidney

Functions of Kidney

Maintain volume and composition of body fluids.

Retain certain useful substances by renal threshold.

Excrete waste products.(urea, uric acid, creatinine).

Homeostasis of electrolytes, minerals, anions and cations, salt and water.

Metabolic and hormonal functions.

Functions of Genitalia

Male Genitalia :

Produce spermatozoa and testosterone – pubertal growth, prostate and seminal vesicle, development of secondary sexual characters.

Female Genitalia :

Ovaries produces ova and hormones.

Secondary sexual characters.

Renal Disorders: Different types of Renal disorders are commonly studied

Acute Renal Failure

Acute Nephritic syndrome

Nephrotic Syndrome

Chronic Renal Failure

Asymptomatic urinary abnormalities

Urinary Tract Infection

Renal Tubular Defects

Nephrolithiasis

Urinary Tract Obstruction

Assessment of Renal Disorders is done by:

Face : Puffiness

Eyes :Oedema

Ear : Tophi

Neck : JVP for fluid overload

Skin :Purpura, Bruising, Pruritis

Extremities :Oedema, Clubbing, Peripheral Neuropathy

Investigations to be done in case of Renal disorders

Urine Examination :

Volume

Colour and Transparency

Urine Concentration and Osmolality

Specific Gravity

Urine Dilution

pH

Abnormal Constituents of Urine

Proteinurea

Haematuria

Sugars

Ketones

Bilirubin

Cells

Casts

Crystals

Micro-organisms

Chapter 1.2

Urinary Tract Infection (UTI)

UTI is defined as the presence and multiplication of bacteria in the urinary tract.It cann be uncomplicated or complicated, upper or lower.The clinical features, diagnosis, treatment and prognosis depends on the organism, site of infection and structural and functional integrity of urinary tract.

It is an infection that affects part of the urinary tract.

Cystitis :when it affects the lower urinary tract it is known as a **bladder infection.**

Pyelonephritis :when it affects the upper urinary tract.

Signs & Symptoms

Burning with urination

Frequent urination

Pain above the pubic bone or in the lower back

Flank pain,

Fever

Nausea

Vomiting

Rarely bloody urine

Pus in urine

Causes of UTI

E. coli

Staphylococcus saprophyticus

Urinary catheterization

Intercourse

Contraceptives : spermicides, diaphragm, etc

Investigations

Urineanalysis : MSU

Urine may be turbid, blood stained and foul smelling.

pH depends on pathogenic bacteria.

Mild proteinuria

Urine microscopy shows pus cells and pus cell casts.

USG

Plain X Ray KUB

Intra venous Urography

CT

DMSA

Cystoscopy

Homoeopathic Management

Thuja Occidentalis

Nux vomica

Sarsaparilla

Aconitum napellus

Apismellifica

Belladonna

Berberis vulgaris

Borax

Cantheris

Staphysagria

Clematis erecta

Sulphur

Lycopodium

Chapter 1.3

Acute Renal Failure or Acute Kidney Injury

It Is characterized by the sudden impairment of kidney function resulting in the retention of nitrogenous and other waste products normally cleared by the kidneys. AKI is not a single disease but, rather, a designation for a heterogeneous group of conditions that share common diagnostic features.An increase in the blood urea nitrogen (BUN) concentration.An increase in the plasma or serum creatinine (SCr) concentration. Associated with a reduction in urine volume.

AKI is a clinical diagnosis and not a structural one.It can range in severity from asymptomatic and transient **changes in laboratory parameters** of glomerular filtration rate (GFR), to overwhelming and rapidly fatal derangements in effective **circulating volume regulation** and **electrolyte and acid-base composition** of the plasma.

A sudden and usually reversible loss of renal function, which develops over days or weeks and is usually accompanied by a reduction in urine volume.An abnormally high creatinine is important to establish if this is acute, acute on chronic, or chronic kidney disease.Measurements of renal function should be sought for comparison.A renal ultrasound demonstrating two small kidneys indicates chronicity.

Pathophysiological Classification

The classification is done according to the anatomical location:

Pre Renal

Intrinsic renal

Post renal

Pre renal Causes :

These aremost common.Renal dysfunction caused by decrease in renal perfusion.

Restoration of renal perfusion results in rapid recovery.

Intrinsic renal Causes : Most common cause is Acute tubular necrosis. These are caused by ischemic or nephrotoxic injury.

Post renal Causes : Due to urinary tract obstruction.

Clinical Features:

Pre renal failure :

Oliguria

Symptoms of Hypovolaemia, Thirst, dizziness, orthostatic hypotension.

Evidence of excessive fluid loss.

Intrinsic renal failure :

ATN should be suspected presenting after a period of hypotension, hemorrhage, sepsis, drug overdose, surgery.

Postrenalfailure : It occurs in old men with prostatic obstruction, symptoms of urgency, frequency, hesitancy, and obstruction to flow of urine.

Investigations

Urinalysis : Daily urine output. Presence of abnormal constituents.

Blood urea and Serum creatinine.

Urine electrolytes.

Ultrasound examination. For detecting intrinsic renal disease.

Serologic tests. Anitnuclear antibody.

Renal Biopsy : when diagnostic investigations are inconclusive.

Creatinine S

Female 44–80 μmol/L 0.5–0.9 mg/dL

Male 53–106 μmol/L 0.6–1.2 mg/dL

Urea nitrogen S 2.5–7.1 mmol/L 7–20 mg/dL

Management

Treatment of Underlying cause

Maintenance of body fluids and electrolyte balance.

Maintenance of Nutritional status.

Monitor life threatening complications.

Renal replacement when indicated.

Chapter 1.4

Chronic kidney disease

It is an irreversible deterioration in renal function which classically develops over a period of years.Initially, manifested as biochemical abnormality.

There is loss of :

Excretory,

Metabolic and

Endocrine functions of the kidney leads to the clinical symptoms and signs of renal failure referred as Uraemia.

Normal GFR = 120^{+}. 25ml/min/1.73m^2

Uremic features includes

Anorexia, nausea and vomitingfollowed by drowsiness, apathy, confusion, muscle twitching hiccoughs, fits and coma.

Stages of Chronic Renal Diseases

Stage I : Kidney damage with normal or high GFR : > 90,

Stage II : Kidney damage with slightly low GFR : 60-89

Stage III : Moderately low GFR : 30-59

Stage IV : Severe low GFR : 15-29

Stage V : Kidney Failure : <15

Common Causes

Any condition which destroys the normal structure and function of the kidney.

Congenital and inherited - polycystic kidney disease,Alport syndrome

Renal artery stenosis

Hypertension

Glomerular diseases

Interstitial diseases

Systemic inflammatory diseases – SLE, vasculitis

Diabetes mellitus

Unknown

Retarding Progression of CKD

Plasma creatinine above 3.4mg/dl, progressive deterioration in kidney function irrespective of etiology.

Control of Blood Pressure.

Diet: restrict protein

Management of Adverse effects of CKD

Anemia

Fluid and electrolyte balance

Acidosis

Infection

Bleeding

Renal osteodystrophy

Myopathy

Others

Anemia: correlates with severity of CRF

Causes:

Relative deficiency of erythropoietin

Diminished erythropoesis due to toxic effects of uremia

Reduced red cells survival

Increased blood loss due to capillary fragility

Reduced intake and absorption.

Rx - Iron supplementation is needed

Fluid electrolyte balance : reduced ability to concentrate urine.

Limitation of potassium intake (70mmol/day) and sodium intake (100mmol/day).

Acidosis : declining renal function is associated with metabolic acidosis.

Sodium bicarbonate supplements.

Cardiovascular disease and lipids:

Atherosclerosis is accelerated by hypertension. Vascular calcification develops. Hypercholestrolemia is significant with proteinuria and increased triglyceride level.

Treatment of these abnormalities.

Infection : Second most common cause of death in dialysis patient. Cellular and humoral immunity is impaired.

Treatment of the cause.

Bleeding :

Platlet function is impaired and bleeding time is prolonged.

Cutaneous ecchymosis and mucosal bleeds.

Adequate dialysis corrects bleeding tandency.

Renal osteodystrophy :Mixture of osteomalacia, hyperparathyroid bone disease, osteoporosis, and osteosclerosis.

Deficiency of 1 aplha hydroxylase enzyme

Parathyroidectomy

Myopathy :

Generalised myopathy is due to combination of poor nutrition, hyperparathyroidism, Vit D Deficiency and disorders of electrolyte metabolism.

Other adverse effects :

Sensory neuropathy causes paresthesiae.

Motor neuropathy causes foot drop.

Uraemic autonomic neuropathy causes delayed gastric emptying, diarrhoea, and postural hypotension.

Gastrointestinal manifestations include anorexia, nausea, vomiting.

High incidence of peptic ulcer.

Depression is most common in patients approaching renal replacement therapy.

Chapter 1.5

Alport Syndrome

It affects adults. Autosomal recessive disease. It arise from mutation or deletion of Gene on X chromosome COL4A5 which encodes Type IV collagen. There is accumulation of abnormal collagen results in a progressive degeneration of GBM.

Affected patients progress from haematuria to ESRF in late teens or twenties.Associated with sensorineural deafness and ocular abnormalities.

Pathogenesis

Disturbances in water, electrolyte and acid base balance contribute to the clinical picture.

Exact pathogenesis is unknown.

Many substances in abnormal concentration in plasma.

Uremia is caused by accumulation of intermediary products of metabolism.

Clinical Features

Raised blood urea and creatinine.

Hypertension,

Proteinuria

Anaemia.

Diseases is Slowly progressive and patients are asymptomatic until GFR falls below 30 mL/min/1.73 m2

Nocturia , due to loss of concentrating ability and increased osmotic load.

Tiredness or breathlessness

Pruritus, anorexia, nausea and vomiting

Deep respiration related to metabolic acidosis, muscular twitching, fits, drowsiness and coma ensue.

Investigations

Urea and Creatinine

Urinalysis and quantification of proteinuria.

Electrolytes

Calcium, phosphate, parathyroid hormone

Albumin

Full blood count (± Fe, ferritin, folate, B12)

Lipids, glucose ± HbA1c

Renal ultrasound

Hepatitis and HIV serology

ECG

Other tests

Chapter 1.6

Glomerulonephritis

Glomerular diseases causes :

Proteinurea

Haematuria

Loss of filtration capacity (GFR)

Hypertension

Acute Glomerulonephritis

Most common in children but can occur at any age.

Pathologically diffuse inflammatory changes in glomeruli.

It is an immune complex mediated glomerulonephritis.

Preceded by **Streptococcal infection**.

Following streptococcal infection the streptococcal antigens stimulates antibody production.

The antigen antibody combine to form immune complexes.

It gets deposited in kidney tissues and initiate acute inflammatory reaction.

There are contributions of cellular immunity, humoral immunity, and other inflammatory mediators.

Clinically it is abrupt onset of :

Puffiness of face

Macroscopic hematuria

Proteinuria

Oedma

Hypertension

Impaired renal function with or without oliguria.

Clinical Features

Puffiness of the face.

Scanty and smoky urine, frank blood.

Infection symptoms : Fever, bodyache,vomiting.

Cerebral symptoms : Headache, convulsion.

Weakness, anorexia, pallor.

Accidental discovery on routine urine examination.

Signs & Symptoms

Oedema : Gradual or abrupt onset.

<morning.

Swelling of face to generalized anasarca.

Hypertension : Diastolic pressure 90-120mm of Hg.

Returns to normal after diuresis.

JVP is raised. Peripheral oedema with a picture of CHF.

Renal functions are impaired. : oliguria

Lab Investigations

Urine examination : Oliguria, reddish brown with protein. Red cells and casts are present.

Evidence of streptococcal infection : Presence of A Beta hemolytic streptococcus in throat or skin lesion.

Hematology :PolymorphoneuclearLeucocytosis, Raised ESR.

Osmolarity : Usually higher.

Renal biopsy : When the diagnosis is not clear.

Prognosis

90% of the children recovery is uneventful and complete.

Proteinuria and Hematuria may persist for few weeks to months before returining to normal.

Complications

Acute renal failure

Acute heart failure with pulmonary edema.

Hypertensive encephalopathy

UTI

Arthritis

Some children may develop Nephrotic syndrome or chronic glomerulonephritis.

Management

Bed rest : 3 weeks to 3 months.

Restricted fluids : Fruit juices, water with caution. Low salt, Low protein diet.

Antibiotics : Erythromycin, Benathinepenicilline.

Management of complications : Diazepam for **convulsion**, ACE inhibitors for **hypertension**.

Dialysis : in case of rapid rising serum potassium and blood urea.

Renal transplant

Chapter 1.7

Nephrotic Syndrome

A clinical condition in characterized by

Oedema

Proteinuria

Hypoproteinemia

Irrespective of etiology or any other additional abnormal clinical features.

A condition characterized by

Heavy proteinuria of > 3.5gm/1.73m2 body surface area in 24 Hrs.

It is associated with :

Hypoalbuminemia

Edema

Hyperlipidemia

Hypercoagulable state

Etiology

Idiopathic

Primary Glomerular disease

Secondary causes

Primary Glomerular disease

Minimal change disease

Focal segmental glumerulosclerosis.

Membranous nephropathy.

Membranoproliferative glomerulonephritis

IgA Nephropathy

Secondary Causes

Infections : Bacterial, viral, protozoal, helminthic etc

Neoplasm : Solid tumors : lung, stomach, breast, leukemia, etc.

Drugs : NSAID's, Penicillamine, gold, mercury, etc

Multisystem disease : SLE, rheumatoid arthritis, amyloidosis.

Familial and metabolic diseases : diabetes mellitus, graves disease, etc

Misc: pre-eclampsia, malignant nephrosclerosis, obesity etc.

Clinical Features

Age &Sex : Common in childhood with peak incidence 2-3 years. M:F 2.5:1. Equal in adults.

Oedema : Peripheral. Lower limbs. Face and abdomen affected in children. Generalized anasarca.

GI : Anorexia with severe malnutrition. Diarrhea and vomiting due to edema of intestinal wall.

General : Anorexia, lethargy, tiredness, frequent infections and muscle wasting. Dyspnoea may occur.

Blood Pressure : Hypertension.

Lab Investigations

Urine :

Oliguria

Proteinuria :> 5gm/day.

Casts : Fatty casts.

Blood :

Anemia : normochromic

Hypoalbuminemia

Hyperlipoproteinemia

Renal biopsy

Prognosis

90% children respond to prednisolone.

Adult response rate is 60%.

Relapse rate is high.

Management

General management includes fluid restriction, low sodium diet, rest. These helps in controling edema.

Corticosteroids : produce rapid and complete remission.

Immunosuppressive drugs : Cyclophosphamide.

Chapter 1.8

Nephritic Syndrome

Also called **Acute Glomerulonephritis**

It is characterized by :

Azotemia -abnormally high levels of nitrogen-containing compounds urea, creatinine etc.

Hypertension

Edema,

Hematuria, proteinuria, and sometimes oliguria.

Salt and water retention are due to reduced glomerular filtration rate (**GFR)** result **in circulatory congestion**.

Red blood cell (RBC) casts on urinalysis confirms diagnosis.

Clinical course of nephritic syndrome depends on underlying lesion :

Acute poststreptococcal GN

Postinfectious GN

Rapidly progressive glomerulonephritis

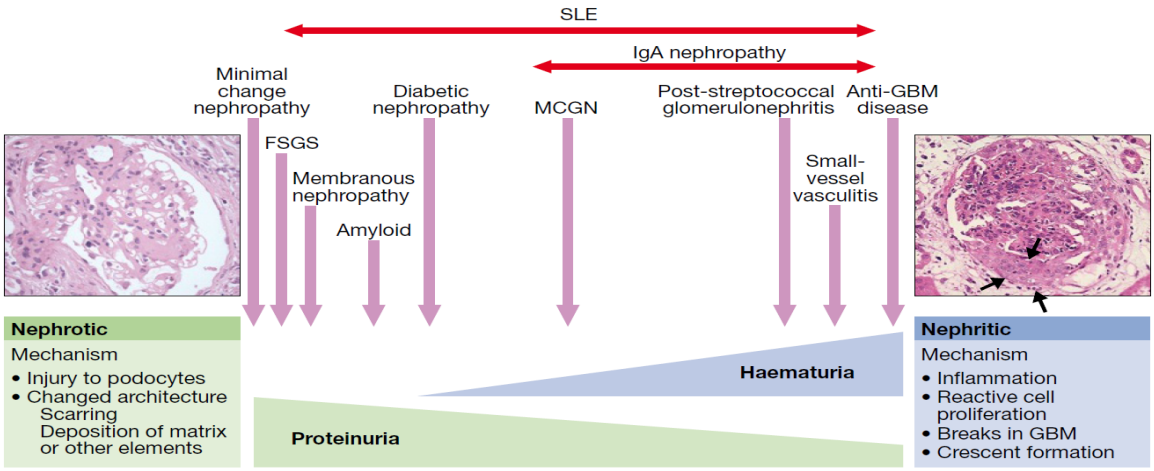


Fig 1.2 Glomerular diseases. Courtsy Harrison Principles of Internal Medicine 19th Edition

Classifications of glomerulonephritis are largely histopathological.

A renal biopsy is required to make the diagnosis and determine therapy in NS.

Glomerulonephritis = Nephrotic Syndrome + Nephritic Syndrome.

Minimal change nephropathy

Primary focal segmental glomerulosclerosis (FSGS)

Membranous nephropathy

IgA nephropathy and Henoch–Schönleinpurpura

Glomerulonephritis associated with infection

Acute post-infectious glomerulonephritis

Rapidly progressive glomerulonephritis

Minimal Change Nephropathy

Causes about 10–15% of idiopathic.

Occurs at all ages but 70–90% of NS in children.

Electron microscopy shows fusion of podocytes foot process.

Blood pressure is normal, GFR is normal or slightly reduced, urinary sediment is benign or may show few RBCs.

Proteinuria usually remits on high-dose corticosteroid therapy.

Relapse cases frequently need maintenance corticosteroids.

It does not progress to CKD

Primary focal segmental glomerulosclerosis (FSGS)

Glomerulosclerosis refers to scarring or hardening of the glomeruli.

It can be primary or secondary.

The primary FSGS group present with **idiopathic nephrotic syndrome** and no other cause of renal disease.

It recurs after renal transplantation, and sometimes proteinuria recurs almost immediately and may lead to loss of the allograft.

Treatment of primary FSGS typically begins with an extended course of steroids.

It shows little response to corticosteroid treatment and often progress to renal failure.

Secondary FSGS occurs in the **late stages of any form of kidney disease** associated with nephron loss.

It presents with variable proteinuria and outcome.

There is no benefit of glucocorticoids or other immunsuppressive agents in secondary FSGS.

Clinical history, kidney size, biopsy findings, and associated conditions helps in **differentiation of primary from secondary causes.**

The histological appearances of FSGS but lesser proteinuria, focal scarring reflects healing of previous focal glomerular injury.

There is no specific treatment.

Membranous Nephropathy

The most common cause of nephrotic syndrome in adults.

It is characterized by subepithelial**IgG deposits**.

Patient present with edema and nephrotic proteinuria.

Blood pressure, GFR, and urine sediment are usually normal at initial presentation. Hypertension, mild renal insufficiency, and abnormal urine sediment develop later.

One-third **remit spontaneously**, one-third remain in a **nephrotic state**, and one-third show progressive **loss of renal function.**

Treatment with high doses of corticosteroids and alkylating agents improve both the nephrotic syndrome and the long-term prognosis.

IgA nephropathy and Henoch– Schönleinpurpura

IgA nephropathy is the most commonly recognised type of glomerulonephritis and can present in many ways.

Haematuria is the earliest sign and is almost universal,

Proteinuria a later feature

Hypertension very common

In some cases severe proteinuria or loss of renal function can occur.

It is a common cause of ESRD

The exacerbations in young adults are acute self-limiting, often with gross haematuria, in association with minor respiratory infections.

The exacerbations are so acute as to resemble **acute post-infectious glomerulonephritis**, with **fluid retention, hypertension** and **oliguria** with dark or red urine.

The duration from clinical infection to nephritis is short, a few days or less.

IgA nephropathy progresses rapidly and crescent formation may be seen.

Systemic vasculitis in children and adults in response to infections is called **Henoch–Schönleinpurpura**.

It is characterized by :

Petechial rash (cutaneous vasculitis) typically affecting buttocks and lower legs.

Abdominal pain (gastrointestinal vasculitis)

Mild glomerulonephritis indicated by haematuria.

Renal biopsy shows mesangial IgA deposition and appearances indistinguishable from acute IgA nephropathy.

The response to immunosuppressive therapy is poor.

The management of less acute disease is control of blood pressure to prevent or retard progressive renal disease.

Glomerulonephritis associated with infection/ Mesangiocapillary glomerulonephritis

Subacute bacterial infections(bacterial endocarditis), causes a variety of histological patterns of glomerulonephritis, most typically with membranous and mesangiocapillary lesions(MCGN), with plentiful immunoglobulin deposition.

Glomerulonephritis more commonly occurs following hepatitis B, hepatitis C, schistosomiasis, leishmaniasis, malaria and other chronic infections.

Proving a causative relationship between renal disease and infection in individual cases is difficult.

Acute post-infectious glomerulonephritis

Most common following infection with certain strains of streptococcus and therefore is often called post-streptococcal nephritis.

More common in children.

Latency is about 10 days after a throat infection or longer after skin infection.

Rare in the developed world (Compare IgA nephropathy is most common)

It suggests an immune mechanism rather than direct infection.

Symptoms : Sodium retention, hypertension and oedema.

Reduction of GFR, proteinuria, haematuria and reduced urine volume.

Urine has a red or smoky appearance.

There are low serum concentrations of C3 and C4.

Evidence of streptococcal infection ASO titre, culture of throat swab, and other microbiological sampling.

Management by fluid and sodium restriction and use of diuretic and hypotensive agents.

Treatment consists of correction of fluid and electrolyte imbalance.

It usually resolves spontaneously.

Rapidly Progressive Glomerulonephritis

An extreme inflammatory nephritis which causes rapid loss of renal function over days to weeks.

A subacute reduction in GFR of >50%, with evidence of a **proliferative GN**, causes overlap with those of **acute GN.**

Three major subtypes on the basis of renal biopsy findings and pathophysiology:

Immune complex–associated, e.g., in systemic lupus erythematosus (SLE).

Pauci-immune associated with antineutrophil cytoplasmic antibodies (ANCA).

Associated with anti–glomerular basement (anti-GBM) antibodies, e.g., in Goodpasture's syndrome.

SLE (Lupus)

Renal involvement is due to deposition of circulating immune complexes.

Non renal symptoms of SLE include arthralgias, "butterfly" skin rash, serositis, alopecia (hair loss), and central nervous system disease.

Nephrotic syndrome with renal insufficiency is common.

Renal biopsy reveals mesangial, focal, or diffuse GN and/or membranous nephropathy.

It is characterized by an active sediment, severe proteinuria, and progressive renal insufficiency.

Treatment includes glucocorticoids and cytotoxic agents. Oral or IV monthly cyclophosphamide for a period of 6 months.

Antineutrophil Cytoplasmic Antibody (ANCA) Focal Segmental Necrotising glomerulonephritis.

Defining characteristic is the presence of circulating ANCA.

These are detected by immunofluorescence of alcohol-fixed neutrophils.

Symptoms : prodromal, "flulike" syndrome, which may encompass myalgias, fever, arthralgias, anorexia, and weight loss.

Treatment of rapidly progressive GN includes methylprednisolone and cyclophosphamide, corticosteroids and cytotoxic agents.

Segmental inflammation and or necrosis in some glomeruli.

Small Vessel Vasculitis.

Anti–Glomerular Basement Membrane Disease (Goodpasture's syndrome)

It is caused by antibodies against the α3 NCI.

Circulating anti-GBM antibody and linear immunofluorescence on renal biopsy establish the diagnosis.

Goodpasture's syndrome encompasses GN and lung hemorrhage.

Treatment with corticosteroids, cyclophosphamide and plasma exchange to remove circulating autoantibodies.

Chapter 1.9

Nephrolithiasis

Renal calculi are common, affecting ~1% of the population, and recurrent in more than half of patients.

Stone formation begins due to supersaturated of urine with insoluble components due to :

Low urinary volume

Excessive or insufficient excretion of selected compounds,

Other factors (e.g., urinary pH) that diminish solubility.

Types of Calculi

Approximately 75% of stones are Ca-based **Ca oxalate**, Ca phosphate and other mixed stones),

15% struvite (magnesium-ammoniumphosphate),

5% uric acid,

1% cystine,

Reflecting the metabolic disturbances from which they arise.

Signs & Symptoms

Asymptomatic or cause hematuria alone.

With passage, obstruction may occur at any site along the collecting system.

Severe pain, often radiating to the groin.

Nausea, vomiting, diaphoresis, light-headedness.

Hematuria, pyuria, urinary tract infection (UTI), and hydronephrosis.

Staghorn calculi may be completely asymptomatic, presenting with loss of renal function.

It is associated with recurrent UTI with urea-splitting organisms.

Symptoms

Pain : Ureteric colic : Cramping, sharp, often excruciating pain, fluctuating in intensity but not completely remitting.

Pain is associated with vomiting and sweating.

Pain extends from loin down the line of ureter to groin.

Loin pain.

Haematuria : with or without colic.

Signs

Loin tenderness : manifestation of associated infection.

Palpable kidney in case of staghorn calculus.

Stone Composition

Most stones are composed of **Ca oxalate.**

Associated with **hypercalciuria**and/or **hyperoxaluria**.

Hypercalciuria: very high-Na diet, loop diuretic therapy, renal tubular acidosis (RTA), sarcoidosis, Cushing's syndrome, aldosterone excess.

Conditions associated with hypercalcemia (e.g., primary hyperparathyroidism, vitamin D excess, etc).

It may be idiopathic.

Hyperoxaluriais seen with intestinal malabsorption syndromes (e.g., inflammatory bowel disease, pancreatitis),

Due to reduced intestinal secretion of oxalate and/or the binding of intestinal Ca by fatty acids within the bowel lumen.

With enhanced absorption of free oxalate.

Ca phosphate stones

Much less common and tend to occur at an abnormally high urinary pH (7–8), usually in association with a complete or partial distal RTA.

Struvite stones form in the collecting system when infection with urea-splitting organisms is present.

Proteus, Klebsiella, Providencia, Morganella, and others.

Struvite is the most common component of staghorn calculi and obstruction.

Risk factors include previous UTI, urinary catheters, and instrumentation.

Uric acid stones develop when the urine is saturated with **uric acid** in the presence of **an acid urine pH**.

Urine pH that is <5.4 and often <5.0.

Asscociated with

Clinical gout

Certain drugs e.g., probenecid, high-dose salicylates.

Cystine stones

More likely to form in acidic urinary pH.

Result of a rare inherited defect in renal and intestinal transport of several dibasic amino acids.

Overexcretion of cysteine.

They occasionally lead to end-stage renal disease.

Workup

Dietary and fluid intake history.

Careful medical history and physical examination, focusing on systemic diseases

Noncontrast helical CT, with 5-mm CT cuts.

Routine UA; presence of crystals, hematuria, measurement of urine pH

Serum chemistries: BUN, Cr, uric acid, calcium, phosphate, chloride, bicarbonate,PTH

Timed Urine collections : Cr, Na, K, urea nitrogen, uric acid, calcium, phosphate, oxalate, citrate, pH

Treatment

Depends on clinical history, and the metabolic workup.

Nncrease in fluid intake to at least 2.5–3 L/d.

Conservative recommendations for Ca oxalate stones :

low-salt, low-fat, moderate-protein diet.

Specific treatment depends upon the type of stone and managed accordingly.

Complications

Impaction and obstruction : at PUJ.

Infection : Pyelonephritis, cystitis.

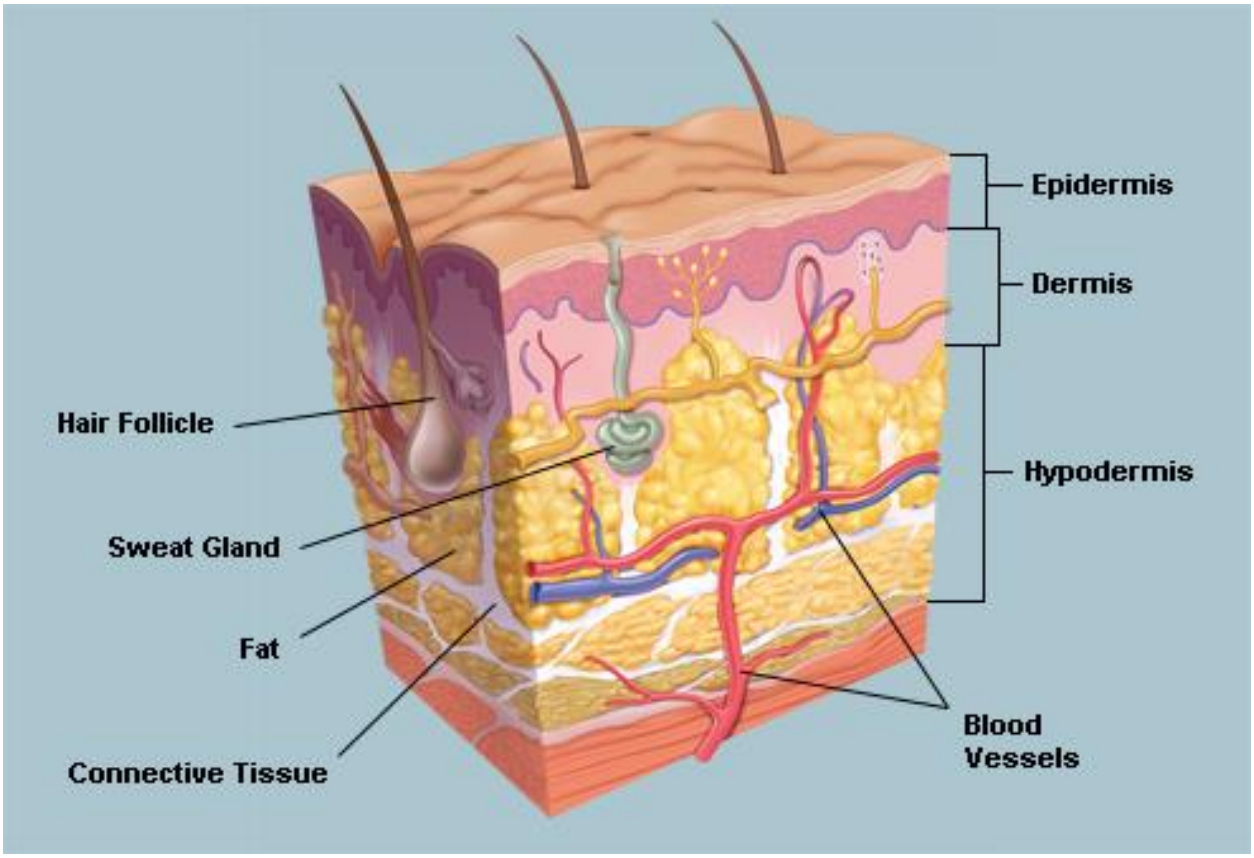
Stricture of ureter.

Malignant change due to chronic irritation of renal pelvis.

Anuria : from obstruction.

Chapter 2

Diseases of Skin



Chapter 2.1

Dermatology Introduction

Skin is the largest organ. It is an Interface between man and environment

Area 2 m^2

Weight 4 kg

Skin has three layers:

The **Epidermis**, the outermost layer of **skin**, provides a waterproof barrier and creates our **skin** tone.

The **Dermis**, beneath the epidermis, contains tough connective tissue, hair follicles, and sweat glands.

The deeper **Subcutaneous tissue** (hypodermis) is made of fat and connective tissue.

Epidermis

Non vascular stratified epithelium.

Thickness : 0.7-0.12 mm

Consists of **Keratinocytes** and **Melanocytes** with small proportion of dendritic cells.

Most important dendritic cells includes **Langerhans** cells and **Merkel cells**.

Layers of Epidermis

Stratum Basalis or germinativum.

Stratum Spinosum or malphigii or prickle cell layer.

Stratum Granulosum.

Stratum Lucidum.

Stratum Corneum.

Stratum Basalis or germinativum:Deepest layer.

Composed of columnar cells.

Whole epidermis germinates from this layer.

StatumMalphigii or Prickle cell layer : Composed of polyhedral cells connected to each other by intercellular bridges called desmosomes.

Plays important role in anchoring.

Stratum Granulosum :

Composed of fusiform cells.

These contains keratohyline and lysosomal enzymes.

Orland bodies are found which form waterproof barrier.

Langerhans cells are found. Play an imp role in graft rejection.

Stratum lucidum : In palmo planter skin, an additional layer.

Transition layer between granulosum and corneum.

Layer contains refratctileeleidin which is precursor of keratin.

Stratum corneum : Most superficial layer

Consists of many layers of non nucleated, flattened, cornified cells.

Thickest on palms and soles, thinnest at lips, glans penis and eyes.

Layers of Dermis

Twolayers :

The superficial area adjacent to the epidermis called the **papillary dermis.**

Deep thicker area known as the **reticular dermis.**

Functions of Skin

Protection against: Chemicals, particles, desiccation,Ultraviolet radiation, antigens, microbes.

Maintenance of fluid balance : Prevents loss of water, electrolytes and macromolecules.

Shock absorber : Strong, elastic and compliant covering.

Sensation :nerve endings mediating pain leading to withdrawal, and itch leading to scratch.

Vitamin D synthesis

Temperature regulation

Protection, and fine manipulation of small objects

Hormonal : Testosterone synthesis

Pheromonal : Apocrine sweat glands

Psychosocial, grooming and sexual behaviour : Hair, nails, appearance and tactile quality of skin

Dermatology is a visual speciality

Accurate diagnosis requires :

Adequate history

Careful examination

Lab investigation

History

Information regarding skin condition :

Site

Onset

Mode of spread and duration of the disorder

Personal history

Family history of skin disease

Atopy

Previous medical condition and treatment recieved.

OTC drugs taken

Social history

Occupational history

Recent travel

Sexual activity

Examination

The part should be fully exposed

Colour and Pigmentation :

Note the color of skin

Pallor :

Temporary : Shock or hemorrhage

Persistent : anemia or peripheral vasoconstriction.

Redness (erythema) :

Overexertion

Overheating

Sunburn

Febrile

Inflammatory skin disease.

Cyanosis : Blue or purple tinge

Jaundice :

Subicteric lemon yellow in pernicious anemia to various shades of yellow, orange or dark olive green in obstructive jaundice.

Increased pigmentation may be raical or connected with various diseases.

Other conditions of pigmentation :

Pregnancy – nipples and areola

Chloasma

Localized pigmentation in pellagra

Scars due to X ray irradiation

Venous hypertension in legs associated with chronic purpura – hemosiderin pigmentation

Erythema ebigne : Ladies who sit near fire

Lichen planus : Slightly raised, flat topped papules having a violacious hue.

Psoriasis lesions are symmetrical.

Keloid – raised slightly pigmented.

Skin Lesion & Eruptions

Examination in reference to :

Morphology

Distribution

Arrangement

Importance is given to

Color

Size

Consistency

Configuration

Margination

Surface characteristics

DESCRIPTION OF PRIMARY SKIN LESIONS

Macule: A flat, colored lesion, <2 cm in diameter, not raised above the surface of the surrounding skin.

Patch: A large (>2 cm) flat lesion with a color different from the surrounding skin. This differs from a macule only in size.

Papule: A small, solid lesion, <0.5 cm in diameter, raised above the surface of the surrounding skin and hence palpable (e.g., a closed comedone, or whitehead, in acne).

Nodule: A larger (0.5–5.0 cm), firm lesion raised above the surface of the surrounding skin. This differs from a papule only in size (e.g., a dermal nevomelanocytic nevus).

Tumor: A solid, raised growth >5 cm in diameter.

Plaque: A large (>1 cm), flat-topped, raised lesion; edges may either be distinct (e.g., in psoriasis) or gradually blend with surrounding skin (e.g., in eczematous dermatitis).

Vesicle: A small, fluid-filled lesion, <0.5 cm in diameter, raised above the plane of surrounding skin. Fluid is often visible, and the lesions are translucent e.g., vesicles in allergic contact dermatitis.

Pustule: A vesicle filled with leukocytes.

Bulla: A fluid-filled, raised, often translucent lesion >0.5 cm in diameter.

Wheal: A raised, erythematous, edematous papule or plaque, usually representing short-lived vasodilatation and vasopermeability.

Telangiectasia: A dilated, superficial blood vessel.

Petechiae : pinhead size small macules of blood.

Purpura : Large petechiae which do not blanch on pressure.

Ecchymosis : Large extravasation of blood in skin (bruise).

Hematoma : Swelling due to gross bleeding.

Poikiloderma : Atrophy, reticulate hyperpigmentation and telengiectasia.

Erythema : Redness of skin

Burrow : Linear or curved elevations of the superficial skin due to infestation by female scabies mite

Comedo: Dark horny keratin and sebaceous plugs within pilosebaceous openings.

Description Of Secondary Skin Lesions

Lichenification: A distinctive thickening of the skin that is characterized by accentuated skin-fold markings.

Scale: Excessive accumulation of stratum corneum.

Crust: Dried exudate of body fluids that may be either yellow (i.e., serous crust) or red (i.e., hemorrhagic crust).

Erosion: Loss of epidermis without an associated loss of dermis.

Ulcer: Loss of epidermis and at least a portion of the underlying dermis.

Excoriation: Linear, angular erosions that may be covered by crust and are caused by scratching.

Atrophy: An acquired loss of substance. In the skin, this may appear as a depression with intact epidermis.

Scar: A change in the skin secondary to trauma or inflammation. Sites may be erythematous, hypopigmented, or hyperpigmented depending on their age or character. Sites on hair-bearing areas may be characterized by destruction of hair follicles.

Stria : Atrophic pink or white linear lesion due to changes in connective tissue

Distribution of Lesion on Skin

Symmetrical or Asymmetrical

Centripetal or centrifugal

Flexor or extensor surface

Sunlight or any other external causative factor.

Genitalia involvement

Configuration of Skin Lesions

Nummular/ discoid : Round or coin like

Annular : Ring like

Circinate : Circular

Arcuate : Curved

Gyrate/ Serpiginous : Wave like

Linear : In a line

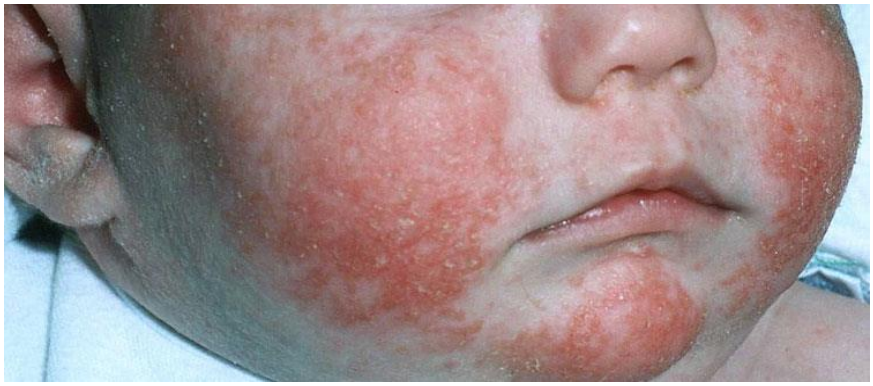
Grouped : Clustered

Reticulate : Net like

Chapter 2.2

Dermatitis

Dermatitis is a general term that describes an inflammation of the skin. Dermatitis can have many causes and occurs in many forms. It usually involves an itchy rash on swollen, reddened skin.



Skin affected by dermatitis may blister, ooze, develop a crust or flake off.

Eczema is a type of dermatitis, and these terms are often used synonymously.

Eczema is a polymorphic inflammatory reaction pattern of the skin involving the epidermis and dermis.

Term eczema means **Boil over**.

Eczema is a reaction pattern that presents with variable clinical findings and thecommon histologic finding of **spongiosis**(intercellular edema of the epidermis).

Etiology : Unknown

Classification :

Endogenous(Due to internal or constitutional factors)

Exogenous(Due to external agents)

Unclassified

Endogenous :

Atopic dermatitis

Seborrheic dermatitis

Nummular eczema

Stasis eczema

Pompholyx

Exogenous :

Allergic contact dermatitis

Irritant contact dermatitis

Photodermatitis

Unclassified :

Asteatoic eczema

Lichen simplex chronicus

Presenting Feature

Acute eczema :

Pruritis

Erythema

Edema

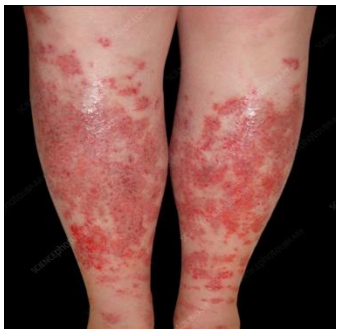
Vasiculation

Oozing

Crusting

Scaling

Acute eczema show intercellular and intracellular edema with resultant vesicle formation, dermal vasodilatation and chronic inflammatory infiltration.



Chronic eczema :

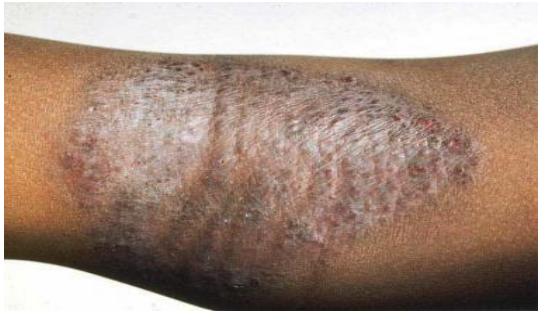
Prutitis

Lichenification

Excoriation

Hypo or hyperpigmentation

Chronic eczema is associated with thickening of epidermis and retention of nuclei by some corneocytes. Dermal vessels are dilated with inlammatory mononuclear cell infiltration.



Dermatitis Lesions

Primary lesions includes :

Erythematous macules

Papules

Vesicles which can coalesce to form patches and plaques.

Secondary lesionsmarked by

Weeping

Crusting

In chronic eczematous conditions, *lichenification*(cutaneous hypertrophy and accentuation of normal skin markings) may alter the characteristic appearance.

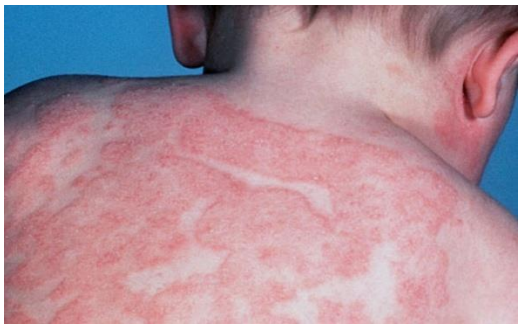
Endogenous Eczema

Endogenous Eczema is classified in following subtypes

Atopic Dermatitis

Atopy is a predisposition toward developing certain allergic hypersensitivity reactions.

AD is the cutaneous expression of the atopic state, characterized by a family history of asthma, allergic rhinitis, or eczema.



Clinical Features

Pruritus and scratching

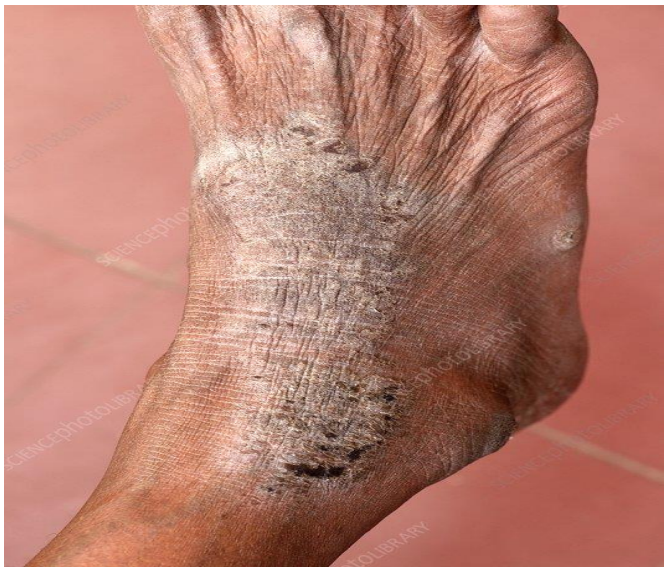
Course marked by exacerbations and remissions

Lesions typical of eczematous dermatitis

Personal or family history of atopy (asthma, allergic rhinitis, food allergies, or eczema)

Clinical course lasting >6 weeks

Lichenification of skin



Etiology :etiology of AD is only partially defined, but there is a clear genetic predisposition.

A mutation in the gene encoding filaggrin, a structural protein in the stratum corneum.

When both parents are affected by AD, >80% of their children manifest the disease. When only one parent is affected, the prevalence drops to slightly over 50%.

Characteristic defect in AD that contributes to the pathophysiology is an impaired epidermal barrier.

Exacerbating factors :

Infection with Staphylococcus aureus.

Seasonal variation : <winters, >summers

Clothing : Wool and fur <

Emotional stress

It display a variety of immunoregulatoryabnormalities :

Increased serum IgE levels.

Impaired, delayed-type hypersensitivity reactions.

The clinical presentation often varies with age :

Half of patients present within the first year of life.

80% present by 5 years of age.

About 80% ultimately coexpress allergic rhinitis or asthma

Infantile pattern is characterized by :

Weeping inflammatory patches

Crusted plaques on the face, neck, and extensor surfaces.

The childhood and adolescent pattern is typified by dermatitis of flexural skin :

Antecubital fossae.

Popliteal fossae.

Clinical Features

Pruritis is the main feature

Itch – scratch – rash – itch.

Infantile phase : Lesions occurs on cheeks, forehead and scalp later on trunk and extremities

Childhood phase : Papules, lichenified plaques, erosions and crusts mainly on ante cubital and popliteal fossa, neck, wrist and ankles.

Adult phase :Lichenification and excoriation in flexor distribution.

Diagnosis

Based on clinical findings :

Infancy onset

Severe pruritis

Typical distribution

Morphology of lesions

Personal or family history of atopy.

Differential Diagnosis

Seborrheic dermatitis

Irritant and allergic contact dermatits

Psoriasis

Nummular eczema

Dermatophytosis

It may resolve spontaneously, but approximately 40% of all individuals affected as children will have dermatitis in adult life.

Adults have localized disease manifesting as lichen simplex chronicus or hand eczema.

Treatment of Atopic Dermatitis

Avoidance of cutaneous irritants,

Adequate moisturizing

Topical anti-inflammatory agents

Topical glucocorticoids

Antihistamines

Seborrheic Dermatitis

Chronic inflammatory dermatitis characterized by erythema and scaling in regions where sebaceous glands are most active.

In scalp it causes flaking (dandruff).

More common in males.

Incidence increases with age.

HIV infected individuals have an increased incidence of disease.

Etiology :

Unknown.

Malassezia furfur plays an important role.

Abnormalities in sebaceous gland activity.

Zinc, niacin, pyridoxine deficiency.

Clinical Features :

Scalp and face involvement : greasy or dry scaling, erythematous macules and papules.

It affects the eyebrows, eyelids, glabella, and nasolabial folds

Petaloid lesions over the presternal area.

Flexural : In axilla, groins, anogenital and submammary area.

Pitrosporumfolliculitis with papules or pustules over the back.

Course and prognosis :

Affects majority of individuals .

Recurrances and remissions are common.

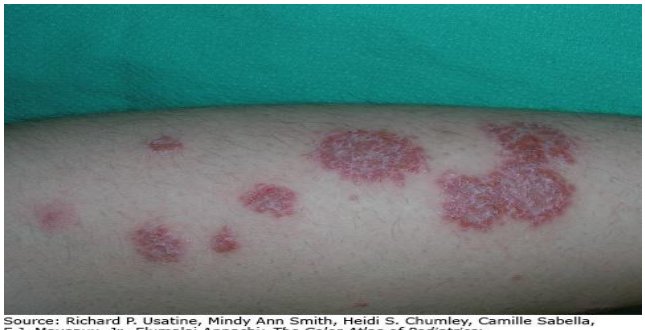
Infantile and seborrheic dermatitis disappears with age.

Management :

Shampoo containing selenium sulphide, zinc pyrithione, ketoconazole, flucinoloneacetonide twice a week.

Topical glucocorticoids creams

Nummular Eczema/ Discoid Eczema



Chronic pruritic inflammatory dermatitis characterized by coin shaped erythematous plaques with exudation and crusting.

Severely pruritic with excoriation.

Lesions clustered on lower parts of legs and trunk in males and hands and fingers in females.

It may be generalized and scattered.

Course from weeks to months.

Secondary infections are common.

Management :

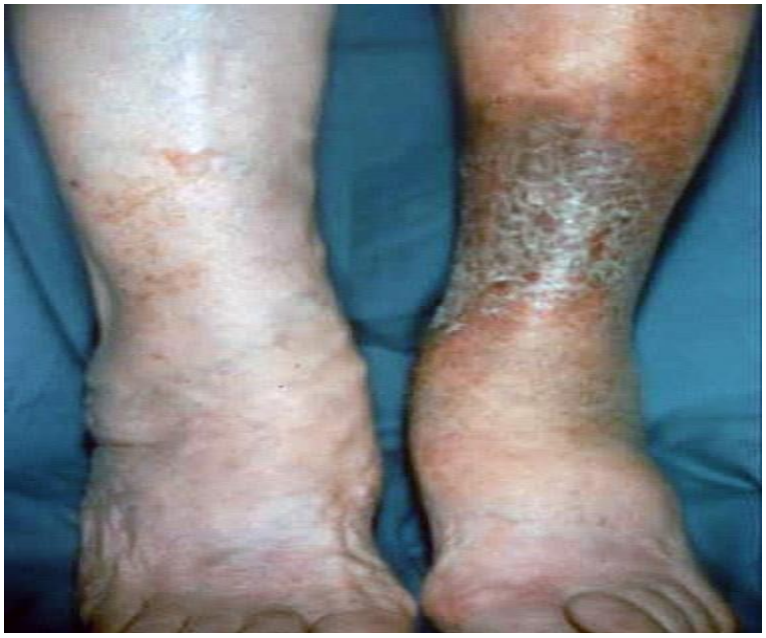
Topical steroids with antibiotics.

Moisturizers and antihistamines.

Syestemic antibiotics

Photochemotherapy (PUVA)

Stasis /Gravitational Eczema



Chronic venous insufficiency leads to stasis eczema (Varicose eczema) in lower parts of legs and feet.

Pathogenesis

Incompetence of deep perforating veins increases the hydrostatic pressure in dermal capillaries.

Pericapillary fibrin deposition leads to pathological changes.

Clinical Features :

Inflammatory edema

Papules

Scaling

Crusting

Erosion

Pigmentation stippled with hemorrhage

Dermal sclerosis

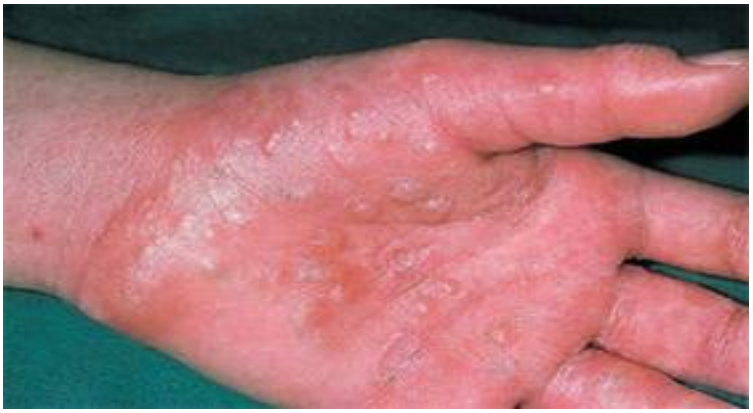
Management

Emollients

Topical steroids

Pompholyx

Dyshidrotic Eczema



A very common, chronic skin disorder in which both exogenous and endogenous factors play important roles.

Occupation-associated skin disease.

Chronic, excessive exposure to water and detergents, harsh chemicals, or allergens.

Present with dryness and cracking of the skin of the hands as well as with variable amounts of erythema and edema.

Acute , chronic or recurrent dermatosis of lateral aspects of fingers, palms and soles.

Characterized by symmetrical deep seated pruritic, clear vesicles and later by scaling, fissures and lichenification.

Spontaneous remission occurs in 2-3 weeks.

Recurrence is the rule.

Secondary infection may occur.

Hyperhidrosis is common.

Management :

Wet compresses with saline

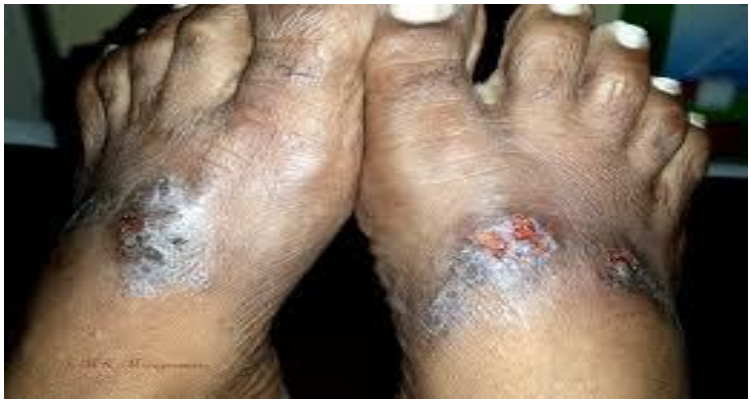
Topical corticosteroids

Topical PUVA therapy.

Exogenous Eczema

It includes the following:

Contact Dermatitis



Acute chronic inflamatory or chronic inflammatory reaction to substances that come in contact with the skin.

The clinical lesions of contact dermatitis may be acute (wet and edematous) or chronic (dry, thickened,and scaly)

Two types :

Irritant contact dermatitis (ICD) caused by chemical irritants.

Allergic contact dermatitis (ACD) caused by allergens showing type IV hypersensitivity reactions.

Irritant Contact Dermatitis

Exposure if skin to chemicals or physical agents.

May occur within minutes after exposure or may be delayed upto 24 hours.

Changes vary from erythema to vesiculation..

Erosion, crusting and scaling follows.

Papules are not seen.

Lesions are sharply demarcated to the site of contact.

Clinical Features

Hands are commonly affected.

Dryness

Chapping

Erythema

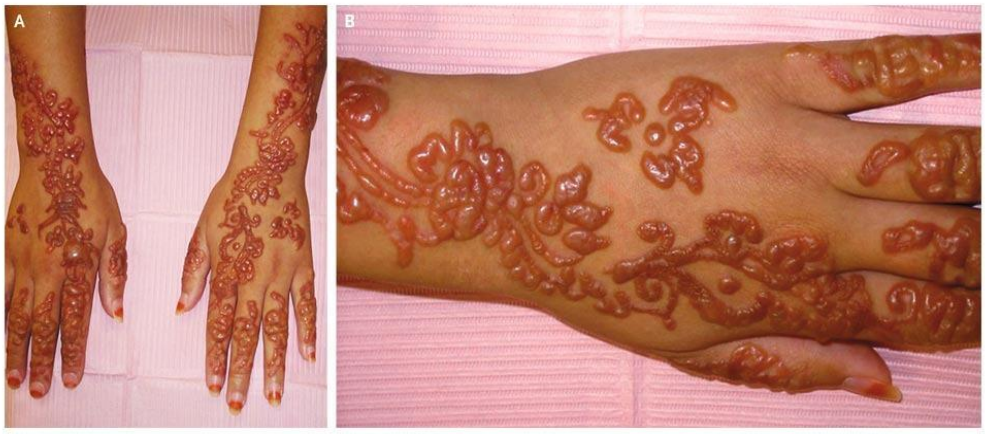
Scaling

Fissuring

Crusting

Stinging and itching

Allergic Contact dermatitis



Dependent on the sensitization and occurs only in sensitized individuals.

Develops within 48 hours of exposure.

Worsens on repeated exposure.

Clinical Features

Intense pruritis.

Lesions are confined to the area of contact later spread to surrounding areas.

In acute stage there is :

Erythema

Papules

Vesicles

Erosions

Scaling

Crusting

In chronic case :

Lichenification

Fissuring

Scaling

Crusting

Patch test

Application of allergen to any area of normal skin provokes eczematous reactions.

Management

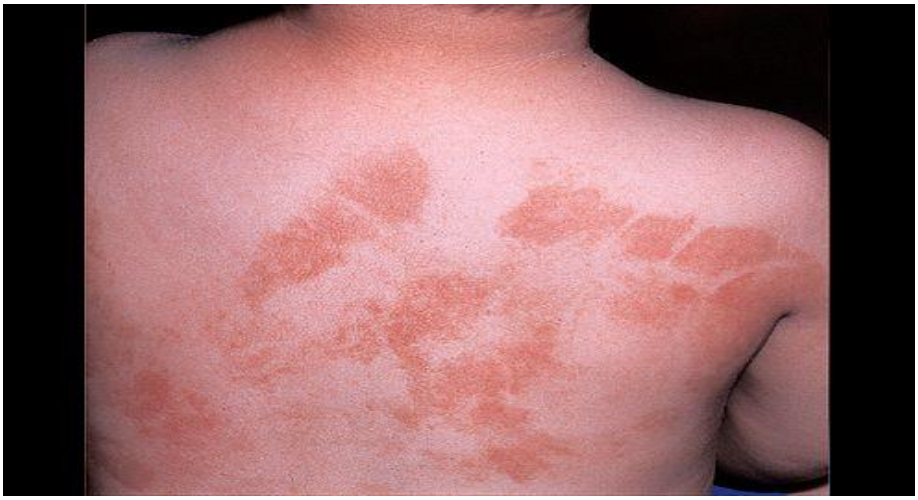
Avoidance of allergens

Wet dressing and topical steroids

Systemic glucocorticoides

In chronic cases lubricating creams and high potency topical steroids.

Photosensitive Dermatitis



Normal pigmentation increases with exposure to sunlight.

Abnormal response to sunlight and effects the sunexposed parts of body like forehead, malar region, nose, sides and back of neck, V area of chest and extensor aspect of distal extremities.

UVA, UVB are primary inducers.

Management

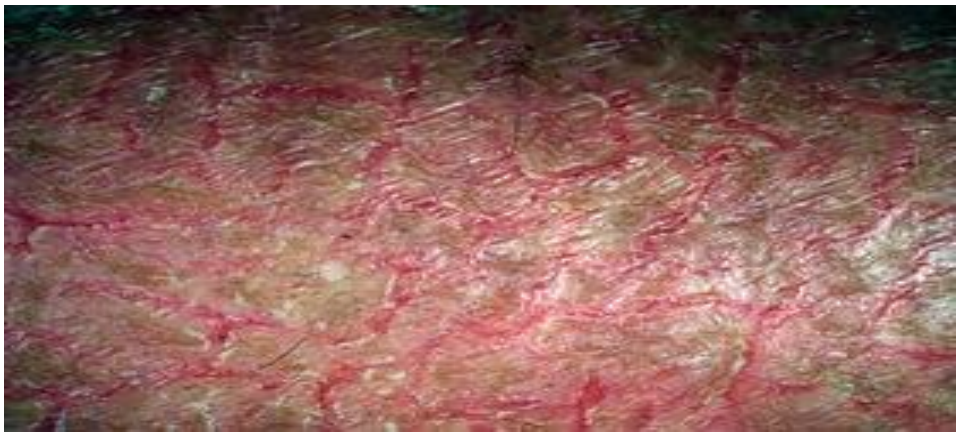
Avoidance of sunlight

Sunblock cream like zinc oxide

Topical glucocorticoids

Antihistamines

Asteatoic Eczema (Eczema Craquele)



Also known as *xerotic eczema* or *"winter itch",* Dry eczema with fissuring and cracking of skin.

Affecting limbs in elderly.

Clinical features

Skin is :

Dry

Erythematous

Itchy

Crazy paving pattern of fissuring.

Worse :

Dry winter climate

Overwashing

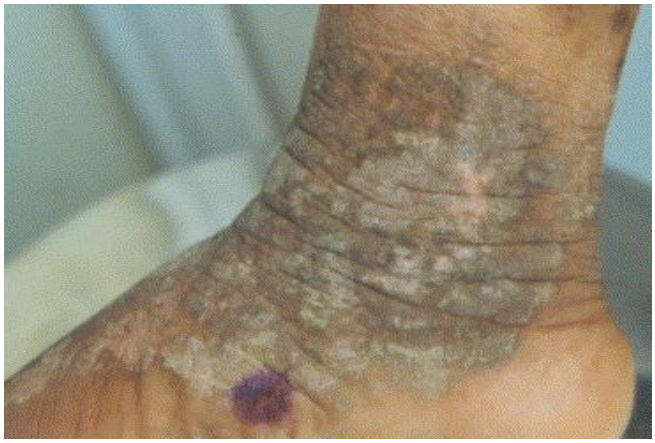
Hypothyroidism

Management :

Topical emollients

Mild topical steroids

Lichen Simplex Chronicus (LSC)



It represent the end stage of a variety of pruritic and eczematous disorders, including Atopic Dermatitis.

It consists of a circumscribed plaque or plaques of lichenified skin due to chronic scratching or rubbing.

Common areas involved include the posterior nuchal region, dorsum of the feet, and ankles.

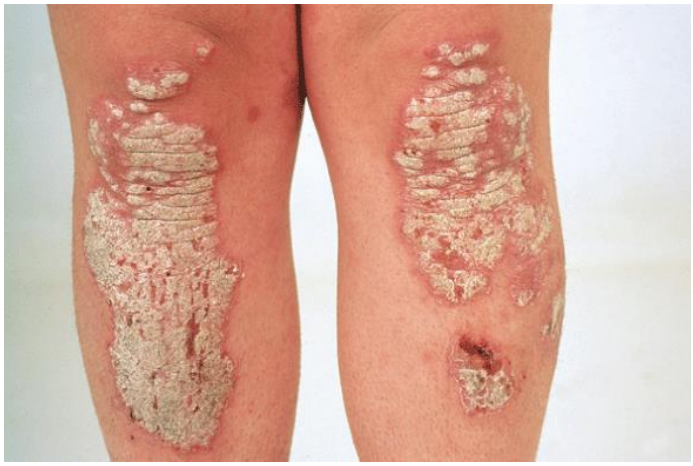
Management

Breaking the cycle of chronic itching.

High-potency topical glucocorticoids

Chapter 2.3

Psoriasis



Psoriasis is a non-infectious, chronic inflammatory disease of the skin, characterised by well-defined erythematous plaques with silvery scale.

Predilection for the extensor surfaces and scalp.

It has chronic fluctuating course.

Epidemiological patterns :

Type 1 : Early onset. HLA Cw6

Type 2 : Late fifties or sixties

Aetiology *:*is complex and large number of genes are involved.

Two key pathophysiological features :

Hyperproliferation of keratinocytes, retention of nuclei in stratum corneum.

Inflammatory cell infiltration.

Precipitating Factors

Trauma

Infection :β-haemolytic streptococcal throat infections.

Sunlight

Drugs

Emotion

Types of Psoriasis

Stable Plaque Psoriasis

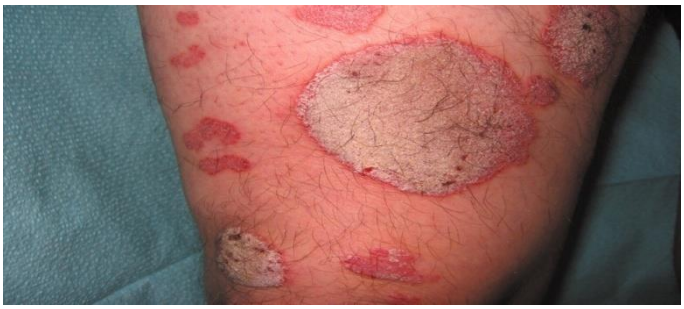
Guttate Psoriasis

Erythrodermic Psoriasis

Pustular Psoriasis

Compications:Arthropathy

Stable plaque psoriasis



Most common type.

Well demarcated lesions ranging from a few millimetres to several centimetres in diameter.

Lesions are red, with a dry silvery-white scale, obvious after scraping the surface.

The elbows, knees and lower back are commonly involved.

Other sites :

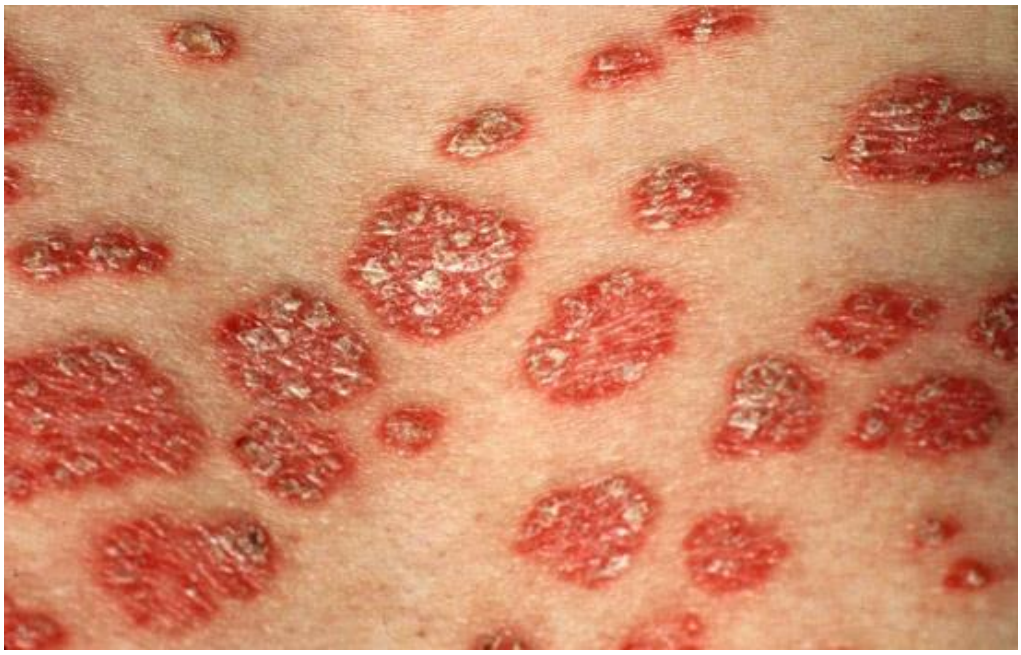
Scalp :involved in 60% of cases

Nails :thimble pitting, onycholysis.

Flexures :submammary and axillary folds is not scaly but red, shiny and symmetrical.

Palms :poorly demarcated and barely erythematous.

Guttate Psoriasis



Most common in children and adolescents.

β-haemolytic streptococcal throat infections preceedsGuttate psoriasis.

Seen particularly on the trunk and proximal parts of limbs.

Rash often appears rapidly.

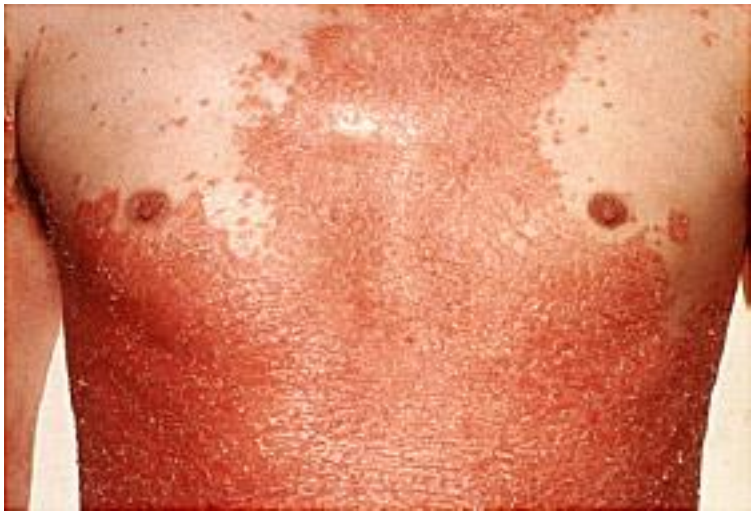
Individual lesions are droplet shaped, seldom over 1 cm in diameter and scaly.

Bouts of guttate psoriasis may clear within a few months.

Respond well to early treatment with phototherapy.

Majority of patients develop plaque psoriasis later in life.

Erythrodermicpsoriasis :



It occurs in two forms.

The skin becomes universally red or scaly, or more rarely just red with very little scale.

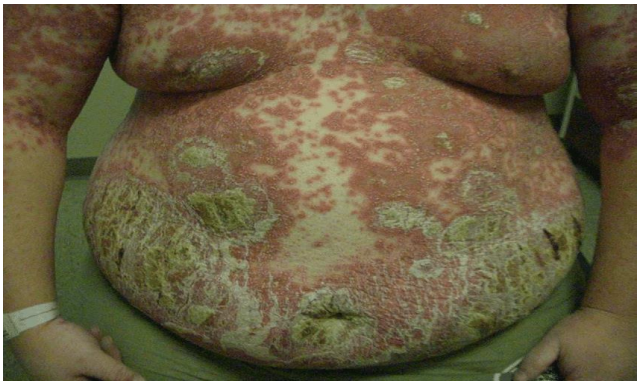
Temperature regulation becomes compromised with a danger of either hypothermia or hyperthermia.

Chronic lesions evolve gradually into exfoliative phase – extensive plaque psoriasis involving all the cutaneous surface.

The psoriatic characteristics retained, mild treatment is well tolerated.

Prognosis is good

Pustular Psoriasis



Two varieties of pustular psoriasis

Generalised

The generalised form is rare but serious.

Onset is usually sudden, with large numbers of small sterile pustules erupting on a red base.

Pyrexia coinciding with the appearance of new pustules.

Require urgent assessment and hospital admission.

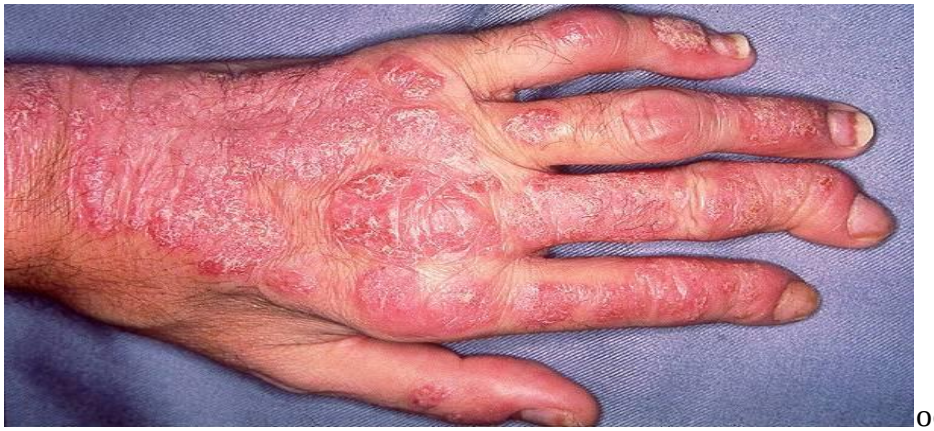
Localised : Primarily affects the palms and sole.

Small sterile pustules which lie on a red base, and resolve to leave brown macules or scaling.

Arthropathy :5 to 10% of individuals with psoriasis develop a chronic seronegative inflammatory arthropathy (Rheumatoid factor negative inflammatory arthropathy).

It takes a number of patterns (Central- as in ankylosingspondylosis, peripheral -as in rheumatoid arthritis)

Psoriatic arthritis (PsA)

 occurs in about 1 in 1000 of the general population and in 7% of patients with psoriasis.

Age : Between25 and 40 years

Clinical features

Joint pain and swelling

Asymmetrical inflammatory oligoarthritis: affects in 40% cases. It has a combination of synovitis and adjacent periarticular inflammation. Hands and feet.

Symmetrical polyarthritis :occurs in about 25% cases. Symmetrical involvement of small and large joints in both upper and lower limbs.

Distal IPJ arthritis :mainly affecting men. It targets finger distal IPJs and surrounding periarticular tissues accompanying nail dystrophy.

Management

NSAID and analgesics

Intra-articular injections of corticosteroid

Methotrexate

Investigations

Biopsy is seldom necessary.

Throat swabbing for streptococci.

Joint symptoms require a formal rheumatology assessment.

General management

Explanation, reassurance and instruction.

Treatment :

*Topical agents :*Corticosteroids

Ultraviolet and PUVA therapy

Ultraviolet B (UVB) administered therapeutically improves psoriasis

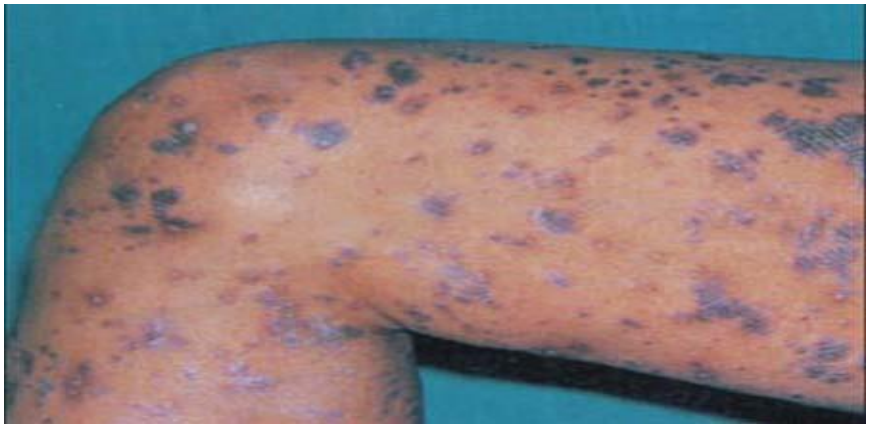
Psoralens can be applied in a bath before irradiation with UVA

*Systemic treatment :*methotrexate and hydroxyurea

*Biological therapies :*monoclonal antibodies, fusion proteins and cytokines.

Chapter 2.4

Lichen Planus



A chronic inflammatory and immune mediated disease that affects the skin, scalp, hair, nails and mucous membranes.

It is a type of Papulo squamous disorder.

A papulosquamous disorder is a condition which presents with both papules and scales, or both scaly papules and plaques. Examples include psoriasis, lichen planus, and pityriasisrosea and Dermatophytosis.

Aetiology

The cause is unknown but an immune pathogenesis is suspected.

Pathology

Pathology includes :

Hyperkeratosis

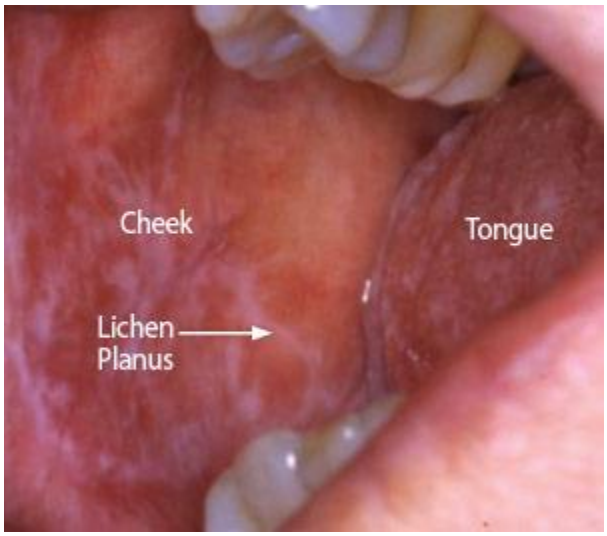
Basal cell degeneration

T-lymphocyte infiltration in the upper dermis

The Primary cutaneous lesions are pruritic, polygonal, flat topped, violaceous papules.

Close examination of the surface of papules often reveals a network of grey lines (Wickham's striae).

Wickham striae are whitish lines visible in the papules of lichen planus, typically in the oral mucosa. The macroscopic appearance shows hypergranulosis. It is named after Louis Frédéric**Wickham**.

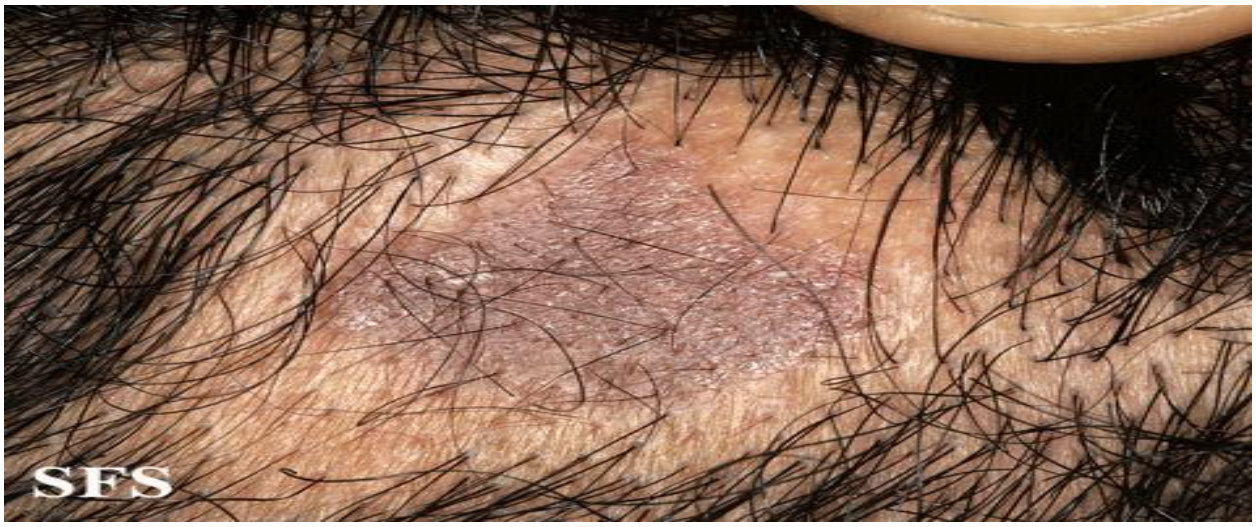


Lesions have a predilection for wrist, shin, lower back and genitalia.

Nail involvement leads to permanent deformity or loss of fingernails or toenails.

Involvement of scalp lichen planopilaris may lead to scarring alopecia.

The peripheral perifollicular macules are usually violet colored.



LP commonly involves mucous membrane particularly buccal mucosa.

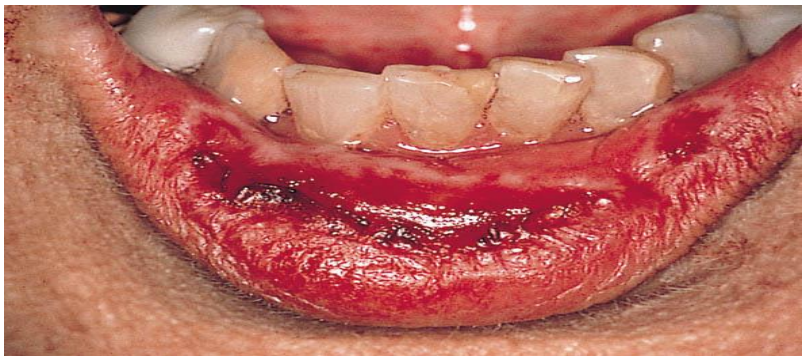
Ususal location is buccal mucosa, tongue, lips and skin.

Clinical features includes :Striae, white plaques, red areas, ulcers in mouth, Purplish papules on skin.

These may be asymptomatic or can be sore or painful.

The symptoms can be mild, including white reticular eruption to severe, erosive stomatitis.

Erosive stomatitis persists for years. Has increased risk of oral squamous cell carcinoma.



Clinical Features

Start on the distal limbs, most commonly on the volar aspects of the wrists and on the lower back.

Intensely itchy, flat-topped, pinkpurplish papules.

Some have characteristic fine white network on their surface (Wickham's striae).

New lesions may appear at the site of trauma (Köbner phenomenon) and the rash may spread rapidly to become generalised.

Mucous membrane involvement, comprising an asymptomatic fine white lacy network of pinhead-sized white papules, occurs in about two-thirds of patients.

Nails affected in 10% cases with changes from longitudinal grooving to destruction of the nail fold and bed.

Diagnosis

Diagnosis by clinical appearance

Skin biopsy can be helpful.

Differential Diagnosis

Guttate psoriasis

Pityriasisrosea

Pityriasislichenoides

Drug eruptions

Lichenoid Eruptions

LP may be associated with Hepatitis C infection

LP have been observed after administration of numerous drugs including thiazide diuretics, gold, antimalarials, penicillamine, and phenothiazines.

Patients with skin lesions of chronic graft verses host disease.

People handling colour developers.

Variants of the classic picture are rare but often diagnostically challenging.

Variants include :

Annular,

Atrophic,

Bullous,

Follicular,

Hypertrophic

Ulcerative types.

Management

Usually self-limiting with spontaneous remission in 6 months to 2 years.

Topcal Glucocorticoids are mainstay therapy.

Nonsteroidal creams or ointments.

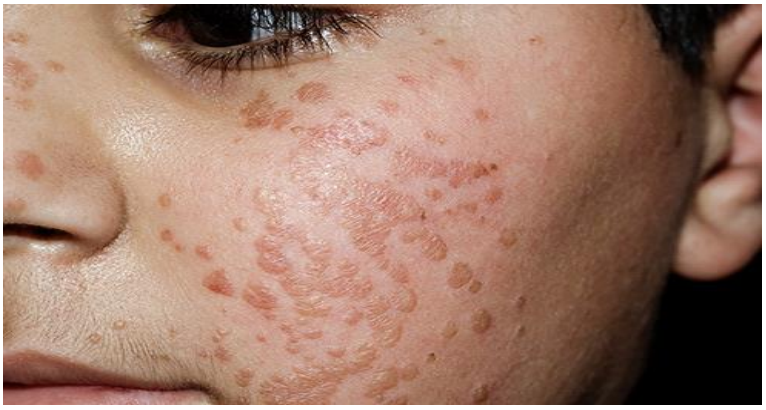
Antihistamines

Retinoids

Phototherapy may be required.

Chapter 2.5

Verrucae (Warts)



Cutaneous neoplasms caused by Human papillomaviruses (HPVs).

A typical wart, *verruca vulgaris,* is sessile, dome-shaped, and usually about a centimeter in diameter.

The surface is hyperkeratotic, consisting of many small filamentous projections.

Other varients :

Plantar warts

Flat warts (*verruca Plana)*

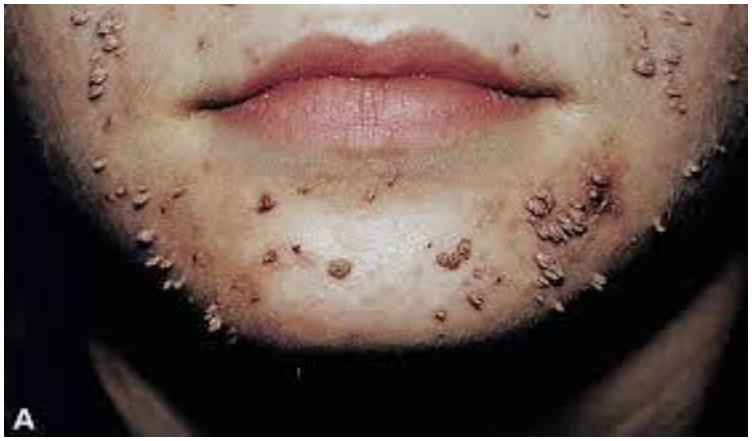
Filiform warts.

Genital warts

Plantar warts are endophytic and are covered by thick keratin.

Paring of the wart will generally reveal a central core of keratinized debris and punctate bleeding points.

Filiform warts are most commonly seen on the face, neck, and skinfolds and present as papillomatous lesions on a narrow base.



Flat warts are only slightly elevated and have a velvety, non verrucous surface.

Seen commonly on face, arms, and legs and are often spread by shaving.

Genital warts begin as small papillomas that may grow to form large, fungating lesions.

In women, involve the labia, perineum, or perianal skin. The mucosa of the vagina, urethra, and anus can be involved.

In men, the lesions occur in the coronal sulcus but may be seen on the shaft of the penis, the scrotum, or the perianal skin or in the urethra.

Transmission : is by direct contact with the virus, in either living skin or fragments of shed skin, and is encouraged by trauma and moisture (e.g. in swimming pools, fishmongers etc.).

Genital warts are spread by sexual activity, and show a clear relationship with cervical and intra-epithelial cancers of the genital area.

Management

Majority of warts resolve spontaneously within 1–2 years.

Many modalities available to treat warts, but no single therapy is universally effective.

Choice of therapy include :

Location of the wart,

The extent of disease,

The age

Immunologic status of the patient

The patient's desire for therapy.

Cryotherapy with liquid nitrogen.

Keratolytic agents such as salicylic acid plasters or solutions.

Genital warts, in-office application of a podophyllin solution is moderately effective

Topical imiquimod has been approved for treatment of genital warts.

Laser surgical procedures are also effective.

A highly effective vaccine for selected types of HPV has been approved by the FDA, and its use appears to reduce the incidence of anogenital and cervical carcinoma.

Chapter 2.6

Acne

Acne is a disorder of the sebaceous gland.

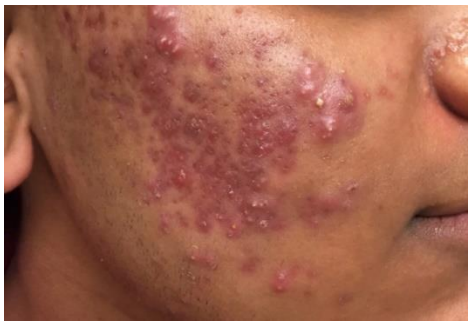
The hair follicles become plugged with oil and dead skin cells.

It often causes whiteheads, blackheads or pimples.

It appears on the face, forehead, chest, upper back and shoulders.

It is most common among teenagers, though it affects people of all ages

ACNE VULGARIS



Acne vulgaris is a chronic inflammatory disease of the pilosebaceous follicles.

It is characterized by formation of comedones, erythematous papules and pustules less frequently nodules and pseudocyst and accompanied by scarring.

It is a self-limited disorder primarily of teenagers and young adults.

10–20% of adults may continue to experience some form of the disorder.

Most common in teenage years but may persist into the third decade and beyond.

Aetiology

Increased sebum excretion is necessary for the development of acne.

Androgens and progestogens increases sebum excretion, whilst oestrogens reduce it.

Propionibacterium acnescolonises the pilosebaceous ducts and acts on lipids to produce pro-inflammatory factors, and the pilosebaceous unit becomes occluded.

Small cysts, called *comedones*, form in hair follicles due to blockage of the follicular orifice by retention of keratinous material and sebum.

Clinical Features

Lesions are usually limited to the face, shoulders, upper chest and back, but may extend to the buttocks.

Seborrhoea (greasy skin) is often obvious.

Open comedones (blackheads) due to plugging by keratin and sebum of the pilosebaceous orifice.

Closed comedones (whiteheads) due to accretions of sebum and keratin deeper in the pilosebaceous ducts.

Combination of keratin breakdown products and bacterial products gives rise to the black colour seen in blackheads.

Comedones are usually accompanied by inflammatory lesions: papules, pustules, or nodules.

The earliest lesions seen in adolescence are generally mildly inflamed or noninflammatorycomedones on the forehead.

Subsequently, more typical inflammatory lesions develop on the cheeks, nose, and chin.

Most disease remains mild and does not lead to scarring.

A small number of patients develop large inflammatory cysts and nodules, which may drain and result in significant scarring.

Exogenous and endogenous factors can alter the expression of acne vulgaris.

Friction and trauma (from headbands or chin straps of athletic helmets), application of comedogenic topical agents (cosmetics or hair preparations).

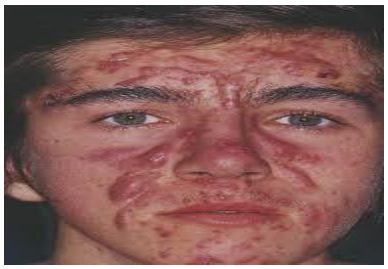
Chronic topical exposure to certain industrial compounds may elicit or aggravate acne.

Glucocorticoids, topical or systemic, may also elicit acne.

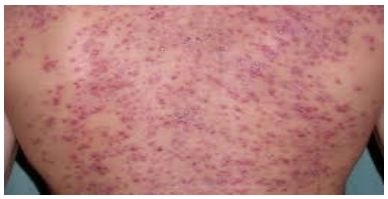
Systemic medications such as oral contraceptive pills, lithium, isoniazid, androgenic steroids, halogens, phenytoin, and phenobarbital may produce acneiform eruptions or aggravate preexisting acne.

Genetic factors and polycystic ovary disease may also play a role.

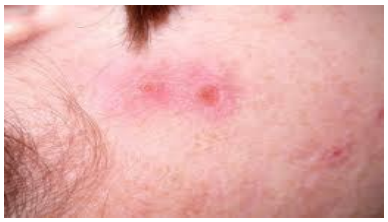
Conglobate acne is characterised by comedones, nodules, abscesses and sinus tracks, often accompanied by keloidal scarring. Epidermoid cysts are common later on in acne.



Acne fulminans - Extentisve inflammatory lesions esp on the trunk.Itis accompanied by fever, joint pains and markers of systemic inflammation such as a raised ESR.



*Acne excoriee*refers to the effects of scratching or picking, principally on the face of teenage girls with acne.



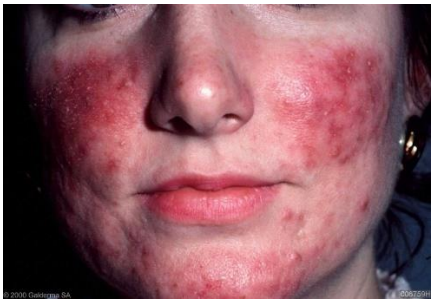
Management

Directed toward elimination of comedones by normalizing follicular keratinization, decreasing sebaceous gland activity, decreasing the population of Propionibacterium*acnes*, and decreasing inflammation.

Topical agents such as retinoic acid, benzoyl peroxide, or salicylic acid may alter the pattern of epidermal desquamation, preventing the formation of comedones and aiding in the resolution of pre-existing cysts.

Topical antibacterial agents (such as azelaic acid, erythromycin, clindamycin, or dapsone) are also useful.

Acne Rosacea



It is persistent facial eruption of unknown cause.

An inflammatory disorder predominantly affecting the central face.

Persons most often affected are Caucasians of northern European background, but also occurs in patients with dark skin.

Sebum secretion is normal.

Common in middle aged <30 years old more common in women, but those most severely affected are men.

Affects cheeks, chin and central forehead.

Characterized by the presence of erythema, telangiectases, and dome-shaped papules and pustules.

Comedones are absent.

Rarely involves the chest or back.

Rosacea initially demonstrate a pronounced flushing reaction in response to heat, emotional stimuli, alcohol, hot drinks, or spicy foods.

Flush persists longer and longer and may become permanent.

Very long standing rosacea lead to connective tissue overgrowth of the nose (*rhinophyma*).

Complicated by various inflammatory disorders of the eye, keratitis, blepharitis, iritis, and recurrent chalazion.

Management

Treatment topically or systemically.

Mild disease responds to topical metronidazole, sodium sulfacetamide, or azaleic acid.

Severe disease requires oral tetracyclines, doxycycline, or minocycline,

Residual telangiectasia may respond to laser therapy.

Topical glucocorticoids should be avoided because chronic use of these preparations elicit rosacea.

Acne from homoeopathic point of view can be caused by various causative factors:

Menstruation

Pregnancy

Food habits and allergies – highly seasoned food

Emotions- stress

Chapter 2.7

Scabies

Sarcoptes scabiei

Cause: The human itch mite, *Sarcoptesscabiei*var. *hominis*

It causes itching dermatosis, infesting ~300 million persons worldwide at any point of time.

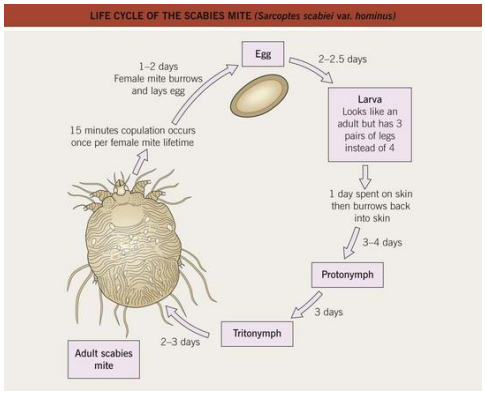
Scabies should be considered in patients with pruritus and symmetric superficial, excoriated, papulovesicular skin lesions in characteristic locations, particularly if there is a history of household contact with an infested person.

Life Cycle of Itch mite

Gravid female mites (~0.3 mm in length) burrow superficially within the stratum corneum, depositing three or fewer eggs per day.

Six-legged larvae mature to eight-legged nymphs and then to adults. Gravid adult females emerge to the surface of the skin about 8 days later and then reinvade the skin of the same or another host.

Newly fertilized female mites are transferred from person to person mainly by direct skin-to-skin contact



Transfer is favoured by:

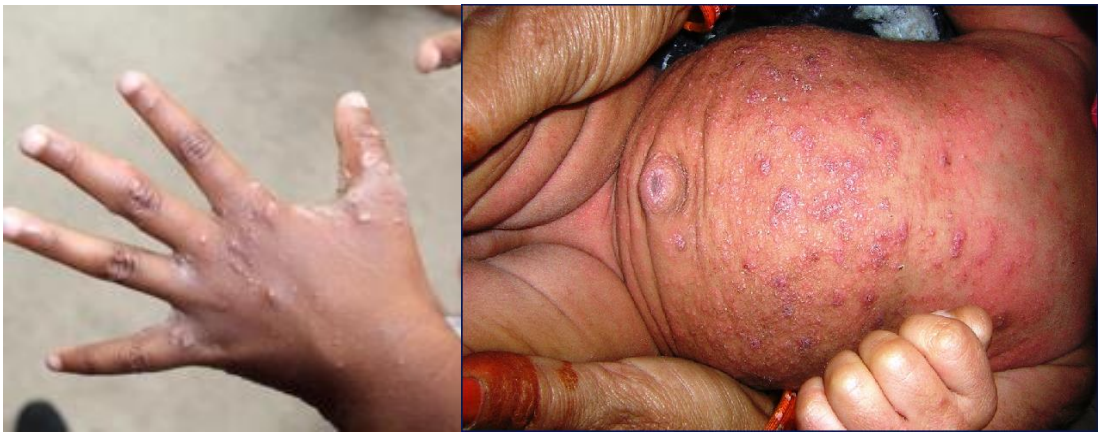
Crowding

Poor hygiene

Sex with multiple partners.

Outbreaks occur in preschools, hospitals, nursing homes, and other residential institutions.

Mites die within a day or so in the absence of host contact.



Clinical Features

Asymptomatic for up to 6 weeks before the onset of intense pruritus.

Pruritus < at night and after hot shower.

Burrows become surrounded by inflammatory infiltrates composed of eosinophils, lymphocytes, and histiocytes,

A generalized hypersensitivity rash later develops in remote sites.

Location and Appearance

Burrows appear as dark wavy lines in the upper epidermis and are 3–15 mm long.

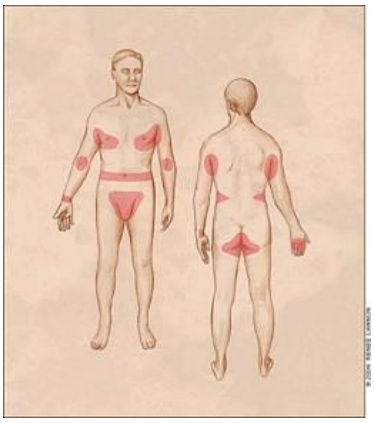
Most common on the volar wrists and along the digital web spaces.

In males, the penis and scrotum become involved.

Appear symmetrically at those sites; within intertriginous areas.

Around the navel and belt line; in the axillae; and on the buttocks and upper thighs.

Small papules and vesicles, often accompanied by eczematous plaques, pustules, or nodules, appear symmetrically at those sites

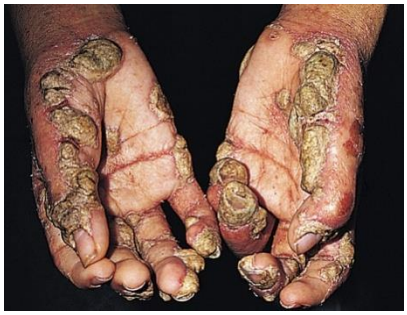


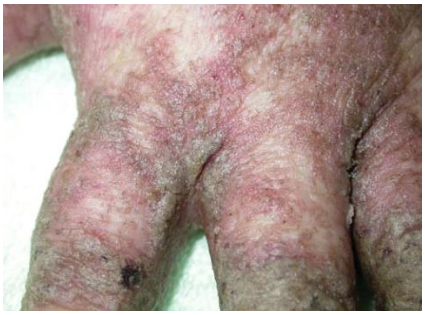
Immunity and associated scratching limit most infestations to <15 mites per person.

Hyperinfestation with thousands of mites, a condition known as *crusted scabies occurs in* result from glucocorticoid use, immunodeficiency, and neurologic or psychiatric illnesses.

Crusted scabies often resembles psoriasis, both are characterized by widespread thick keratotic crusts, scaly plaques, and dystrophic nails.

Characteristic burrows are not seen in crusted scabies, and patients usually do not itch but are highly contagious.





Diagnosis

History of pruritus

Clinical examination

Epidemiologic link

Burrows are sought and unroofed with a sterile needle or scalpel blade.

The scrapings is examined microscopically for mites, eggs, and fecal pellets.

Examination of skin biopsies or scrapings.

Dermatoscopic imaging of papulovesicular lesions.

Microscopic inspection of clear cellophane tape lifted from lesions also may be diagnostic.

Management

Permethrin cream.

Scabicides are applied thoroughly behind the ears and from the neck down after bathing.

Successful treatment of crusted scabies requires preapplication of a keratolytic agent such as 6% salicylic acid and then of scabicides to the scalp, face, and ears.

Within 1 day of effective treatment, scabies infestations become noncommunicable.

Antihistamines, salicylates, and calamine lotion relieve itching during treatment, and topical glucocorticoids are useful for pruritus that lingers after effective treatment.

Unnecessary re-treatment with topical agents may provoke contact dermatitis.

Precautions

Bedding and clothing should be washed and dried on high heat or heat-pressed.

Close contacts of confirmed cases, even if asymptomatic, should be treated simultaneously.

Scabies mites of other animals may cause transient irritation, but they do not reside or reproduce in human hosts.

In humans the causative factor is ***Sarcoptesscabiei*var. *hominis.***

Chapter 2.8

Dermatophytosis

Dermatophytes are fungi that infect skin, hair, and nails.

It include members of the genera *Trichophyton*, *Microsporum*, and *Epidermophyton.*

Typical infections consist of erythematous, scaly plaques, with an annular appearance that accounts for the common name "ringworm" with central clearing and scale along peripheral advancing border.

They can originate from :

Soil (geophilic)

Animals (zoophilic),

Confined to human skin (anthropophilic).

The lesions start as a papule and spreads in a ring like form peripherally, with central clearing.

The lesions are characteristically circular with sharply defined, active and raised edges, consisting of vesicles and scaling.

Single lesion occurs and thereby multiple plaques may coalesce into larger lesions.

The central skin of the lesions show post inflammatory pigmentation, change of texture or residual erythematous dermal nodules.

There is great itching present but individual characteristic feature depends upon the type of lesion.

It can involve any area of body; due to infection of stratum corneum, nail plate, or hair.

Common sites:

Scalp (tineacapitis).

Involvement of the body (Tineacorporis)

Groin (Tineacruris)

Palm (Tineamanuum)

Foot (Tineapedis)

Nails (Tineaunguium or onychomycosis),

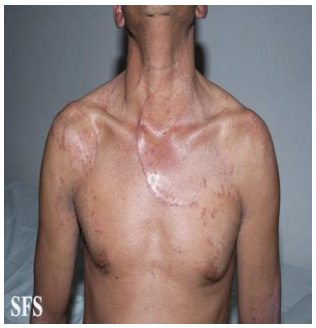
Tineacorporis*:*lesions are erythematous, annular and scaly, with a well-defined edge and often central clearing.

Can be single or multiple.

Usually asymmetrical.

Inflammation depends on the causative fungus and host immunity.

Microsporumcanis(from dogs) and *Trichophytonverrucosum*(from cats) are common cause.



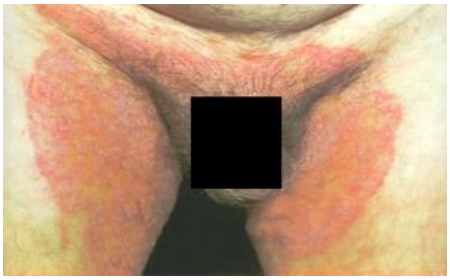
Tineacruris :

Caused by *Trichophytonrubrum.*

Most common world-wide affection, more in males than in females.

Affects the groin region.

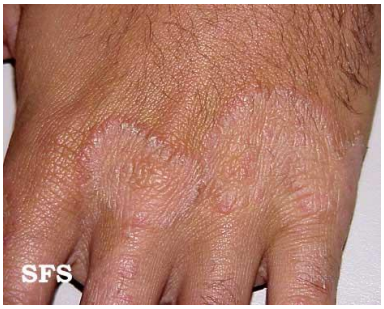
Itchy erythematous plaques extend from the groin flexures on to the thighs.



Tineamannum:

Caused by Trichophytonrubrum.

Unilateral involvement of palm which becomes dry and hyperkeratotic, with mild scaling around the palmer creases.



Tineapedis (athlete's foot) *:*

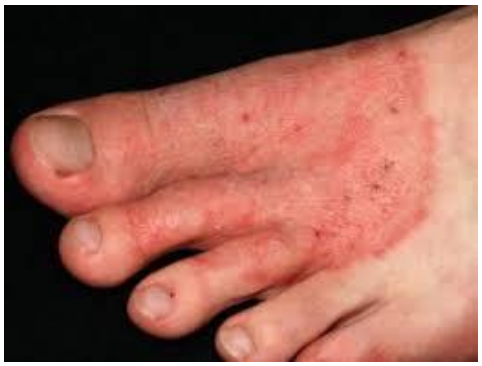
Caused by *Trichophytonrubrum, T. mentagrophytes*and *Epidermophytonfloccosum.*

Characterized by variable erythema, edema, scaling, pruritis and occasionally vesiculation.

Itchy rash between the toes, with peeling, fissuring and maceration.

Involvement of one sole or palm with a fine scaling is characteristic of *T. rubrum*infection.

Vesiculation or frank blistering is more commonly seen with *T. mentagrophytes.*



Tineaunguium or onychomycosis :

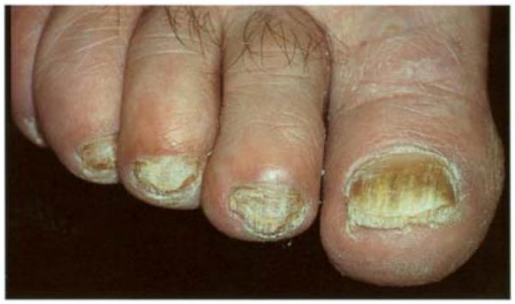
Characterized by opacified, thickened nails and subungual debris.

Yellow-brown discoloration and crumbling of the plate which starts distally and spreads proximally.

More commonly toenails.

The distal-lateral variant is most common.

Proximal subungualonychomycosis is a marker for HIV infection or other immunocompromised states.

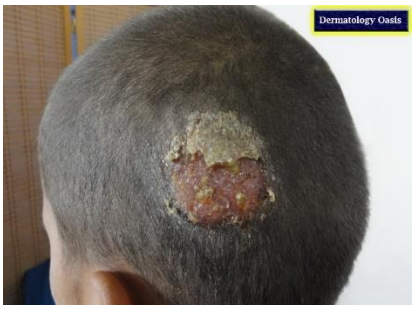


Tineacapitis :

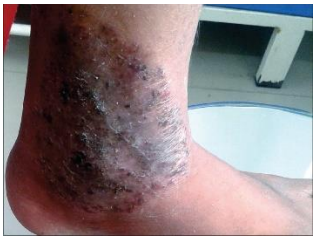
Caused by *Trichophytontonsurans.*

Noninflammatory infection with mild scale and hair loss that is diffuse or localized.

Markedly inflammatory dermatosis with edema and nodules latter presentation is a *kerion*.



Tinea incognito: disguising and worsening of the signs due to Inadvertent topical corticosteroid application.



Diagnosis

Skin scraping or nail clippings by culture or direct microscopic examination .

Hyphae are often seen on KOH preparation.

Tineacapitis and tineacorporis may require culture or biopsy.

Management

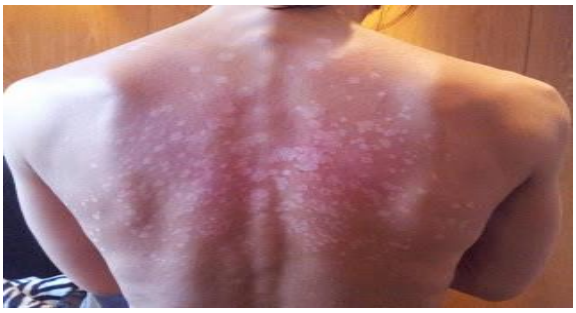
Depends on affected site and type of infection.

Topical imidazoles, triazoles, and allylamines may be effective.

Griseofulvin, 500 mg/d, if systemic therapy required.

Itraconazole or terbinafine may be effective for nail infections.

Tineaversicolor



It is caused by a nondermatophytic, dimorphic fungus, *Malassezia furfur*, a normal inhabitant of the skin.

Infection is promoted by heat and humidity.

The typical lesions consist of oval scaly macules, papules, and patches concentrated on the chest, shoulders, and back.

Rarely on the face or distal extremities.

On dark skin the lesions often appear as hypopigmented areas

On light skin they are slightly erythematous or hyperpigmented.

Diagnosis:

KOH preparation from scaling lesions will demonstrate a confluence of short hyphae and round spores (“spaghetti and meatballs”).

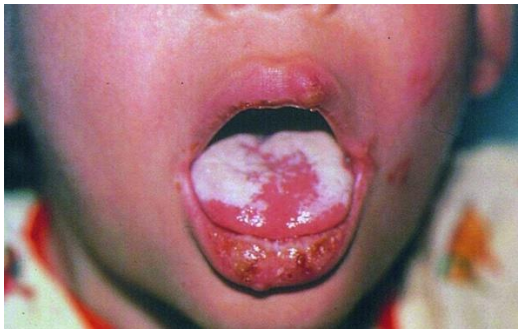
Management:

Lotions or shampoos containing sulfur, salicylic acid, or selenium sulphide.

A very short course of ketoconazole has been used, as have itraconazole and fluconazole.

Chapter 2.9

Candidiasis



It is Fungal infection caused by a related group of yeasts.

Manifestations may be localized to the skin or rarely systemic and life-threatening.

Superficial candidiasis is caused by Candida spp yeasts (mainly C. albicans).

Candida is a small, thin-walled, ovoid yeast that measures 4–6 μm in diameter and reproduces by budding.

These organisms are normal saprophytic inhabitants of the gastrointestinal tract.

May overgrow due to :

Broad-spectrum antibiotic therapy,

Diabetes mellitus

Immunosuppression and cause disease.

Predisposing factors:

Diabetes mellitus,

Cellular immune deficiencies

HIV

Common manifestations:

Oropharyngeal (‘thrush’)

Vaginal candidiasis

Intertrigo

Chronic paronychia.

Recurrent vaginal or penile candidiasis may be an early manifestation of diabetes mellitus.

Oropharyngeal

Lesions may occur on the tongue or buccal mucosa (*thrush*) and appear as white plaques.

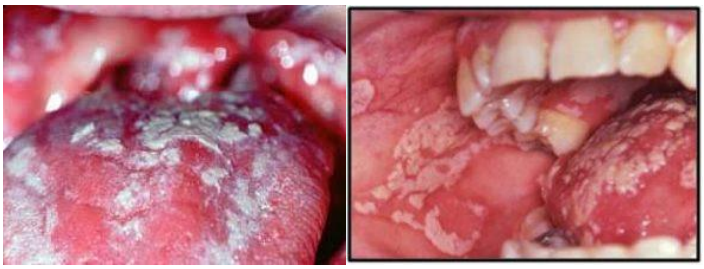
The yeast Candida albicansis a normal mouth commensal but it may proliferate to cause thrush.

Thrushis characterized by white, adherent, painless, discrete or confluent patches in the mouth, on the tongue, or in the oesophagus, occasionally with fissuring at the corners of the mouth.

Can also occur at points of contact with dentures.

White patches are seen on the tongue and buccal mucosa.

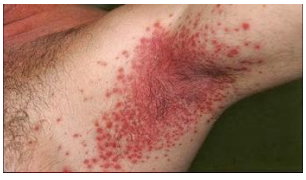
Odynophagia or dysphagia suggests pharyngeal and oesophageal candidiasis.



Candidal infections have an affinity for sites that are chronically wet and macerated, including the skin around nails (onycholysis and paronychia), and in intertriginous areas.

Intertriginous lesions are characteristically edematous, erythematous, and scaly, with scattered "**satellite pustules**"

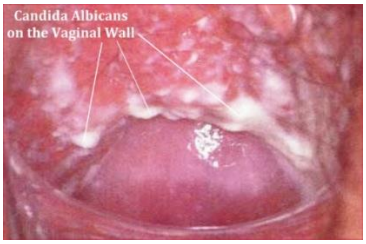
In males, there is involvement of the penis and scrotum as well as the inner aspect of the thighs. In contrast to dermatophyte infections, candidal infections are frequently painful and accompanied by a marked inflammatory response.



Intertrigo is characterised by inflammation in skin folds with surrounding ‘satellite lesions’.

Chronic paronychia is associated with occupations involving frequent wetting of the hands.

Vulvovaginal candidiasis is accompanied by pruritus, pain, and vaginal discharge which is usually thin but may contain whitish “curds” in severe cases.



Generalized disseminated cutaneous candidiasis, another form of infection that occurs primarily in infants, is characterized by widespread eruptions over the trunk, thorax, and extremities.

Diagnosis

A clinical diagnosis is sufficient to instigate therapy, although brushings or biopsies can be obtained for mycological examination.

Diagnosis of candidal infection is based upon the clinical pattern and demonstration of yeast on KOH preparation or culture.

Visualization of pseudohyphae or hyphae on wet mount (saline and 10% KOH), tissue.

Gram’s stain, periodic acid–Schiff stain, or methenamine silver stain in the presence of inflammation.

Treatment

Removal of predisposing factors

Topical nystatin or azoles (miconazole, clotrimazole, econazole, or ketoconazole)..

Systemic therapy reserved for immunosuppressed patients, unresponsive chronic or recurrent disease.

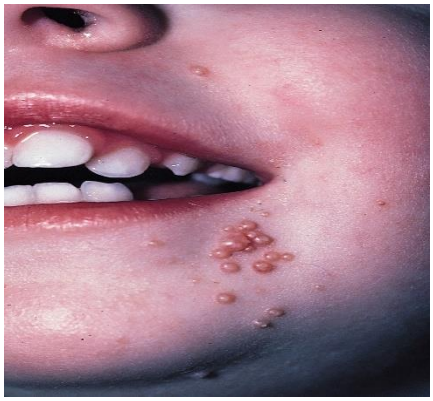
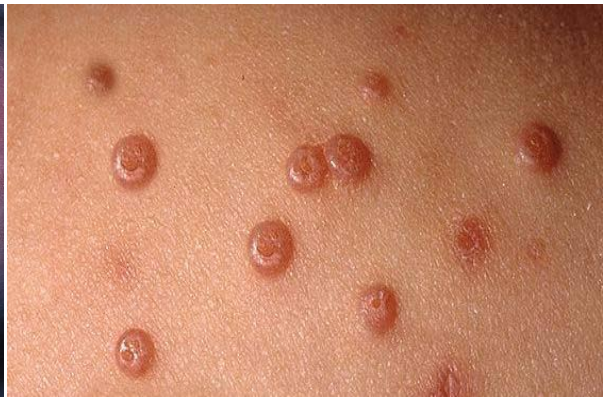
Vulvovaginal candidiasis responds well to a single dose of fluconazole, 150 mg.

Oral thrush is treated using nystatin or amphotericin suspensions or lozenges.

Resistant cases or immunosuppressed patients may require oral fluconazole.

Chapter 2.10

MolluscumContagiosum

It is a common cutaneous infection with a poxvirus.

The poxvirus family includes a large number of related DNA viruses that infect various vertebrate hosts.

Molluscumcontagiosum virus is an obligate human pathogen that causes distinctive proliferative skin lesions.

These lesions measure 2–5 mm in diameter and are pearly, flesh-colored, and umbilicated, with a characteristic dimple at the centercalled centralpunctum.

A relative lack of inflammation and necrosis distinguishes these proliferative lesions from other poxvirus lesions.

Distribution*:* Lesions may be found—singly or in clusters anywhere on the body except on the palms and soles.

It may be associated with an eczematous rash.

Age incidence: It can affect any age group but usually targets children over the age of 1 year.

Molluscumcontagiosum is highly prevalent among children and is the most common human disease resulting from poxvirus infection.

Genital lesions are more common in adults, to whom the virus may be transmitted by sexual contact.

Incubation Period : The incubation period ranges from 2 weeks to 6 months, with an average of 2–7 weeks.

Location : The lesions tend to be multiple and are often found in sites of apposition such as the side of the chest and the inner arm.

Transmission : Close contact, Swimming pools are a common vector for transmission.

Atopy and compromise of skin integrity increase the risk of infection.

The disease can be more generalized, severe, and persistent in AIDS patients than in other groups.

Moreover, it can be exacerbated in the immune reconstitution inflammatory syndrome (IRIS) associated with the initiation of antiretroviral therapy.

Diagnosis

Typically based on its **clinical presentation**

Can be confirmed by histologic demonstration of the cytoplasmic eosinophilic inclusions (***molluscum bodies***) that are characteristic of poxvirus replication.

Prognosis

The disease is self-limited and regresses spontaneously after 3–4 months in immunocompetent hosts.

Prior to resolution, they often become inflamed and may leave small, discrete, depressed scars.

There are no systemic complications, but skin lesions may persist for 3–5 years.

Management

There is **no specific systemic treatment** for molluscumcontagiosum, but a variety of techniques for **physical ablation** have been used.

Individual lesions can be removed by **curettage under local anaesthetic.**

Gentle squeezing with forceps after bathing can stimulate regression.

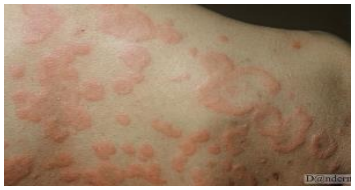
Topically applied chemicals such as salicylic acid, podophyllin and trichloroacetic acid cause marked inflammation and results vary.

Cryotherapy can be tried.

Topical 5% imiquimod cream has recently been shown to be effective.

Chapter 2.11

Urticaria and Angioedema



Urticaria are commonly seen condition with transient eruptions of raised and circumscribed erythematous or edematous swellings of the superficial dermis, associated with itching.

Urticaria (hives) are transient lesions that are composed of a central wheal surrounded by an erythematous halo or flare.

Individual lesions are round, oval, or figurate and are often pruritic.

Pruritic, edematous, pink to erythematous plaques with a whitish halo around margin of individual lesions.

Lesions range in size from papules to giant coalescent lesions (10–20 cm in diameter).

Often due to drugs, systemic infection, or foods (esp. shellfish).

If individual lesions last >24 h, consider diagnosis of urticarial vasculitis.

An area of focal dermal oedema secondary to a transient increase in capillary permeability.

Discrete and confluent, edematous, erythematous papules and plaques characteristic of whealing eruptions.

Can either acute or chronic, characterized by evanescent (individual lesions lasting <24 h)

Acute and chronic urticaria have a wide variety of allergic etiologies and reflect **edema in the dermis**.

Acute Urticaria is self limited and wheals resolve within 24 hours but may last upto 4-6 weeks. Common in young adults of both sexes.

Chronic urticarial the wheals continue daily or on most of the days for longer than 6 weeks. Common in women in fourth or fifth decade of life.

Pathogenesis of Urticaria

Urticaria is dermal edema resulting from **vascular dilatation** and **leakage of fluid into the skin** in response to molecules released from mast cells.

Mast cell degranulation occurs in :

Type I hypersensitivity causing massive degranulation and sometimes anaphylaxis.

Spontaneous mast cell degranulation in chronic urticaria.

Chemical mast cell degranulation.

Autoimmunity

Causes

Primary cutaneous disorders

Systemic diseases

I. Primary cutaneous disorders

A. *Acute and chronic urticaria*

B. *Physical urticaria*

1. Dermatographism

2. Solar urticaria

3. Cold urticaria

4. Cholinergic urticaria

C. *Angioedema* (hereditary and acquired)

II. Systemic diseases

A. Urticarial vasculitis

B. Hepatitis B or C infection

C. Serum sickness

D. Angioedema (hereditary and acquired)

Causes of Acute or chronic Urticaria :

Autoimmune due to production of antibodies that cross-link the IgE receptor on mast cells.

Allergens: in foods, inhalants and injections, (fruits, shellfish, fish, milk products, chocolate, peanuts).

Drugs: Salicylates, aspirin, codeine and NSAIDs, antibiotics, dextran and ACE inhibitors

Contact: e.g. animal saliva, latex.

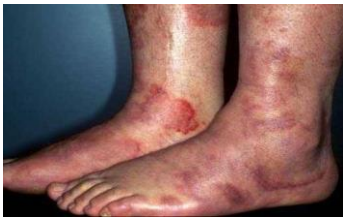
Physical: e.g. heat, cold, pressure, sun, water

Infection : e.g. viral hepatitis, infectious mononucleosis, HIV .

Other conditions: e.g. systemic lupus erythematosus (SLE), pregnancy, intestinal parasites

Idiopathic

Urticarial vasculitis



It is an immune complex disease that may be confused with simple urticaria.

Lesions last longer than 24 h and develops central petechiae observed even after the urticarial phase has resolved.

The patient complain of burning rather than pruritus.

Causes of Urticarial vasculitis

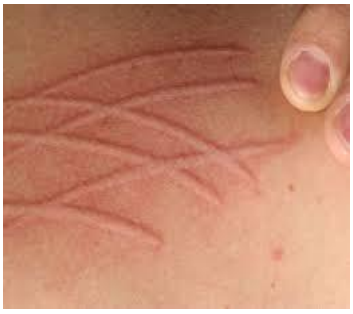
Hepatitis B

SLE

Idiopathic

Physical Urticaria

Dermatographism : linear wheals following minor pressure or scratching of the skin. Affect around 5% population.



Solar urticaria : occurs within minutes of sun exposure.

Cold urticaria : occurs on exposure to the cold.

Cholinergic urticaria: precipitated by heat, exercise, or emotion

Clinical Features

Itchy erythematous macules develop into wheals consisting of pale, pink or red, oedematous raised skin areas of varying shapes and sizes with a surrounding flare.

Transient and migratory lesions with linear, annular or arcuate pattern anywhere in body.

Itching is present usually at night, but patient tends to rub rather then scratching.

Lesions resolve leaving a normal skin surface, without any excoriation marks.

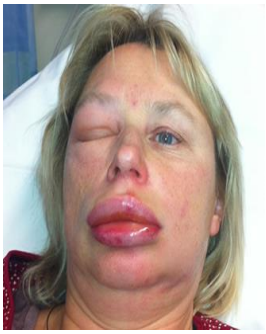
If the lesions lasts for >24 Hours Urticarial vasculitis is suspected.

Half of the cases of Uritcaria are associated with angioedema.

Urticaria result from dermal edema,whereas subcutaneous edema leads to *angioedema*.

Few cases can be associated with malaise, fever, headache, dizziness, nausea, vomiting, abdominal pain, arthralgia, feeling of lump in throat, wheezing, shortness of breath syncope and in severe acute form anaphylaxis.

Angioedema



It is characterized by large non pruritic or slightly itchy, non pitting, pale or pink diffuse swelling occurring on the face, affecting the eyelids, lips, tongue, pharynx and larynx, hands, feet, genitalia, ears and neck.

The lesions of angioedema may last for several days.

Sites: the eyelids, lips, tongue, larynx, and gastrointestinal tract as well as the subcutaneous tissue.

Angioedema occurs alone or in combination with urticaria, including urticarial vasculitis and the physical urticarias.

Urticarial lesions can also be seen in patients with mastocytosis (urticaria pigmentosa), hypo- or hyperthyroidism, and systemic-onset juvenile idiopathic arthritis (Still's disease).

In both juvenile- and adult-onset Still's disease, the lesions coincide with the fever spike, are transient, and are due to dermal infiltrates of neutrophils.

Diagnosis

History, with special attention to possible offending exposures and/or ingestion as well as the duration of lesions.

Skin testing to food and/or inhalant antigens.

Physical provocation, e.g., challenge with vibratory or cold stimuli.

Full blood count, including eosinophil count in case of underlying parasites.

ESR: may be elevated in cases of vasculitis

Urea and electrolytes, thyroid and liver function tests: for underlying systemic disorder.

Total IgE and specific IgE to possible allergens, e.g shellfish or peanuts

Antinuclear factor: may be positive in chronic urticaria or urticarial vasculitis.

Skin biopsy: helpful if urticarial vasculitis.

Management

Identification and avoidance of offending agents.

H1 antihistamines : Non-sedative antihistamines cetirizine, levocetirizine. (1/3-1/3-1/3)

H2 Blockers : ranitidine

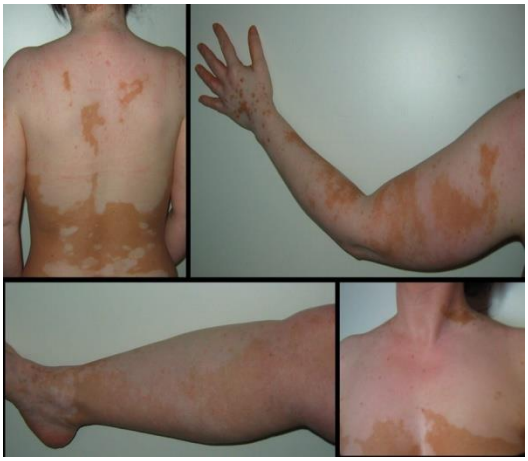
Patients with history of angioedema or anaphylaxis, should carry a self-administered injection kit of adrenaline.

Topical glucocorticoids are of no value.

Systemic glucocorticoids should not be used because of their long-term toxicity.

Chapter 2.12

Vitiligo



Vitiligo is an acquired condition in which circumscribed depigmented patches develop surrounded by normal skin.

It affects 1% of the population world-wide.

It involves focal areas of melanocyte loss.

Based on the distribution on the body it can be classified as:

Localized

Generalized

Universal

Halo naveus

Incidence :

Worldwide distribution.

Most common in India (Gujrat and Rajasthan), Egypt and tropical countries.

Affects all age groups.

Both sex have equal predilection

Many cases start at five, fifteen an at menopause.

There may be a positive family history of the disorder in those with generalised vitiligo.

Etiopathogenesis

Autoimmunity: antibodies against adrenal, thyroid cytoplasm. Actually a reaction pattern to drugs, infection and toxins.

Neurohormonal factors: neurotoxic agents norepinephrine, catecholamines inhibit melanogenesis and have toxic effect on melanocytes

Autocytotoxicity: self destruction of melanocytes

Exogenous chemical exposure: thiols, phenols inhibit action of tyrosinase and cause cytotoxic action on melanocytes.

Etiology

Genetic predisposition : 35% with family history

Nutritional : Low nutritious diet, low protien, copper, vitamin and minerals.

Endocrine : Thyrotoxicosis, hypothyroidism, diabetes etc are associated.

Psychological : Emotional stress and strain

Infection : Enteric fever, focal sepsis etc

Drugs : Quinines, broad spectrum antibiotics etc

Other factors : Chemical dyes, food adultrants, contaminated food and water.

Pathology

Defect in Tyrosinase is responsible for vitiligo.

Melatonin secreted from the nerve endings inhibits tyrosinase, thus interfering in pigment formation.

There is marked absence of melanocytes and melanin in the epidermis.

In inflammatory vitiligo there is a raised erythematous border, with infiltration of lymphocytes and histeocytes.

Clinical Features

Can occur at any age.

50% develops before age of 20 years

Slow progressive. Onset is slow and insidious, come to a halt and increases again.

Distribution :

Found commonly on face, axilla, groins, areola and genitalia.

Areas subjected to repeated friction and trauma eg. Dorsum of hand, feet, elbow, knees etc.

Usually symmetrical.

Clinical Criteria for Classification of Vitiligo

Stage of Vitiligo (V)

Active (V1)

New lesions are developing

Lesions are increasing in size

Border ill defined

Quiescent/stable (V2)

No new lesions developing

Lesions stationary in size

Border hyperpigmented and well defined

Improving (V3)

Lesions decreasing in size

No new lesions developing

Borders defined and signs of spontaneous repigmentation (follicular and peripheral)

Zostiformis/segmental.

Unilateral distribution of lesions, preferably along the course of nerves.

Diagnosis

Usually apparent by appearance.

Wood lamp examination is of great importance.

Prognosis

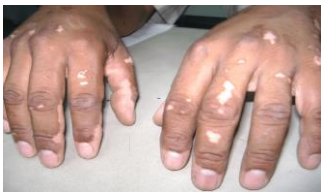
Resistant areas :

Lips, over the joints, acral areas.

Patches with depigmented hairs.

Patches with lack of vascularity.

Patches with thickened integument, scaly and tendency to fibrosis.



Poor response is seen in cases of :

Poor nutrition

Emotional stress

Recurrent infections

Extensive coverage

Endemic areas

Age above 60 years.

Management

PUVA (Psoraleans +UVA) is very effective.

Alternative sunlight exposure

Systemic steroids in case of extensive involvement

Laser treatment

Skin grafting

Homoeopathic Medicines

Selection of Homoeopathic Medicine is done on the constitutional medicine based on the totality of symptoms after proper case taking as explained by our master Hahnemann in Aphorism 83-104

But in many cases where complete case taking can not be done some homoepathic medicines can be given on the therapeutic basis some of which are discussed below.

Anthracinum :boils and boil-like eruptions, acne. Terrible burning. Induration of cellular tissue, abscess.

AntimoniumCrudum :Pimples, pustules, and boils on face. Yellow crusted eruption on cheeks and chin.

ArsenicumBromatum :Acne rosace*a*, with violet papules on nose; worse in the spring. Acn*e* in young people.

AsteriasRubens :Red. Pimples on side of nose, chin and mouth. Disposition to pimples at adolescence.

Arsalb: Itching, burning, swellings; œdema, eruption, papular, dry, rough, scaly. <cold and scratching.

Arsiod: Dry, scaly, itching. Marked exfoliation of skin in large scales, leaving a raw exuding surface beneath.

BerberisAquifolium :Pimply, dry, rough, scaly. Acne

Bovista : Pimples cover the entire body

Eugenia Jambos : Acne, simple and indurated. The pimples are painful for some distance around. Acne rosacea

JuglansRegia : Comedones and acne of the face.

KaliumBromatum :*Acne* of face, pustules. Itching; worse on chest, shoulders, and face

Lappaarctium:ruptions on the head, face, and neck; pimples; acne. Styes and ulcerations on the edge of the eyelids, Crops of boils and stye.

Graphitis: Rough, hard, persistent dryness of portions of skin. Eruptions, oozing out a sticky exudation.

Petroleum :Skin dry, constricted, very sensitive, rough and cracked, leathery. Burning and itching *worse in winter.*

Sepia : Herpes circinatus in isolated spots. Itching worse in bends of elbows and knees. Sweat on feet.skin feels itchy but scratching provides no relief. the symptoms worsen when on exposure to open air.

Sulphur : Dry, scaly, unhealthy skin. Itching, burning; worse scratching and washing. Excoriation, especially in folds.Pruritus, especially from warmth, in evening. Itching recurs in spring-time, in damp weather.skin is itchy and irritated, itch worsens with the application of water or heat. symptoms aggravated during night .

Calcareacarb:Unhealthy; readily ulcerating . Arthritic nodosities, Swelling of joints.

Mezereum : intolerable itching worse in bed surrounded by vesicles and shining, fiery-red areola.thick scabs under purulent matter exudes. long bones, inflamed and swollen.

Mercsol : Vesicular and pustular eruptions. *Itching*, worse from warmth of bed. yellowish-brown crusts, considerable suppuration. *C*onstantly moist skin.

Rhustox: Skin Red, swollen; intense itching. Burning eczematous eruptions with tendency to scale formation.

Staphysagria : Eczema of head, ears, face, and body; thick scabs, dry, and itch violently; scratching changes location of itching. Inflammation of phalanges. Arthritic nodes.

Kreosotum :Itching, worse towards evening.

Psorinum: Intolerable itching on scalp and bends of joints with itching; worse, from warmth of bed.

Chrysarobinum: Vesicular or squamous lesions, associated with foul smelling discharge and crust formation, tending to become confluent and to give the appearance of a single crust covering the entire area. Violent itching, thighs, legs and ears. Dry, scaly eruption, especially around eyes and ears, scabs.

Tellurium metallicum: Itching of hands and feet. Herpetic spots; ringworm. Ring-shape lesions.

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