Women University Press
Jayoti Publication Desk
ISBN No. : 978-93-90892-69-3

ESSENCE OF PRACTICE OF MEDICINE VOL-2

JV'n Dr. Ravi Jain

JAYOTI VIDYAPEETH WOMEN'S UNIVERSITY, JAIPUR

UGC Approved Under 2(f) & 12(b) | NAAC Accredited | Recognized by Statutory Councils

Printed by :
JAYOTI PUBLICATION DESK

Published by :
Women University Press
Jayoti Vidyapeeth Women's University, Jaipur

Faculty of Homoepoathic Science

Title: Essence of Practice of Medicine Part 2

Author Name: Dr. Ravi Jain

Published By: Women University Press

Publisher's Address: Jayoti Vidyapeeth Women's University, Jaipur
Vedant Gyan Valley,
Village-Jharna, Mahala Jobner Link Road, NH-8
Jaipur Ajmer Express Way,
Jaipur-303122, Rajasthan (India)

Printer's Detail: Jayoti Publication Desk

Edition Detail:

ISBN: 978-93-90892-69-3

Essence of Practice of Medicine
Part 2

By: JV'n Dr Ravi Jain

MD (Hom)
Assistant Professor
Department of Practice of Medicine
Faculty of Homoeopathic Science
Jayoti Vidyapeeth Women's University, Jaipur
Email: ravij0000@gmail.com
Mob: 9413349417

Foreward

This book is the second book in the series Essence of Medicine. The first volume was much appreciated by the students which made me to finish the book in such a short span of time.

I would like to give my gratitude towards our Honorable Chairperson Mam Jv'nVidushi Garg and our Honorable Founder and Advisor sir JV'n Dr Panckaj Garg sir for providing me an opportunity to write this book and publish in University press for the need of our students at Jayoti Vidyapeeth Women's University Jaipur.

This volume of the book is dedicated to the Students of BHMS, BAMS, BNYS. This is the second part of the series Essence of Medicine. This part covers diseases of Neurology & Psychiatry together in one chapter and Diseases of Locomotor system in the second chapter in a comprehensive and easy way. This is not a textbook and can neither replace any rather a compilation of notes from my classroom teachings. This provides a quick review to the students of the chapters Diseases of Neurology & Psychiatry and Diseases of Locomotor system.

Neurology and Psychiatry are very vast topics but an attempt has been made to present the topics in a simplified form.
Diseases of Locomotor system are very common and Homoeopathy provides wonderful response in such cases.
Hence these two topics are included in this volume. The topics are explained in short and only points are given for a quick review. The source books have to be consulted for and this is never a substitute. 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

Neurology Introduction

Neurological disorders are diseases of the brain, spine and the nerves that connect them.

These are diseases of the central and peripheral nervous system.

Structural, biochemical or electrical abnormalities in the brain, spinal cord or other nerves can result in a range of symptoms.

There are more than 600 diseases of the nervous system, some relatively common, but many rare.

Knowledge of the relevant anatomy and physiology of the nervous system helps to determine the site of the lesion.

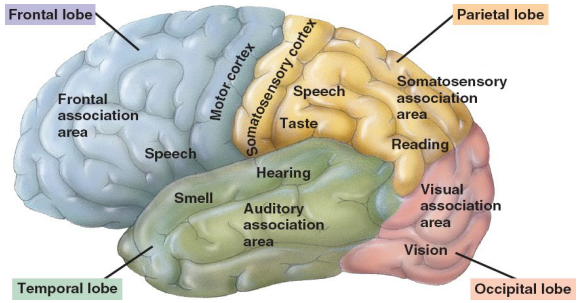


Fig.1 Brain with Function of different parts of brain.

Causes of Neurological Disease

Genetic disorders

Congenital abnormalities

Infections

Lifestyle or environmental - malnutrition.

Injury : brain, spinal cord, nerve.

Metal poisoning

Idiopathic

Symptoms

Paralysis

Muscle Weakness

Poor Coordination

Loss of Sensation

Seizures

Confusion

Pain

Altered levels of consciousness.

Intervention

Preventative measures

Lifestyle changes

Physiotherapy or other therapy

Neuro-rehabilitation

Pain management

Medication

Operations

Specific diet

Cranial Nerves with Functions

The Cranial Nerves			
Nerve Number and Name		Composition	Some Functions
I	Olfactory	Sensory only	Olfaction (smell)
II	Optic	Sensory only	Vision
III	Oculomotor	Motor and sensory	Serves muscles of the eye
IV	Trochlear	Motor and sensory	Serves the superior oblique eye muscle
V	Trigeminal	Motor and sensory	Sensory from face and mouth; motor to muscles of mastication (chewing)
VI	Abducens	Motor and sensory	Serves the lateral rectus eye muscle
VII	Facial	Motor and sensory	Serves the muscles of facial expression, lacrimal glands, and salivary glands
VIII	Vestibulocochlear	Sensory only	Equilibrium and hearing
IX	Glossopharyngeal	Motor and sensory	Serves the pharynx (throat) for swallowing, posterior third of tongue, parotid salivary gland
X	Vagus	Motor and sensory	Sensations from visceral (internal) organs, and parasympathetic motor regulation of visceral organs
XI	Accessory	Motor and sensory	Serves muscles that move head, neck, and shoulders
XII	Hypoglossal	Motor and sensory	Serves muscles of the tongue

Fig. 2 Cranial Nerves and their functions. Courtesy Harrison Principles of Internal Medicine 19th Edition

Chapter 1.2

Headache

Headache is the symptom of pain anywhere in the region of head and neck.

It has **multifactorial origin** and can be a sign of stress or emotional distress, or it can result from a medical disorder, such as migraine or high blood pressure, anxiety, or depression.

A painful sensation in any part of the head, ranging from sharp to dull, that may occur with other symptoms.

The most common reasons patients seek medical attention, and responsible for more disability than any other neurologic problem.

Intensity of head pain rarely has diagnostic value.

The location can suggest involvement of local structures.

Temporal pain in giant cell arteritis.

Facial pain in sinusitis.

Ruptured aneurysm has instant onset.

Cluster headache has a peak over 3–5 minutes.

Migraine pain increases over minutes to hours.

Provocation by environmental factors suggests a benign cause.

Types of Headache

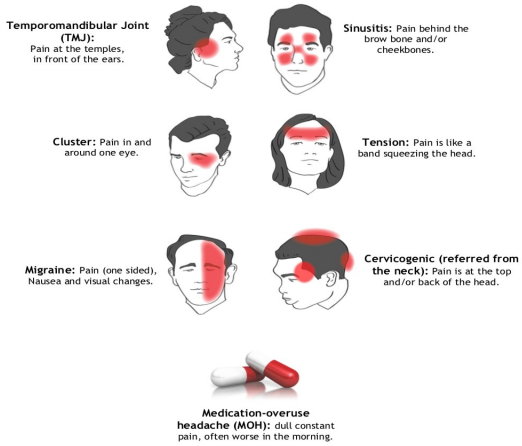


Fig Showing Types of headache

Classification of Headache

International Headache Society has classified headache as :

Primary Headache : those in which headache and its associated features are the disorder in itself, they are not the result of another medical condition.

Secondary Headache : headaches are those which are caused by exogenous disorders, when it is caused by another condition.

Primary Headache

Tension-type - 69%

Migraine - 16%

Idiopathic stabbing pain – 2%

Exertional 1%

Cluster - 0.1 %

Primary headache often results in considerable disability and a decrease in quality of life.

Secondary Headache

Systemic infection - 63%

Head injury - 4%

Vascular disorders - 1%

Subarachnoid hemorrhage - <1%

Brain tumor - 0.1%

Physiology of Headache

Pain usually occurs due to :

Stimulation of peripheral nociceptors (pain receptors) in response to tissue injury, visceral distension, or other factors.

It is a normal physiologic response mediated by a healthy nervous system.

Damage or inappropriate activation of pain producing pathways of the peripheral or central nervous system.

Headache may originate from either or both the mechanisms.

Clinical Evaluation

New, severe headache has a differential diagnosis that is quite different from the patient with recurrent headaches over many years because of probability of finding a potentially serious cause in new cases.

Serious causes to be considered: meningitis, subarachnoid hemorrhage, epidural or subdural hematoma, glaucoma, tumor, and purulent sinusitis.

History Taking

The overall pattern (intermittent or continuous)

The speed of onset

The time of day of onset of maximal pain

The effect of posture, coughing and straining

The location of the pain

Any associated symptoms

Complete neurologic examination is important in evaluation of headache.

If examination is abnormal or if serious underlying cause is suspected, an imaging study (CT or MRI) is indicated as a first step.

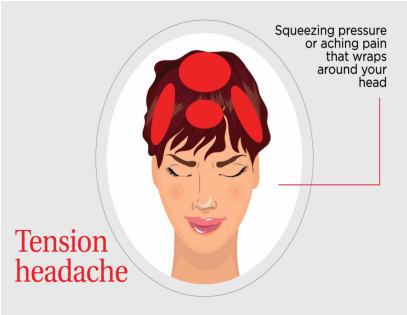
Lumbar puncture (LP) is required when meningitis (stiff neck, fever)

The psychological state of the patient should also be evaluated to prevent depression.

Primary Headache Types

The classification of primary headache includes

Tension-Type Headache:



Common in all age groups.

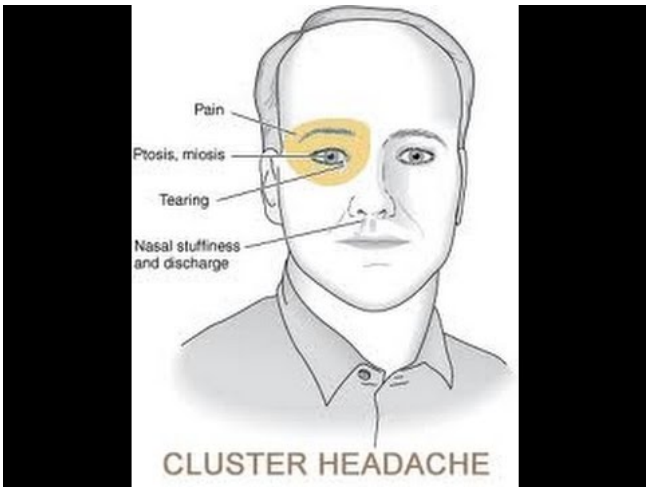
Usually builds slowly.

May persist for hours or days.

It is described as bilateral tight, band like discomfort.

Pain is managed generally with simple analgesics - acetaminophen, aspirin, or NSAIDs.

Cluster Headache:



These are a series of relatively short but extremely painful headaches every day for weeks or months at a time, usually occur in cyclical patterns called *cluster* periods.

Characterized by episodes of recurrent, deep, unilateral, retro-orbital searing pain.

Unilateral lacrimation and nasal and conjunctival congestion may be present.

Prophylaxis with verapamil or prednisone.

High-flow oxygen is useful for the acute attack.

Migraine

It is a neurological condition that can cause multiple symptoms.

It is characterized by **intense, debilitating headaches**.

Symptoms includes :

Nausea, vomiting, difficulty speaking, numbness or tingling, and sensitivity to light and sound.

Classic triad:

Premonitory visual (scotoma or scintillations), sensory, or motor symptoms (Aura)

Unilateral throbbing headache

Nausea and vomiting.

It is the second most common cause of headache, and the most common neurologic cause of disability in the world.

It affects approximately 15% of women and 6% of men annually.

Onset usually in childhood, adolescence, or early adulthood, however, initial attack may occur at any age.

Family history is often present.

Women may have increased sensitivity to attacks during menstrual cycle.

It is often be recognized by its activators, referred to as **triggers**.

The patient is sensitive to environmental and sensory stimuli, to which they do not habituate easily.

It is amplified during the menstrual cycle.

The triggering factors includes glare, bright lights, sounds, hunger, stress, physical exertion, hormonal fluctuations, lack of sleep, alcohol, or other chemical stimulation.

Pathogenesis

The exact pathophysiology is unknown.

The sensory sensitivity is due to dysfunction of **monoaminergic sensory control systems** located in the brainstem and hypothalamus.The blood level of histamine, serotonin and norepinephrine is increased.

Activation of cells in the **trigeminal nucleus** results in the release of vasoactive neuropeptides, particularly calcitonin gene–related peptide (CGRP), at vascular terminations of the trigeminal nerve. CGRP receptor antagonists,have been shown to be effective in the acute treatment of migraine.

Dopaminergic stimulation can induce symptoms of migraine.

Diagnostic Criteria for Migraine

Repeated attacks of headache **lasting 4–72 hours** with a **normal physical examination**, no other reasonable cause for the headache, and :

At Least Two of the Following Features :

Unilateral pain

Throbbing pain

Aggravation by movement

Moderate or severe intensity

Plus at Least One of the Following Features:

Nausea/vomiting

Photophobia and phonophobia

Classification of Migraine

1.1 Migraine without aura

1.2 Migraine with aura

1.2.1 Migraine with typical aura

1.2.1.1 Typical aura with headache

1.2.1.2 Typical aura without headache

1.3 Chronic migraine

1.4 Complications of migraine

1.5 Probable migraine

1.6 Episodic syndromes that may be associated with migraine

The migraine aura, consisting of visual disturbances with flashing lights or zigzag lines moving across the visual field or of other neurologic symptoms.

It is reported in 20–25% of patients.

Chronic migraine : When episodes of migraine occurs daily or near-daily, for 15 days or more a month.

Acephalgic migraine : typical aura without headache, there are recurrent neurologic symptoms, often with nausea or vomiting, but with little or no headache. Vertigo is prominent.

One-third of patients referred for vertigo or dizziness have a primary diagnosis of migraine.

Symptoms

Nausea	87%
Photophobia	82%
Lightheadedness	72%
Scalp tenderness	65%
Vomiting	56%

Visual disturbances 36%

Paresthesias 33%

Vertigo 33%

Photopsia 26%

Alteration of consciousness 18%

Diarrhea 16%

Fortification spectra 10%

Syncope 10%

Seizure 4%

Confusional state 4%

Migraine Disability Assessment Score

Enquire about the headaches over the last 3 months.

Questions 1−5 will be used to calculate the MIDAS score

MIDAS Questionnaire

1. On how many days in the last 3 months did you miss work or school because of your headaches?

2. How many days in the last 3 months was your productivity at work or school reduced by half or more because of your headaches? (Exclude Q1 days)

3. On how many days in the last 3 months did you not do household work because of your headaches?

4. How many days in the last 3 months was your productivity in household work reduced by half or more because of your headaches? (Exclude Q3 days)

5. On how many days in the last 3 months did you miss family, social, or leisure activities because of your headaches?

A. On how many days in the last 3 months did you have a headache?

B. On a scale of 0–10, on average how painful were these headaches?

Interpretation

Grade I—Minimal or Infrequent Disability: 0–5

Grade II—Mild or Infrequent Disability: 6–10

Grade III—Moderate Disability: 11–20

Grade IV—Severe Disability: > 20

Treatment

Three approaches

Nonpharmacological - avoidance of triggers

Pharmacological- Simple analgesics, NSAIDs, etc

Prophylaxis - tricyclic antidepressants, verapamil etc

Chapter 1.3

Trigeminal Neuralgia

Trigeminal neuralgia is a chronic pain condition that affects the **trigeminal** nerve, which carries sensation from the face to the brain.

The trigeminal (fifth cranial) nerve supplies sensation to the skin of the face and anterior half of the head.

It divides into **three divisions**, **ophthalmic , maxillary and mandibular.**

It is predominantly sensory, and motor innervation is exclusively carried in mandibular.

Incidence

It is relatively common.

Annual incidence of 4–8 per 100,000 individuals.

Middle-aged and elderly persons are affected.

More common in women.

Clinical Manifestations

Onset is typically sudden.

It is characterized by **severe paroxysmal pain** in the lips, gums, cheek, or chin, rarely, in the distribution of the **ophthalmic division** of the fifth nerve.

Pain lasts a few seconds to a few minutes.

The pain is so intense that the patient winces, hence the term tic.

The paroxysms, experienced as single jabs or clusters, which recur frequently, both day and night, for several weeks at a time.

Onset is typically sudden, and bouts tend to persist for weeks or months before remitting spontaneously.

The pain occurs spontaneously or with movements of affected areas by speaking, chewing, or smiling.

Trigger zones, that provoke attacks on the face, lips, and tongue.

Tactile stimuli like washing the face, brushing the teeth, or exposure to a draft of air can generate excruciating pain.

Remissions are longlasting, but in most, the disorder ultimately recurs.

Pathophysiology

Symptoms result from ectopic generation of action potentials in pain-sensitive afferent fibers of the fifth cranial nerve root.

Compression or other pathology in the nerve leads to demyelination of large myelinated fibers that do not themselves carry pain sensation but become hyperexcitable and electrically coupled with smaller unmyelinated or poorly myelinated pain fibers.

Compression of the trigeminal nerve root by a blood vessel at the entry of brainstem is the source of trigeminal neuralgia.

Increased vascular thickness and tortuosity causes the prevalence of trigeminal neuralgia in later life.

Differential Diagnosis

Pain arising from diseases of the jaw, teeth, or sinuses.

Pain from migraine or cluster headache.

Temporal arteritis.

Multiple sclerosis

Table 4
Differential diagnosis of trigeminal neuralgia

Diagnosis	Distinguishing Characteristics
Cluster HA	Pain lasts longer; orbital or supraorbital; autonomic symptoms
Dental pain	Localized; sensitive to hot and cold foods; abnormal oral examination
Giant cell arteritis	Persistent pain, jaw claudication
Glossopharyngeal neuralgia	Pain in tongue, mouth, or throat brought on by swallowing, talking, or chewing
Intracranial tumors	Other neurologic signs possible
Migraine	Longer-lasting pain, family history, photophobia, phonophobia
Multiple sclerosis	Eye symptoms, other neurologic symptoms
Otitis media	Pain localized to ear; abnormal ear examination
Paroxysmal hemicrania	Pain in forehead or eye; autonomic symptoms; responds to indomethacin
Post-herpetic neuralgia	Continuous pain, tingling, history of zoster; usually in ophthalmic division of V
Sinusitis	Persistent pain; associated nasal symptoms
SUNCT	Ocular or periocular, autonomic symptoms
TMJ syndrome	Persistent pain; localized tenderness, jaw abnormalities
Trigeminal neuropathy	Persistent pain; associated sensory loss

Abbreviations: SUNCT, shorter lasting unilateral neuralgiform, conjunctival injection, and tearing; TMJ, temporomandibular joint.
Adapted from Krafft R. Trigeminal neuralgia. Am Fam Physician 2008;77(9):1291–6; with permission.

Table: Source Harrison Principles of Internal Medicine 19th Edition.

Lab Investigations

ESR is raised in temporal arteritis.

Neuroimaging studies are usually unnecessary.

It can be valuable if MS is a consideration or in assessing overlying vascular lesions in order to plan for decompression surgery.

High-resolution magnetic resonance angiography is useful preoperatively to visualize the relationships between the fifth cranial nerve root and nearby blood vessels.

Treatment

Drug therapy with carbamazepine is effective.

Dizziness, imbalance, sedation, agranulocytosis side effects of carbamazepine.

Oxcarbazepine is an alternative to carbamazepine.

If drug treatment fails, surgical therapy - microvascular decompression to relieve pressure on the trigeminal nerve.

Gamma knife radiosurgery of the trigeminal nerve root is also used for treatment.

Chapter 1.4

Bell's palsy

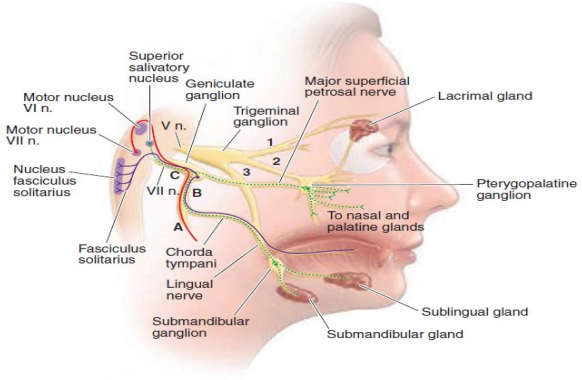


Fig Facial nerve supplies all the muscles concerned with facial expression.

It is the most common form of idiopathic facial paralysis.

Annual incidence is 25 per 100,000 annually.

It affects 1 in 60 persons over a lifetime.

All ages and both sexes are affected.

Risk factors are pregnancy and diabetes mellitus.

The seventh cranial nerve supplies all the muscles concerned with facial expression.

Pathophysiology

There is inflammation of the facial nerve with mononuclear cells, consistent with an infectious or immune cause.

Herpes simplex virus was frequently detected.

Reactivation of varicella-zoster virus is the second most frequent cause.

Increased incidence was reported among recipients of inactivated intranasal influenza vaccine-reactivation of latent virus.

Clinical Manifestations

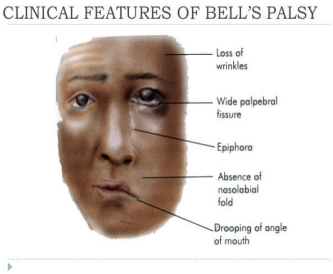
Onset is abrupt, with maximal weakness being attained by 48 h as a general rule.

Pain behind the ear precedes the paralysis for a day or two.

Taste sensation may be lost unilaterally, and hyperacusis may be present.

80% of patients recover within a few weeks or months.

Evidence of denervation after 10 days indicates axonal degeneration.



Inability to open or close your eye on the affected side

Drooling

Difficulty eating and drinking

An inability to make facial expressions, such as smiling or frowning

Facial weakness

Muscle twitches in the face

Dry eye and mouth

Headache

Sensitivity to sound

Irritation of the eye on the involved side

A **complete interruption of the facial nerve** at the **stylomastoid foramen** paralyzes all muscles of facial expression.

The corner of the mouth droops, the creases and skinfolds are effaced, the forehead is unfurrowed, and the eyelids will not close.

Upon attempted closure of the lids, the eye on the paralyzed side rolls upward (Bell's phenomenon).

The lower lid sags and falls away from the conjunctiva, tears spill over the cheek.

Food collects between the teeth and lips, and saliva dribble from the corner of the mouth.

There is heaviness or numbness in the face.

Sensory loss is rarely demonstrable and taste is intact.

If **lesion over middle-ear portion**, taste is lost over the anterior two-thirds of the tongue on the same side. If the nerve to the stapedius is interrupted, there is hyperacusis (sensitivity to loud sounds).

Lesions in the internal auditory meatus may affect the adjacent auditory and vestibular nerves, causing deafness, tinnitus, or dizziness.

The palpebral fissure becomes narrowed, and the nasolabial fold deepens.

Attempts to move one group of facial muscles results in contraction of all (synkinesis).

Facial spasms, initiated by movements of the face, may develop (hemifacial spasm).

If fibers connected with the orbicularis oculi innervate the orbicularis oris, closure of the lids cause a retraction of the mouth.

If innervate the lacrimal gland, anomalous tearing **crocodile tears** occurs with any activity of the facial muscles.

Jaw opening, causing closure of the eyelids on the side of the facial palsy (jaw-winking).

Differential Diagnosis

Lyme disease - Infection with Borrelia burgdorferi.

Ramsay Hunt syndrome - reactivation of herpes zoster.

Sarcoidosis

Guillain-Barre syndrome

Leprosy

Connective tissue diseases - Sjogren's syndrome, and amyloidosis.

Acoustic neuromas - by local compression.

Tumors that invade the temporal bone.

Lab Evaluation

Typical presentation.

No risk factors or pre-existing symptoms for other causes of facial paralysis.

Absence of cutaneous lesions of herpes zoster in the external ear canal.

Normal neurologic examination with the exception of the facial nerve.

MRI shows swelling and enhancement of the facial nerve in idiopathic Bell's palsy.

There is mild cerebrospinal fluid lymphocytosis.

Electromyography may be of some prognostic value.

Prognosis

Presence of incomplete paralysis in the first week is the most favourable prognostic sign.

With or without treatment, most individuals begin to get better within 2 weeks after the initial onset of symptoms and most recover completely, returning to normal function within 3 to 6 months.

Recurrences are reported in 7% of cases.

Treatment

Use of paper tape to depress the upper eyelid during sleep and prevent corneal drying.

Topical ocular therapy

Massage of the weakened muscles.

Glucocorticoids, prednisone is given daily to shorten the recovery period and improves the functional outcome.

Antiviral drugs.

Chapter 1.5

Seizures & Epilepsy

A seizure is a paroxysmal event due to **abnormal excessive** or **synchronous neuronal** activity in the brain.

Seizures are a result of a shift in the normal balance of excitation and inhibition within the CNS.

Abnormal brain activity manifest from experiential phenomena not readily discernible by an observer to dramatic convulsive activity.

Epilepsy is a condition in which a person has recurrent seizures due to a chronic, underlying process.

Recurrent seizures due to correctable or avoidable circumstances, does not necessarily have epilepsy.

The prevalence of epilepsy is around 5–30 persons per 1000.

Trigger Factors for Seizures

Sleep deprivation

Alcohol (withdrawal)

Recreational drug misuse

Physical and mental exhaustion

Flickering lights, including TV and computer screens

Intercurrent infections and metabolic disturbances

Uncommonly: loud noises, music, reading, hot baths

Classification of Seizures

Classification of Seizures is essential for:

Diagnostic approach on etiology.

Selecting appropriate therapy.

Prognosis of the case.

The International League against Epilepsy (ILAE) Commission

Classification of Seizures

Focal seizures

Generalized seizures

May be focal, generalized, or unclear – Epileptic spasm

Classification is based on the clinical features of seizures and associated electroencephalographic findings.

Focal seizures	**Generalized seizures**
Originate within networks limited to one cerebral hemisphere	Arise within and engage network distributed across both cerebral hemispheres.
Usually associated with structural abnormalities of the brain	Result from cellular, biochemical, or structural abnormalities that have a more widespread distribution.

Generalized seizures

a. Absence

Typical

Atypical

b. Tonic clonic

c. Clonic

d. Tonic

e. Atonic

f. Myoclonic

Classification can also be done by

Seizure type

Physiology (EEG)

Anatomical site

Pathological cause

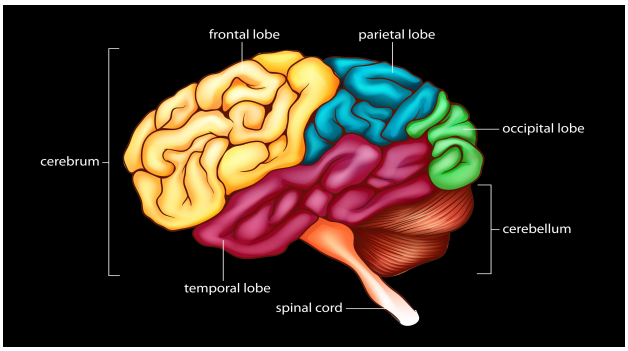


Fig Showing Lobes of Brain

Seizure Type

Partial (simple or complex)

Partial with secondary generalisation

Absence

Tonic clonic

Tonic

Atonic

Myoclonic

Physiology EEG

Focal spikes/sharp waves

Generalised spike and wave

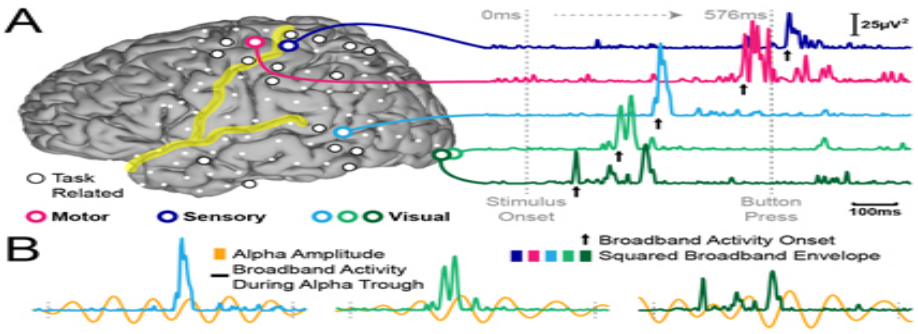


Fig. EEG Wavepattern

Anatomical Site

- **Cortex**

Temporal

Frontal

Parietal

Occipital

- **Generalised (diencephalon)**
- **Multifocal**

Pathological Cause

Genetic

Developmental

Tumours

Trauma

Vascular

Infections

Inflammation

Metabolic

Drugs, alcohol and toxins

Degenerative

Focal Seizures

Localized within one cerebral hemisphere or more broadly distributed but still within the hemisphere.

These are classified **on the presence of cognitive impairment** into:

Focal seizures without dyscognitive features.

Focal seizures with dyscognitive features.

Focal seizures can also evolve into generalized seizures.

Focal Seizures Without Dyscognitive Features

Focal seizures can cause motor, sensory, autonomic, or psychic symptoms without impairment of cognition.

Eg. onset of involuntary movements typically clonic of left handi.e., repetitive, flexion/extension movements at a frequency of ~2–3 Hz.

The EEG recorded during the seizure (Ictal EEG) show abnormal discharges in a very limited region over the appropriate area of cerebral cortex.

Three additional features of focal motor seizures :

Jacksonian march : the abnormal motor movements begins in a very restricted region such as the fingers and gradually progress to include a larger portion of the extremity.

Todd's paralysis : localized paresis for minutes to many hours in the involved region following the seizure.

Epilepsia partialis continua : the seizure continue for hours or days refractory to medical therapy.

Focal seizures may also manifest as :

Changes in somatic sensation (e.g., paresthesias)

Vision (flashing lights or formed hallucinations)

Equilibrium (sensation of falling or vertigo)

Autonomic function (flushing, sweating, piloerection)

Sensation of unusual, intense odors or sounds .

Some describe odd, internal feelings such as fear, a sense of impending change, detachment, depersonalization, deja vu, or illusions that objects are growing smaller (micropsia) or larger (macropsia). These are referred to as ***auras***.

Focal Seizures with Dyscognitive Features

Focal seizures accompanied by a **transient impairment** of the patient's ability to maintain **normal contact with the environment.**

Inability to respond appropriately to visual or verbal commands during the seizure.

Impaired recollection or awareness of the ictal phase.

The seizures frequently begin with an aura.

The ictal phase starts with a sudden behavioral arrest or motionless stare, which marks the onset of the period of impaired awareness.

The behavioral arrest is usually accompanied by **automatisms** ***(***chewing, lip smacking, swallowing, or picking movements of the hands).

The patient is confused following the seizure, and the transition to full recovery of consciousness range from seconds up to an hour.

The seizure is followed by anterograde amnesia.

Generalized Seizures

Focal seizures spread to involve both cerebral hemispheres and produce a generalized seizure, usually of the tonic-clonic variety.

Types :

Typical Absence Seizures

Atypical Absence Seizures

Generalized, Tonic-Clonic Seizures

Atonic Seizures

Myoclonic Seizures

Typical Absence Seizures

It is characterized by sudden, **brief lapses of consciousness** without loss of postural control.

The seizure typically lasts for only seconds, consciousness returns as suddenly as it was lost,

There is **no postictal confusion**.

Seizures are accompanied by subtle, bilateral motor signs -rapid blinking of the eyelids, chewing movements, or small-amplitude, clonic movements of the hands.

Clinical signs of the seizures are subtle, daydreaming, a decline in school performance.

EEG: generalized, symmetric, 3-Hz spike-and-wave discharge that begins and ends suddenly, superimposed on a normal EEG background.

Atypical Absence Seizures

Features deviate both clinically and electrophysiologically from typical absence seizures.

The lapse of consciousness is usually of longer duration and less abrupt in onset and cessation.

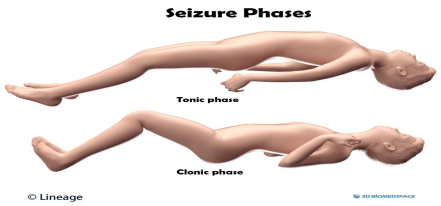
The EEG shows a generalized, slow spike-and wave pattern with a frequency of ≤2.5 per second

Atypical absence seizures are usually associated with diffuse or multifocal structural abnormalities of the brain.

They accompany other signs of neurologic dysfunction such as mental retardation.

The seizures are less responsive to anticonvulsants compared to typical absence seizures.

Generalized, Tonic-Clonic Seizures



The most common seizure in around 10% of all persons with epilepsy.

It results from metabolic derangements and are therefore frequently encountered.

The seizure usually begins abruptly without warning.

The prodrome is distinct from the stereotypic auras associated with focal seizures that generalize.

Tonic Phase

The initial phase of the seizure is usually tonic contraction of muscles throughout the body.

Tonic contraction of the muscles of expiration and the larynx at the onset produces a loud moan or “ictal cry.”

Respirations are impaired.

Secretions pool in the oropharynx, and cyanosis develops.

Contraction of the jaw muscles may cause biting of the tongue.

Marked enhancement of sympathetic tone leads to increases in heart rate, blood pressure, and pupillary size.

The tonic phase lasts for 10–20 secs.

Clonic Phase

The seizure evolves into the clonic phase.

It is produced by the superimposition of periods of muscle relaxation on the tonic muscle contraction.

The periods of relaxation progressively increase until the end of the ictal phase, which usually lasts for 1 min.

The postictal phase is characterized by unresponsiveness, muscular flaccidity, and excessive salivation that can cause stridorous breathing and partial airway obstruction. Bladder or bowel incontinence occur at this point.

Patients gradually regain consciousness over minutes to hours.

There is typically a period of postictal confusion.

Patients subsequently complain of headache, fatigue, and muscle ache that can last for many hours.

The duration of impaired consciousness in the postictal phase can be extremely long and can last for many hours.

EEG Changes during tonic clonic phase

The EEG during the tonic phase of the seizure shows a progressive increase in generalized low-voltage fast activity, followed by generalized high-amplitude, polyspike discharges.

In the clonic phase, the high-amplitude activity is typically interrupted by slow waves to create a spike-and-wave pattern.

The postictal EEG shows diffuse slowing that gradually recovers as the patient awakens.

Atonic Seizures

These are characterized by sudden loss of postural muscle tone lasting 1–2 s.

Consciousness is briefly impaired, but there is usually no postictal confusion.

A brief seizure cause only a quick head drop or nodding movement, whereas a longer seizure will cause the patient to collapse.

The EEG shows brief, generalized spike-and-wave discharges followed immediately by diffuse slow waves that correlate with the loss of muscle tone.

Myoclonic Seizures

It is a sudden and brief muscle contraction that involve one part of the body or the entire body.

The EEG showd bilaterally synchronous spike-and-wave discharges synchronized with the myoclonus.

Myoclonic seizures usually coexist with other forms of generalized seizures

Currently Unclassifiable Seizures

Epileptic spasms : characterized by a briefly sustained flexion or extension of predominantly proximal muscles, including truncal muscles.

These occur predominantly in infants.

EEG shows hypsarrhythmias, which consist of diffuse, giant slow waves with a chaotic background of irregular, multifocal spikes and sharp waves.

Differential Diagnosis

Syncope

Psychological disorders

Metabolic disturbances

Alcoholic blackouts

Psychoactive drugs

Transient ischemic attack (TIA)

Sleep disorders

Movement disorders

Special considerations in children

History And Examination

The diagnosis of a seizure is based solely on clinical grounds—the examination and laboratory studies are often normal.

Careful interview of the witness.

History of febrile seizures, earlier auras or brief seizure, and a family history of seizures.

Epileptogenic factors such as prior head trauma, stroke, tumor, or CNS infection should be identified.

Careful assessment to access the site of lesion.

Lab Studies

Routine blood studies to identify metabolic causes of seizures- electrolytes, glucose, calcium, or magnesium, and hepatic or renal disease.

Lumbar puncture for any suspicion of meningitis or encephalitis.

Testing for autoantibodies in the serum and cerebrospinal fluid.

Electrophysiologic Studies : the presence of electrographic seizure activity during the clinically evident event establishes the diagnosis.

The EEG is used for classifying seizure disorders and aiding in the selection of anticonvulsant medications.

Magnetoencephalography (MEG) : for measuring noninvasively cortical activity of the brain. Instead of measuring electrical activity, it measures the small magnetic fields that are generated by the activity.

Brain Imaging : MRI has been shown to be superior to CT for the detection of cerebral lesions associated with epilepsy.

MRI identifies lesions such as tumors, vascular malformations, or other pathologies that need urgent therapy.

Treatment

Avoidance of precipitating factors.

Treatment of underlying conditions- metabolic disturbances, structural lesions.

Antiepileptic drug therapy - in a patient with recurrent seizures of unknown etiology or a known cause that cannot be reversed.

Drugs : phenytoin, valproic acid, carbamazepine, phenobarbital, etc.

Surgical Treatment – Lobectomy, lesionectomy, hemispherectomy.

Stereotactic radiosurgery, laser thermoablation, and deep brain stimulation are other options for surgical treatment.

Status Epilepticus

Continuous seizures or repetitive, discrete seizures with impaired consciousness in the interictal period.

Subtypes:

Generalized convulsive status epilepticus (GCSE)

Nonconvulsive status epilepticus (NCSE)

Status epilepticus has duration between 15–30 min.

EEG may be the only method of establishing the diagnosis.

Anticonvulsant therapy should then begin without delay.

Generalized convulsive status epilepticus (GCSE)

Characterized by persistent, generalized electrographic seizures, coma, and tonic-clonic movements.

It is an emergency and must be treated immediately, to prevent :

Cardiorespiratory dysfunction

Hyperthermia

Metabolic derangements

Irreversible neuronal injury

Common causes of GCSE are anticonvulsant withdrawal or noncompliance, metabolic disturbances, drug toxicity, CNS infection, CNS tumors, refractory epilepsy, and head trauma.

Nonconvulsive status epilepticus

Characterized by persistent absence seizures or focal seizures with confusion or partially impaired consciousness, and minimal motor abnormalities.

The treatment is less urgent.

The ongoing seizures are not accompanied by the severe metabolic disturbances.

It is associated with cellular injury in the region of the seizure focus.

Chapter 1.6

Neurosis V/S Psychosis

Psychosis is an abnormal condition of the mind that results in difficulties determining what is real and what is not.

Symptoms include false beliefs (delusions) and seeing or hearing things that others do not see or hear (hallucinations).

Other symptoms includes incoherent speech and behavior that is inappropriate for the situation.

There are sleep problems, social withdrawal, lack of motivation, and difficulties carrying out daily activities.

Neurosis is a class of functional mental disorders involving chronic distress but neither delusions nor hallucinations.

There are many different neuroses:

Obsessive–compulsive disorder, obsessive–compulsive personality disorder, impulse control disorder, anxiety disorder, hysteria, and a great variety of phobias.

Neurosis	**Psychosis**
Mild functional neuro-psychical disorders that manifest themselves in specific clinical phenomena in the absence of psychical phenomena.	A severe mental illness characterised by loss of contact with reality and relationship with other people causing social maladaptation.
Doesn't affect personality	Affects personality
The contact with reality is partially lost	The contact with reality is completely lost
Hallucinations and delusions are not present.	Hallucinations and delusions are present.
Lower risk of self-harm	Higher risk of self-harm
Obsessive-compulsive disorders, Somatoform disorders, Depression and Post-traumatic disorders	Schizophrenia and delusional disorders
Causative factor are biological, socio-	Causative factor are genetic, biochemical and environmental.

psychic climate, psychological, pedagogical, and socio-economic. Treatment is usually psychological and medicines can also be prescribed.	Treated by antipsychotic medicines, psychological therapy, social support.

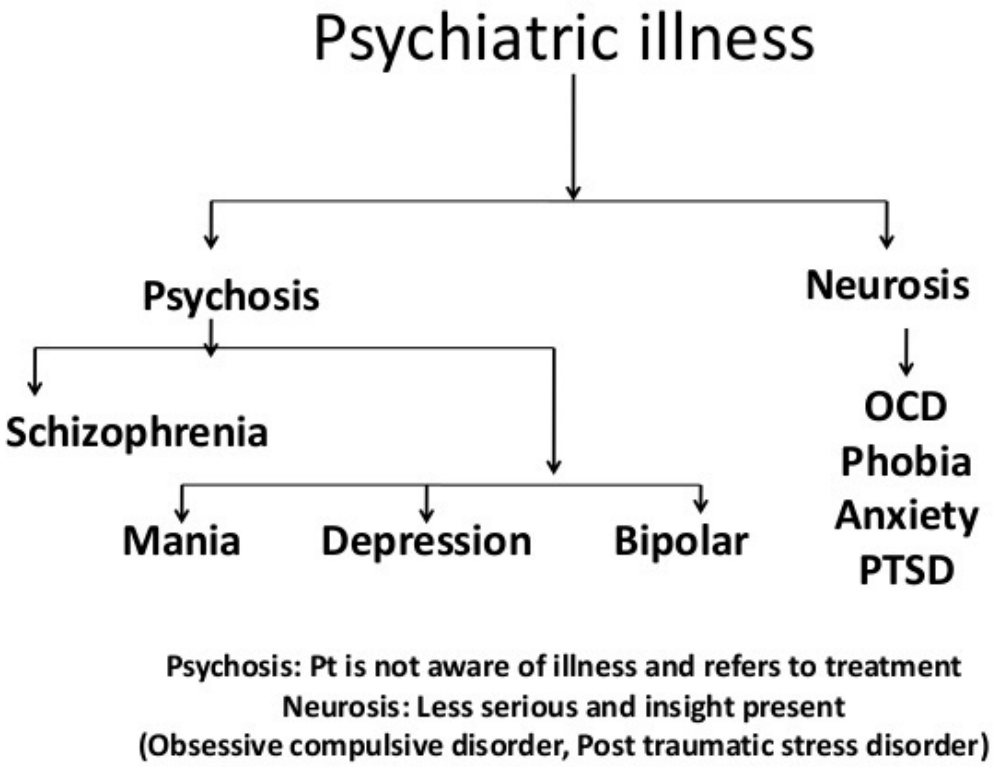


Fig Showing Classification of Psychiatric Illness

Chapter 1.7

Schizophrenia

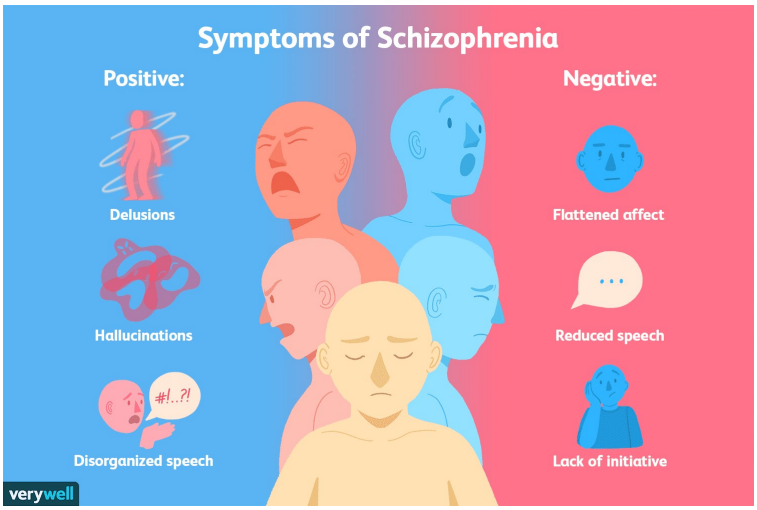


Fig Showing clinical presentation of patient with schizophrenia

Schizophrenia is a psychosis characterised by delusions, hallucinations and lack of insight.

It is a heterogeneous syndrome characterized by perturbations of language, perception, thinking, social activity, affect, and volition.

Occurs in 0.85% of the population worldwide.

The syndrome commonly begins in late adolescence, has an insidious onset, and, often, a poor outcome, progressing from social withdrawal and perceptual distortions to recurrent delusions and hallucinations.

Clinical Menifestations

Positive symptoms: Conceptual disorganization, delusions, or hallucinations.

Negative symptoms: loss of function, anhedonia, decreased emotional expression, impaired concentration, and diminished social engagement.

Disorganized thinking or speech and grossly disorganized motor behavior, including catatonia, may also be present.

Core psychotic features last ≥6 months and include positive symptoms.

Positive: additional which is present in excess

Negative: which should have been present but are actually absent.

Negative symptoms predominate in one-third and are associated with a poor long-term outcome and poor response to treatment.

Prognosis depends not on symptom severity but on the response to antipsychotic medication.

A permanent remission without recurrence does occasionally occur.

About 10% of schizophrenic pts commit suicide.

Comorbid substance abuse is common.

Treatment

Hospitalization is required for acutely psychotic patients dangerous to themselves or others.

Conventional antipsychotic medications are effective against hallucinations, delusions, and thought disorder, risperidone, olanzapine, quetiapine, ziprasidone, aripiprazole, paliperidone,etc.

Educational efforts directed toward families and relevant community resources.

Chapter 1.8

Classification of Psychiatric Disorders

Stress-related disorders

Acute stress disorder

Adjustment disorder

Post-traumatic stress disorder

Anxiety disorders

Generalised anxiety

Phobic anxiety

Panic disorder

Obsessive-compulsive disorder

Affective (mood) disorders

Depressive disorder

Mania and bipolar disorder

Schizophrenia and delusional disorders

Substance misuse disorders

Alcohol

Drugs

Organic disorders

Acute e.g. delirium

Chronic e.g. dementia

Disorders of adult personality and behaviour

Personality disorder

Factitious disorder

Eating disorders

Anorexia nervosa

Bulimia nervosa

Somatoform disorders

Somatisation disorder

Dissociative (conversion) disorder

Pain disorder

Hypochondriasis

Body dysmorphic disorder

Somatoform autonomic dysfunction

Neurasthenia

Puerperal mental disorders

Chapter 1.9

Chronic Fatigue Syndrome

A disorder characterized by persistent and unexplained fatigue resulting in severe impairment in daily functioning.

Concomitant symptoms : pain, cognitive dysfunction, and unrefreshing sleep.

Additional symptoms can include headache, sore throat, tender lymph nodes, muscle aches, joint aches, feverishness, difficulty sleeping, psychiatric problems, allergies, and abdominal cramps.

Diagnostic Criteria

Fatigue lasts for at least 6 months.

Fatigue is of new or definite onset.

Fatigue is not the result of an organic disease or of continuing exertion.

Fatigue is not alleviated by rest.

Fatigue results in a substantial reduction in previous occupational, educational, social, and personal activities.

Four or more of the following symptoms are concurrently present for 6 months:

Impaired memory or concentration,

Sore throat

Tender cervical or axillary lymph nodes.

Muscle pain

Pain in several joints

New headaches

Unrefreshing sleep.

Malaise after exertion.

Exclusion Criteria

Medical condition explaining fatigue.

Major depressive disorder (psychotic features) or bipolar disorder.

Schizophrenia, dementia, or delusional disorder.

Anorexia nervosa, bulimia nervosa.

Alcohol or substance abuse.

Severe obesity (body mass index >40).

Predisposing Factors

Childhood trauma (sexual, physical, emotional abuse, emotional and physical neglect).

Physical inactivity during childhood.

Premorbid psychiatric illness or psychopathology.

Premorbid hyperactivity.

Precipitating Factors

Somatic events : pain, fatigue, emotional distress etc.

Infection (e.g., mononucleosis, Q fever, Lyme disease)

Surgery

Pregnancy

Psychosocial stress

Life events

Perpetuating Factors

Non-acknowledgment by physician

Negative self-efficacy

Strong focus on bodily symptoms

Fear of fatigue

Lack of social support

Low physical activity pattern

Pathophysiology

Exact pathophysiology of CFS is unclear.

It is associated with reduced gray matter volume, which in turn is associated with a decline in physical activity.

Elevations in titers of antinuclear antibodies.

Reductions in immunoglobulin.

Reductions in natural killer cell activity.

Diagnosis

A thorough history, a systematic physical examination is warranted to exclude disorders causing fatigue (e.g., endocrine disorders, neoplasms, heart failure).

Laboratory tests serve primarily to exclude other diagnoses.

Complete blood count, erythrocyte sedimentation rate, C-reactive protein, serum creatinine, electrolytes, calcium, and iron, blood glucose, liver function tests, thyroid stimulating hormone and urinalysis.

Management

Acknowledgement the impact of the patient's symptoms on daily functioning.

Cognitive behavioral therapy (CBT)

Graded exercise therapy (GET)

Treatment for depression and anxiety if present.

Nonsteroidal anti-inflammatory drugs for patients with headache, diffuse pain, and fever.

The patient is encouraged to maintain regular sleep patterns.

To remain as active as possible.

To gradually return to previous levels of exercise and other activity.

Chapter 1.10

Psychiatric disorders

Psychiatric disorders are amongst the most common of human illnesses.

Aetiology

Aetiology is multifactorial, with a combination of biological, psychological and social causes.

Biological factors –

Genetic factors

Brain structure and function

Psychological and behavioural factors

Perceived stress and trauma

Personality

Behaviour

Social and environmental factors

Social isolation

Stressors

Aetiological factors in psychiatric disorders

Predisposing

Increase susceptibility to psychiatric disorder.

Established in utero or in childhood.

Operate throughout patient's lifetime (e.g. genetic factors, congenital defects).

Precipitating

Trigger an episode of illness

Determine its time of onset (e.g. stressful life events, acute physical illness)

Perpetuating

Delay recovery from illness (e.g. lack of social support, chronic physical illness)

Mental State Examination

General appearance and behaviour

Speech

Mood

Thoughts

Abnormal beliefs

Abnormal perceptions

Cognitive function

Cognitive Function

Concentration

Orientation

Intellectual abilities

Memory

Anxiety

A normal and often healthy emotion which can be due to Psychological or Somatic cause.

Psychological symptoms

Apprehension

Irritability

Worry

Fear of impending disaster

Poor concentration

Depersonalisation

Somatic

Palpitations

Fatigue

Tremor

Dizziness

Sweating

Diarrhoea

Frequent desire to pass urine

Chest pain

Initial insomnia

Breathlessness

Headache

Depressed Mood

Depressive disorder is common, The symptoms of depression are both mental and physical.

Psychological

Depressed mood

Reduced self-esteem

Pessimism

Guilt

Loss of interest

Loss of enjoyment (anhedonia)

Suicidal thinking

Somatic

Reduced appetite

Weight change

Disturbed sleep

Fatigue

Loss of libido

Bowel disturbance

Motor retardation (slowing of activity)

Elated Mood

Elation, or euphoria, is the converse of depression and is characteristic of mania.

It may manifest as

Infectious joviality

Over-activity,

Lack of sleep and appetite,

Undue optimism,

Over-talkativeness,

Irritability,

Recklessness in spending and sexual behaviour.

Chapter 1.11

Delusions and Hallucinations

Delusions: Delusions are defined as beliefs that conflict with reality. Despite contrary evidence, individuals with delusions can't let go of their convictions.

Various types of delusion:

Persecutory: such as a conviction that others are out to get one.

Hypochondriacal: such as an unfounded conviction that one has cancer.

Grandiose: such as a belief that one has special powers or status.

The most extreme form of delusion is nihilistic.

Hallucinations: perceptions without external stimuli.

They can occur in any sensory modality. (visual, auditory, olfactory, gustatory, tactile, etc).

Most common are visual or auditory.

The nature of hallucinations is important diagnostically.

Confusion

A vague term used to describe a range of primarily cognitive problems but also includes disturbances in perception, belief and behaviour.

It usually presents as a problem when patients cannot comply with medical care.

Alcohol misuse

Substance misuse

Psychological factors affect medical conditions

Psychological factors influence the presentation, management and outcome of most medical conditions.

Anxiety may present as an increase in somatic symptoms such as breathlessness, tremor or palpitations, or as the avoidance of treatment.

Depression may manifest as increased symptoms such as pain or fatigue and disability.

Chapter 1.12

Medically unexplained somatic symptoms (MUS)

Common examples include:

Pain

Fatigue

Dizziness

Fits

Feelings of weakness.

Treatment

Biological treatments – by modifying brain function. Psychotropic drugs –antipsychotic, antidepressant, mood-stabilising, anti-anxiety

Psychological treatments - These are based on talking with patients, either individually or in groups.

Social interventions

Psychological treatments

General or supportive psychotherapy- Doctor – patient - listening and explaining.

Cognitive therapy - recurring negative thoughts.

Behaviour therapy - to change their behaviour, phobic anxiety.

Cognitive behaviour therapy*:* combines the methods of behaviour therapy and cognitive therapy.

Problem-solving therapy – helps patients actively tackle problems in a structured way.

Interpersonal therapy

Stress-related disorders

Acute stress reaction: Following a stressful event, some people develop a characteristic pattern of transient symptoms, anxiety, anger, depression, altered activity and withdrawal. This stage resolve completely within a few days.

Adjustment disorder: psychological response to the onset or deterioration of a medical illness is a less severe but more prolonged emotional reaction, Grief reactions following bereavement.

Symptoms develop within a month of the onset of the stress.

The predominant symptoms are depression, anxiety insufficiently persistent, anger, aggressive behaviour and excessive alcohol use.

Management

Ongoing contact with and support from a doctor or other who can listen, reassure, explain and advise.

Benzodiazepines and Psychotherapy.

Post-traumatic stress disorder (PTSD)

A protracted response to a stressful event of an exceptionally threatening or catastrophic nature.

Few days to several months between the traumatic event and the onset of symptoms.

Typical symptoms are recurrent intrusive memories (flashbacks) of the trauma, sleep disturbance, nightmares of the traumatic event), avoidance of situations which evoke memories of the trauma.

Anxiety and depression are associated and excessive use of alcohol or drugs.

Management

Immediate counselling and antidepressant medication.

Chapter 1.13

Anxiety disorders

These are characterised by the emotion of anxiety, worrisome thoughts, avoidance behaviour and the somatic symptoms of autonomic arousal.

Phobic anxiety disorder : an abnormal or excessive fear of an object or situation, which leads to avoidance of it. Agoraphobia.

Panic disorder : repeated attacks of severe anxiety, which are not restricted to any particular situation or circumstances. Somatic symptoms such as chest pain, palpitations and paraesthesiae in lips and fingers are common.

Generalised anxiety disorder

Chronic anxiety associated with uncontrollable worry. Somatic symptoms of muscle tension and bowel disturbance

Management

Psychological treatment - Explanation and reassurance, CBT.

Drug treatment: Antidepressants-Benzodiazepines, β-blocker

Obsessive-compulsive disorder

These are characterised by obsessive thoughts, which are recurrent, unwanted and usually provoking anxiety.

Repeated acts performed to relieve feelings of tension.

Management

Antidepressant drugs – clomipramine

CBT

Chapter 1.14

Mood Disorders

Unipolar depression - one or more episodes of low mood and associated symptoms.

Bipolar disorder - episodes of elevated mood interspersed with episodes of depression.

Dysthymia: chronic low-grade depressed mood without sufficient symptoms.

Depression

It is a mood disorder characterized by low mood, a feeling of sadness, and a general loss of interest in things.

Prevalence of 5% in the general population and approximately 10% in chronically ill medical out patients.

A major cause of disability and of suicide .

It magnifies disability, diminishes adherence to medical treatment and rehabilitation, and shorten life expectancy.

Aetiology of Depression

Genetic predisposition

Adversity and emotional deprivation early in life.

Tiggered by stressful life events.

Diagnosis

Symptoms already discussed

Management

Drug treatment - Tricyclic antidepressants, Selective serotonin re-uptake inhibitors

CBT

Bipolar Disorder

Episodic disturbance with interspersed periods of depressed and elevated mood.

Lifetime risk is approximately 1%.

Genetic predisposition.

Bipolar I disorder involves periods of severe mood episodes from mania to depression.

Bipolar II disorder is a milder form of mood elevation, involving milder episodes of hypomania that alternate with periods of severe depression.

Management of Bipolar

Depression – Tricyclic antidepressants, Selective serotonin re-uptake inhibitors

Manic episodes - antipsychotic drugs- lithium, carbamazepine and sodium valproate.

Misuse and Dependence

This can occur due to:

Alcohol

Substance

Sedatives

Stimulants

Hallucinogens

Organic solvent

Chapter 1.15

Personality Disorders

Personality is the set of characteristics and behavioural traits which best describes an individual and their patterns of interaction with the world.

Behavioural traits are enduring tendencies to behave in particular ways.

Diagnosed when an individual's personality causes persistent and severe problems for the person himself or herself or for others.

Psychopathic or antisocial personality describes a persistent pattern of behavior characterized by lack of concern for others.

Personality disorder is classified into 8–10 types depending on the particular behavioural traits.

A patient who meets diagnostic criteria for one subtype commonly meets criteria for two or three others.

Aetiology

They have an inherited aspect but most are more clearly related to an unsatisfactory upbringing and childhood abuse.

Management

Largely untreatable, little benefit from psychotropics, and psychotherapy.

Classification

Eating disorders

Somatoform disorders

Factitious disorders and malingering

Puerperal disorders

Eating Disorders

There are two well-defined eating disorders

Anorexia nervosa

Bulimia nervosa

These share some overlapping features.

99% are females.

There are much higher prevalence of abnormal eating behaviour in the population which does not meet diagnostic criteria of these two conditions.

Obesity is arguably a much greater problem but considered to be more a disorder of lifestyle.

Anorexia Nervosa

Marked weight loss, arising from food avoidance, often in combination with bingeing, purging, excessive exercise, or the use of diuretics and laxatives.

Despite emaciation, patients still feel overweight and are terrified of weight gain.

Anxiety and depressive symptoms are common.

Extreme starvation is associated with a wide range of physiological and pathological bodily changes.

Associated with serious problems are cardiac and skeletal systems.

Aetiology

Unknown but probably includes genetic and environmental factors, including the social pressure on women to be thin.

Diagnosis

Usually emerges in adolescence.

Weight loss of at least 15% of total body weight (or BMI ≤ 17.5)

Avoidance of high-calorie foods

Distortion of body image so that patients regard themselves as fat even when grossly underweight

Amenorrhoea for at least 3 months

Diagnosis is made by the the presence of a pronounced fear of fatness despite being thin, and on the absence of alternative causes of weight loss.

Management

By addressing abnormal beliefs and behaviour.

Psychological treatments by CBT and family therapy.

Psychotropic drugs are of little benefit.

Compulsory admission and refeeding.

Bulimia Nervosa

Patients are near normal weight but display a morbid fear of fatness associated with disordered eating behaviour.

Recurrently embark on eating binges, often followed by corrective measures such as self-induced vomiting.

Begins later in adolescence

Diagnosis

Recurrent bouts of binge eating.

Lack of self-control over eating during binges.

Self-induced vomiting, purgation or dieting after binges.

Weight maintained within normal limits.

Pitted teeth

Calluses on knuckles

Physical complications, including the dental and oesophageal consequences of repeated vomiting.

It causes electrolyte abnormalities, cardiac arrhythmias and renal problems.

Management

CBT

Interpersonal psychotherapy

Somatoform Disorders

These are disorders of somatic symptoms which are not explained by a medical condition and not better diagnosed as part of a depressive or anxiety disorder.

Aetiology:

It is incompletely understood but contributory factors include depression or anxiety.

A family history or previous history of a particular condition shaped the patient's beliefs about illness.

Doctors exacerbate the problem, either by dismissing the complaints.

Somatoform Disorders

Somatisation disorder

Hypochondriacal disorder

Body dysmorphic disorder

Somatoform autonomic dysfunction

Somatoform pain disorder

Neurasthenia (Chronic fatigue syndrome)

Dissociative (conversion) disorder

Somatisation disorder

It is characterised by the occurrence of chronic multiple somatic symptoms for which there is no physical cause.

The symptoms start in early adult life and may be referred to any part of the body.

It is much more common in women.

Common complaints include pain, vomiting, nausea, headache, dizziness, menstrual irregularities and sexual dysfunction.

Patients may undergo a multitude of negative investigations and unhelpful operations.

Hypochondriacal disorder

They have a strong fear or belief that they have a serious, often fatal, disease that persists despite appropriate medical reassurance.

Antipsychotic medication

CBT

Body dysmorphic disorder

They have belief that their body is disfigured in some way.

People with this condition make inappropriate requests for cosmetic surgery. CBT and antipsychotic medication.

Somatoform autonomic dysfunction

These are somatic symptoms referable to bodily organs which are largely under the control of the autonomic nervous system-cardiac neurosis, psychogenic hyperventilation, psychogenic vomiting etc.

Somatoform pain disorder

Disorders with severe, persistent pain which cannot be explained by a medical condition.

CBT and multidisciplinary pain management.

Neurasthenia (Chronic fatigue syndrome)

It is characterised by excessive fatigue after minimal physical or mental exertion, poor concentration, dizziness, muscular aches and sleep disturbance.

Symptoms overlap those of depression and anxiety.

Graded exercise and CBT.

Dissociative (conversion) disorder

It is characterised by a loss or distortion of neurological function not fully explained by organic disease.

The most common symptoms mimic lesions in the motor or sensory nervous system

Dissociative disorders involves psychological functions, especially of memory and general intelligence.

The aetiology is unknown.

There is an association with adverse childhood experiences, including physical and sexual abuse.

Symptoms includes

Gait disturbance, Loss of function in limbs, Aphonia, Non-epileptic seizures, Sensory loss and Blindness.

Treated with CBT or antidepressant drugs.

General management of patients with medically unexplained symptoms

Reassurance

Explanation

Advice

Drug treatment

Psychological treatment

Rehabilitation

Shared care with the GP

Factitious disorders and Malingering

Factitious disorder

Repeated and deliberate production of the signs or symptoms of disease, apparently to obtain medical care.

Presents in young women who work in paramedical professions

These are usually medical but may relate to a psychiatric illness, with reports of hallucinations or depressive illness.

Münchausen's syndrome

Severe chronic form of factitious disorder.

Male of old age, with a solitary, peripatetic lifestyle.

Patient travel widely, visiting several hospitals in one day to obtain medical care.

The history can be convincing enough to persuade doctors to undertake investigations or initiate treatment, including exploratory surgery.

They present similarly elsewhere, often changing name several times.

Management by firm confrontation with clear evidence of the fabrication of illness, with psychological support.

Malingering

It is a behaviour, not a psychiatric diagnosis.

It refers to the conscious simulation of signs of disease and disability.

The motives are clear to the patient but they conceal from doctors.

Motives can be avoidance of burdensome responsibilities, pursuit of financial gain,etc.

Malingering is hard to detect at clinical assessment, but suggested by inconsistency in the history.

Puerperal Disorders

Following childbirth:

Three common psychiatric complications of childbirth.

Post-partum blues

Post-partum depression

Puerperal psychosis

Post-partum blues - begin soon after childbirth.

It is characterised by irritability, labile mood and tearfulness.

The symptoms peak on about the fourth day and then resolve.

No treatment is required.

Reassurance to mother.

Post-partum depression :

Occurs in 10–15% women.

Common in women with a previous history of depression.

Explanation and reassurance.

Psychological and drug treatments for depression should be considered.

Hospital admission to a mother and baby unit.

Puerperal psychosis

Onset in the first 2 weeks after childbirth.

Serious complication form of a manic or depressive psychosis.

Schizophrenic psychosis can also occur.

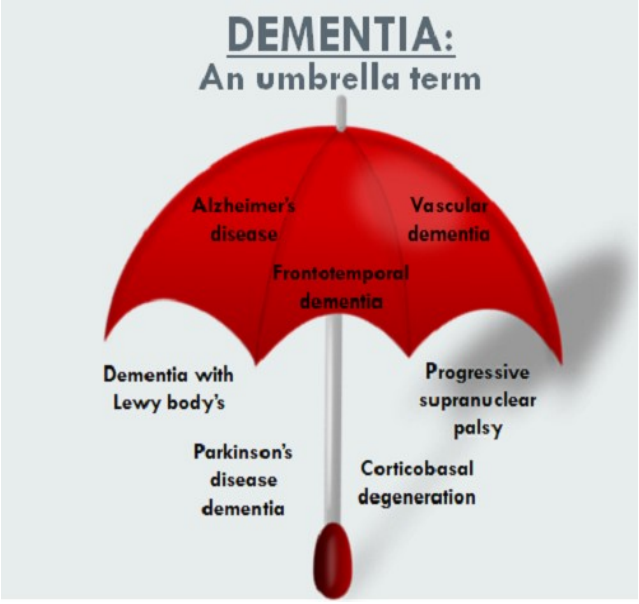
Management depends on the type of psychosis.

It is important to consider the baby, and especially so to establish whether the mother has ideas of harming it.

Admission to a psychiatric mother and baby unit.

Chapter 1.16

Dementia



A Degenerative disorder of nervous system.

It is caused by the degeneration of the cerebral cortex without an identifiable external cause.

Genetic factors play an important role.

It commonly affects up to 5% of the population over the age of 65 years.

It is characterised by a loss of previously acquired intellectual function in the absence of impairment of arousal.

Alzheimer's disease and diffuse vascular disease are the most common causes.

Dementia can be rapidly progressive or slowly progressive.

These are often divided into cortical and subcortical types.

It is usually chronic and progressive.

Causes of Demetia

Vascular

Degenerative: Alzheimer's disease

Neoplasm

Inflammation

Trauma

Hydrocephalus

Toxic/nutritional

Infection

Clinical Features

It is an acquired deterioration in cognitive ability that impairs the successful performance of activities of daily living.

It also affects language, visuospatial ability, calculation, judgment, and problem solving.

Neuropsychiatric and social deficits depression, withdrawal, hallucinations, delusions, agitation, insomnia, and disinhibition develops.

Diagnosis

Mini-Mental State Examination (MMSE)

The Montreal Cognitive Assessment (MOCA)

The Cognistat are useful screening tests.

Imaging of head (CT and/or MRI)

EEG

Blood tests

Chest X-ray

Management

Removing correctable causes.

Providing support for patient.

Anticholinesterases

Homoeopathic

Baryta Carb

Natrum Suph

Nux Vomica

Syphilinum

Chapter 1.17

Alzheimer's Disease

Most common cause of dementia.

AD can manifest as young as the third decade, but it is the most common cause of dementia in the elderly.

Genetic factors play an important role.

15% of cases are familial.

These fall into two main groups:

Early-onset disease with autosomal dominant inheritance.

Later-onset group whose inheritance is polygenic.

Pathophysiology

Macroscopically, the brain is atrophic, particularly the cerebral cortex and hippocampus.

Microscopically neuritic plaques composed in part of Aβ amyloid, derived from amyloid precursor protein (APP), neurofibrillary tangles composed of abnormally phosphorylated tau protein.

The apolipoprotein E (apoE) ε4 allele accelerates age of onset of AD and is associated with sporadic and late-onset familial cases.

Clinical Features

Patients most often present with an insidious loss of episodic memory followed by a slowly progressive dementia that evolves over years.

The cognitive changes begins with memory impairment and progressing to language and visuospatial deficits.

20% of patients presents with non memory complaints such as word-finding, organizational, or navigational difficulty.

There are different stages in the presentation of Alzheimer's disease.

In the early stages of typical amnestic AD, the memory loss may go unrecognized or be ascribed to benign forgetfulness of aging.

The cognitive problems begin to interfere with daily activities, such as keeping track of finances, following instructions on the job, driving, shopping, and housekeeping.

In the middle stages of AD, the patient is unable to work, is easily lost and confused, and requires daily supervision. Language becomes impaired—first naming, then comprehension, and finally fluency.

Apraxia emerges, trouble performing learned sequential motor tasks. Visuospatial deficits begin to interfere with dressing, eating, or even walking, and patients fail to solve simple puzzles or copy geometric figures. Simple calculations become difficult.

In the late stages, some persons remain ambulatory, wandering aimlessly. There is loss of judgment and reasoning. Delusions are common.

In the end stages, AD patients become rigid, mute, incontinent, and bedridden, and help is needed with eating, dressing, and toileting.

Investigation

Brain imaging reveals atrophy that begins in the medial temporal lobes before spreading to lateral and medial parietal and temporal lobes and lateral frontal cortex.

Rest imaging are same as mentioned in dementia.

Treatment

AD cannot be cured, and no highly effective drug exists.

The use of cholinesterase inhibitor drugs, symptomatic management of behavioral problems and building support with the patient and family members.

Drugs : Donepezil, rivastigmine, galantamine, and memantine

Depression, respond to antidepressants.

Control of agitation by antipsychotic medications.

Notebooks and posted daily reminders are useful.

Chapter 1.18

Parkinson's disease

Fig: Showing symptoms of parkinsons disease

A neurodegenerative condition which affects the basal ganglia and presents with differing combinations of slowness of movement.

Parkinsonism is a general term used to define a syndrome manifest as Bradykinesia (slowness of voluntary movements)

Rigidity (Cog wheel, Pipe stem)

Tremor

Age of onset is about 60 years.

Pathophysiology

In most cases, cause is unknown.

Degeneration of pigmented dopaminergic neurons of the substantia nigra.

Accumulation of cytoplasmic intraneural inclusion granules (Lewy bodies).

Mutations in glucocerebrosidase, LRRK2, α-synuclein or parkin genes.

Clinical Features

A classical triad of **tremor, rigidity and bradykinesia**.

General symptoms :

Tiredness

Aching limbs

Mental slowness

Depression

Small handwriting.

Bradykinesia:

Fixed expressionless face with reduced frequency of blinking,

Hypophonic voice,

Drooling,

Impaired rapid alternating movements,

Reduced arm swing,

Flexed “stooped” posture with walking,

Difficulty initiating or stopping walking,

En-bloc turning (multiple small steps required to turn), retropulsion (tendency to fall backwards).

Tremors : Tremor consists of alternating contractions of agonist and antagonist muscles in an oscillating, rhythmic manner.

Tremor (“**pill rolling**” of hands) at rest.

Tremor confined to one limb or one side of body is common.

Rigidity :

Cogwheel in upper limbs

Leadpipe in lower limbs

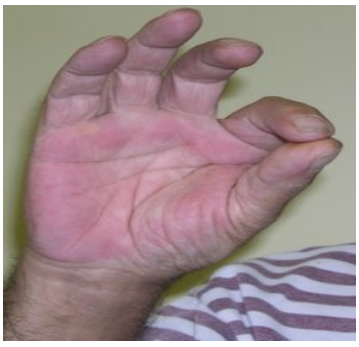


Fig showing rolling movement

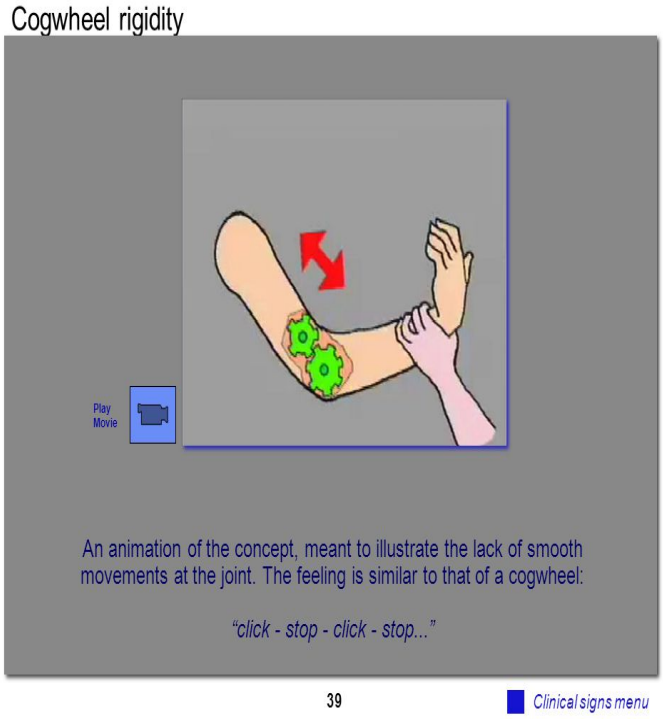


Fig Showing Cogwheel rigidity

Others symptoms:

Depression and anxiety

Cognitive impairment,

Sleep disturbances,

Loss of smell (*anosmia),*

Disturbances of autonomic function.

Investigations

Normal muscular strength, deep tendon reflexes, and sensory examination.

Diagnosis based on history and neuroimaging (CT imaging).

Treatment

To maintain function and avoid drug-induced complications.

Pharmacological : Levodopa, Dopamine agonists.

Anticholinergics :trihexyphenidyl, benztropine.

Surgical treatment: Steriotactic thalamotomy.

Chapter 1.19

Homoeopathic Management

Aconite *:* Pains appear suddenly and are worse on the left side. accompanied by restlessness and an anxious. Pains are intense accompanied by anguish. Pains after exposure to or worsened by dry cold winds.

Anacardium orientale: Fixed ideas. Hallucinations; *thinks he is possessed of two persons or wills*. Anxiety when walking, as if pursued.

Arsenicum album : Pains drawing or burning, as if from needles. Ameliorated by warm applications and aggravated by cold air, worse after midnight. The person is anxious, restless, and thirsty.

Artemisia Vulgaris : convulsive diseases of childhood and girls at puberty. Petit mal. Epilepsy without aura; after fright and other violent emotions and after masturbation. Several convulsions close together. Head drawn back by spasmodic twitchings. Mouth drawn to left. Spasms during menses.

Belladonna : Pains are right-sided with a red face. Muscles of the face twitch with pains, which are severe and intense. Pains are worse with drafts and motion, especially when chewing. Pains appear and disappear rapidly and may worsen at 3 p.m.

Bryonia : Pains are worse with the slightest motion, so the person cannot speak or eat. They are ameliorated by cold and pressure and lying on the painful side. The person has dry mouth and lips, and is likely very thirsty.

Bufo Rana: Convulsive seizures occur during sleep at night.

Cicuta virosa : bending of the head, neck, and spine backwards, with frightful distortions.

Colocynthis : Pains tearing or stitching, mostly on the left side. Pains better with pressure, rest, and warm applications. Pain may be worse around the eye. Attacks worse in evening around 10 p.m.

Cuprum Met: convulsions, beginning in fingers and toes, violent, contractive, tonic and clonic spasms, epileptic attacks. Jerking, twitching of muscles.

Hyoscyamus : Tremulous weakness and twitching of tendons. Subsultus tendinum. Muscular twitchings, spasmodic affections, generally with delirium. Picking at bed-clothes.

Pulsatilla : Pains worse on the right side, drawing or tearing sensation. worse from chewing, warmth, and lying on the painful side. The mouth is dry, but the person does not drink. Pains are ameliorated by cold applications and open air.

Spigelia : Left-sided pains, worse from stooping or moving the head, from noise, during perspiration, from cold water and air, from chewing. Lightning-like pains come on from morning until sunset. The person has a flushed red face on the affected side, and pains come on at certain times of day.

Stramonium: Convulsions of upper extremities and of isolated groups of muscles. Chorea; spasms partial, constantly changing.

Thuja Occidentalis: Fixed ideas, as if a strange person were at his side; as if soul and body were separated; as if something alive in abdomen (Croc). Emotional sensitiveness; music causes weeping and trembling.

Verbascum : Left-sided pains with a pinching sensation as if parts were being crushed by tongs. Pains brought on by clenching teeth, change of temperature, pressure, open air, and motion. Pains involve the chin and lower jaw.

Diseases of Locomotor System

Chapter 2.1

Introduction to Locomotor System

The human musculoskeletal system/locomotor system is an organ system that gives the ability to move using their muscular and skeletal systems.

It is constituted of bones of the skeleton, muscles, cartilage, tendons, ligaments, joints, and other connective tissue that supports and binds tissues and organs together.

It provides form, support, stability, and movement to the body.

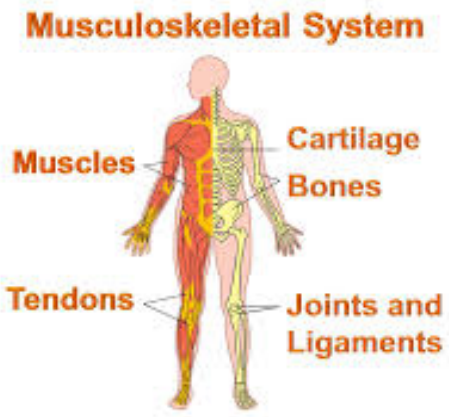


Fig Showing Musculoskeletal System

Functions of Musculoskeletal System

Movement

Haematopoesis

Reservoir for Ca and P Homeostasis

Manifestations of Musculoskeletal Disorders

Pain

Impairment of locomotor function

Pain arises from

Muscles

Tendons

Periarticular structures.

ALGORITHM FOR MUSCULOSKELETAL COMPLAINTS

Musculoskeletal Complaint

Initial rheumatic history and physical examination to determine

1. Is it articular?
2. Is it acute or chronic?
3. Is inflammation present?
4. How many/which joints are involved?

Non Articular Condition

Trauma/fracture

Fibromyalgia

Polymyalgia rheumatica

Bursitis

Tendinitis

If Articular and Complaint < 6 Week

Acute

Acute arthritis

Infectious arthritis

Gout

Pseudogout

Reactive arthritis

Initial presentation of chronic arthritis

If Articular, Complaint > 6 Weeks

Chronic

Is inflammation present?

Is there prolonged morning stiffness?

Is there soft tissue swelling?

Are there systemic symptoms?

Is the ESR or CRP elevated?

If No

Chronic Noninflammatory arthritis.

Are DIP, CMC1, hip or knee joints involved?

If Yes – Osteoarthritis

If No – Osteonecrosis , Charcot arthritis

Chronic

Is inflammation present?

1. Is there prolonged morning stiffness?

2. Is there soft tissue swelling?

3. Are there systemic symptoms?

4. Is the ESR or CRP elevated?

Inflammation Present

Chronic inflammatory arthritis

Ask for

How many joints involved?

1– 3 - Chronic inflammatory mono/oligoarthritis

>3 - Chronic inflammatory polyarthritis

Chronic Inflammatory Mono/Oligoarthritis

Consider

Indolent infection

Psoriatic arthritis

Reactive arthritis

Pauciarticular JIA (Juvenile Idiopathic Arthritis)

Chronic inflammatory polyarthritis

Is involvement symmetric?

No - Consider

Psoriatic arthritis

Reactive arthritis

If **Yes** : Ask if PIP, MCP, or MTP joints involved?

Chronic inflammatory polyarthritis/Symmetric involvement

Are PIP, MCP, or MTP joints involved ?

Yes : Rheumatoid arthritis

No : Consider

SLE

Scleroderma

Polymyositis

Chapter 2.2

Osteoarthritis

It is the most common form of arthritis.

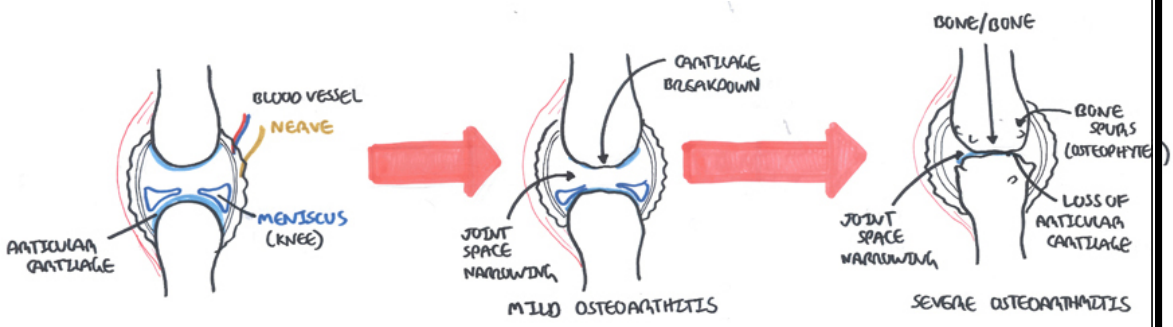
It is a disorder characterized by progressive joint failure in which all structures of the joint have undergone pathologic change.

It is a condition of **synovial joints** characterised by :

Focal loss of articular hyaline cartilage

Proliferation of new bone

Remodelling of joint contour.



It belongs to **Chronic Noninflammatory arthritis** with involvement of DIP, CMC, hip or knee joints.

It targets only certain small and large joints of the body.

The knee and hip are the principal large joints involved.

Epidemiology

Prevalence :

Uncommon in adults under age 40

Highly prevalent over age 60.

More common in women than in men.

80% of people have radiographic evidence

Only 25–30% are symptomatic.

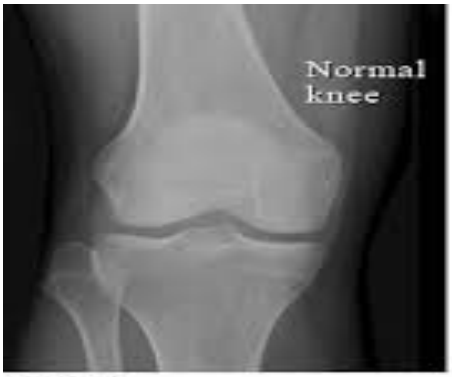


Figure 1

Figure 2

Fig Showing changes in osteoarthritis

Predisposing Factors

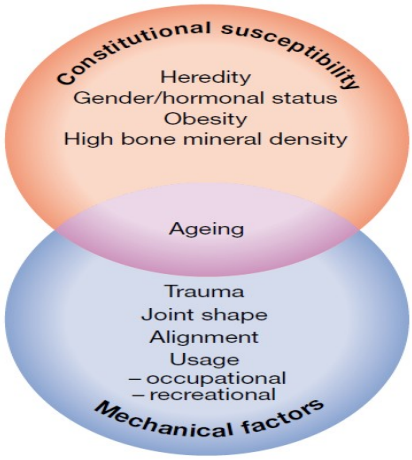


Fig Showing Predisposing factors in osteoarthritis

Aetiology & Pathogenesis

There are numerous pathways that lead to OA, but the initial step is often joint injury in the setting of a failure of protective mechanisms.

Various Factors mechanical, metabolic, genetic or constitutional damages a synovial joint and trigger the need for repair.

The process involves production of new tissue and remodelling of joint shape.

Pathogenesis

This slow but efficient process compensates for the insults, resulting in an anatomically altered but pain-free functioning joint - compensated OA.

Insult is an event which causes damage to a tissue or organ.

After overwhelming insult or a defective repair response, the system fails, resulting in progressive tissue damage, more frequent association with symptoms, and presentation as joint failure – OA.

Changes in Osteoarthritis

Changes occurs at following levels

Cartilage

Bone

Other changes

Cartilage Change

The earliest changes of OA begins in cartilage

Enzymatic degradation takes place in the two major components of cartilage:

Type 2 collagen - provides tensile strength

Aggrecan - provides hydrated gel structure that endows the cartilage with load-bearing properties.

OA cartilage is characterized by gradual depletion of aggrecan, unfurling of the collagen matrix, and loss of type 2 collagen, which leads to increased vulnerability.

Fissuring of the cartilage surface occurs, leading to the development of deep vertical clefts, localised chondrocyte death and decreased cartilage thickness.

Cartilage loss is focal restricted to the maximum load-bearing part of the joint.

These changes in cartilage encourage deposition of calcium pyrophosphate and basic calcium phosphate crystals.

Bony Changes

The subchondral bone shows a mixture of osteolysis and osteosclerosis.

Subchondral cysts develops as a result of small areas of osteonecrosis.

New fibrocartilage at the joint margin undergoes endochondral ossification to form osteophytes.

Severe cartilage loss leads to attrition of bone.

The wear ablate the trabeculae and lead to a smooth, shiny surface with deep linear grooves.

Bone remodelling and cartilage thinning slowly alters the shape of the OA joint, increasing its surface area.

Other Changes

The synovium undergoes variable degrees of hyperplasia.

Osteochondral bodies occurs within the synovium, reflecting chondroid metaplasia or secondary uptake and growth of damaged cartilage fragments.

The outer capsule thickens and contracts, retaining the stability of the remodelling joint.

The muscles acting over the joint shows a non-specific type II fibre atrophy.

Clinical Features

The complaints usually starts after the age of 40 yrs (often > 60)

The onset is insidious over months or years only one or a few joints painful in the beginning.

The main presenting symptoms are pain and functional restriction.

Joint pain from OA is activity-related. Pain comes on either during or just after joint use and then gradually resolves.

Examples include knee or hip pain with going up or down stairs, pain in weight-bearing joints when walking, and, for hand OA, pain when cooking.

The pain is mainly related to movement and weight-bearing, and is relieved by rest.

The joints commonly involved are the knee, hip, spine, and hands. In hands distal interphalangeal (DIP), proximal interphalangeal (PIP), or first carpometacarpal (thumb base) is usually involved.

Initially the pain is worse after over activity of the affected joint and is better by rest.

Later on the pain becomes continuous and is **worse at night**.

Stiffness of the affected joint is prominent, but morning stiffness is usually brief (<30 min).

Clinical signs involves restricted movement of the joint due to capsular thickening, or blocking by osteophyte.

Joint crepitation/crackling due to rough articular surfaces.

Clinical Signs

Bony swelling osteophyte around joint margins.

Joint deformity is usually without instability.

Joint-line or periarticular tenderness.

Muscle weakness, wasting.

No or only mild synovitis.

Nodal Generalized Osteoarthritis

It is seen typically in middle aged women between 40 and 50 years of age who develop **pain, stiffness and swelling** of one or a few finger interphalangeal joints (IPJs).

Gradually, over months, more finger IPJs (distal > proximal) are affected.

The posterolateral swellings on each side of the extensor tendon slowly enlarges and harden to become **Heberden's** (distal IPJ) and **Bouchard's** (proximal IPJ) nodes.

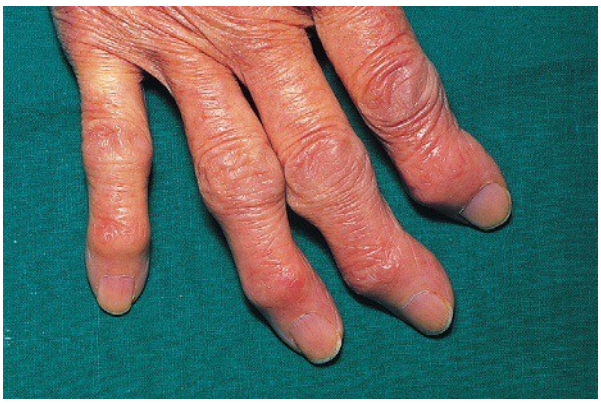


Fig: Showing Nodal osteoarthritis

Affected IPJs often show characteristic lateral deviation, reflecting the asymmetric focal cartilage loss.

Involvement of the first carpometacarpal joint is also common.

Marked osteophyte and subluxation may result in thumb-base squaring of first carpometacarpal joint.

People with nodal OA are at increased risk of OA at other sites.

It has a very strong genetic predisposition.

Knee Osteoarthritis

It targets the patello-femoral and medial tibio-femoral compartments of the knee.

It is common in women and often bilaterally symmetrical.

Trauma is the most common cause.

The pain is worse going up and down stairs or inclines.

Local examination reveals a jerky, asymmetric (antalgic) gait.

A varus, less commonly valgus orfixed flexion deformity.

Local Examination – Osteoarthritis

Weakness and wasting of the quadriceps muscle

Restricted flexion/extension with coarse crepitus

Bony swelling around the joint line.

Deposition of Calcium pyrophosphate dihydrate (CPPD) crystal in association with OA is most common in knee joint.

The deposition of CPPD crystals result in a more overt inflammatory component characterised by stiffness, and effusions and superadded acute attacks of synovitis called pseudogout.

Hip Osteoarthritis

It most commonly targets the superior aspect of the joint.

Such superior pole OA is often unilateral a presentation, often progresses with superolateral migration of the femoral head, and has a poor prognosis.

The less common central (medial) OA shows more central cartilage loss and is largely confined to women. often bilateral at presentation.

The hip shows the best correlation between symptoms and radiographic change.

Hip pain is usually maximal deep in the anterior groin, with variable radiation to the buttock, anterolateral thigh, knee or shin.

Lateral hip pain, worse on lying on that side with tenderness over the greater trochanter. This suggests secondary trochanteric bursitis.

Restricted hip abduction in women may cause pain on intercourse.

Examination Reveals

An antalgic gait

Weakness and wasting of quadriceps and gluteal muscles.

Pain and restriction of internal rotation with the hip flexed.

Anterior groin tenderness just lateral to the femoral pulse.

Fixed flexion, external rotation deformity of the hip.

Ipsilateral leg shortening with severe joint attrition.

Early Onset Osteoarthritis

The typical symptoms and signs of OA may present before the age of 45.

A single joint is affected and there is a clear history of previous trauma.

In Early onset OA multiple joints are not involved.

Commonly affected are patients with endemic OA, due to unknown environmental cartilage toxins.

Erosive Osteoarthritis

This is a rare disease, patients with IPJ OA who have a more prolonged symptom phase.

More overt IPJ inflammation, more disability and worse outcome than those with nodal OA.

Distinguishing features from nodal OA include preferential targeting of proximal IPJs, common development of IPJ lateral instability, subchondral erosions on X-rays, occasional eventual ankylosis of IPJs and lack of association with OA elsewhere.

Physical Examination

Chronic monarthritis or asymmetric oligo/polyarthritis

Bony swellings of the joint margins, e.g., Heberden's nodes or Bouchard's nodes.

Mild synovitis with a cool effusion.

Crepitance—audible creaking or crackling of joint on passive or active movement.

Deformity, e.g., OA of knee may result in varus or valgus deformities.

Restriction of movement, e.g., limitation of internal rotation of hip

Investigations

No blood tests are routinely indicated for workup of patients with OA unless symptoms and signs suggest inflammatory arthritis.

Examination of the synovial fluid is often more helpful diagnostically than an x-ray.

Joint fluid is straw-colored with good viscosity; fluid WBCs <1000/μL; of value in ruling out crystal-induced arthritis, inflammatory arthritis, or infection.

The FBC, ESR and CRP are normal in OA.

CPPD and basic calcium phosphate may also be identified.

Radiographs may be normal at first but as disease progresses may show joint space narrowing, subchondral bone sclerosis, subchondral cysts, and osteophytes.

Plain X-ray may show one or more of the typical features of OA

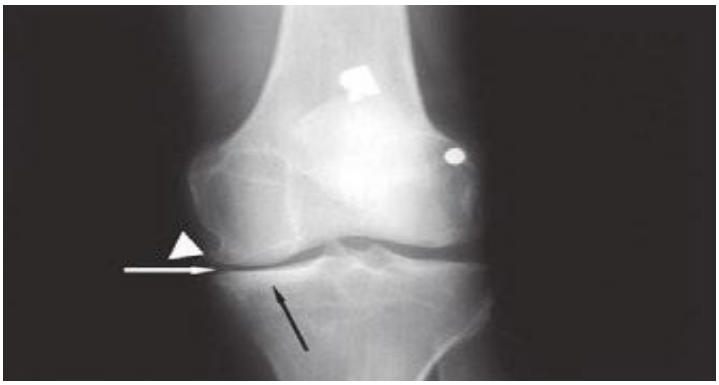
PA view of the pelvis is adequate for assessing hip OA

Standing (stressed) AP radiographs are needed to assess tibiofemoral cartilage loss.

Flexed skyline view is best for assessing patello-femoral narrowing.

Although MRI may reveal the extent of pathology in an osteoarthritic joint, it is not indicated as part of the diagnostic workup.

Radioisotope bone scans performed for other reasons often show, as an incidental finding.



X-ray of knee with medial osteoarthritis. Note the narrowed joint space on medial side of the joint only (*white arrow*),the sclerosis of the bone in the medial compartment providing evidence of cortical thickening (*black arrow*), and the osteophytes in the medial femur (*white wedge*).

Diagnosis

Diagnosis is usually established on basis ofbasis of:

Pattern of joint involvement.

Radiographic features

Normal laboratory tests

Synovial fluid findings

Differential Diagnosis

Inflammatory arthritis is if there is prolonged morning stiffness and many joints are affected.

Bursitis occurs commonly around knees and hips.

Anserine bursitis, medial and distal to the knee, is an extremely common cause of chronic knee pain that may respond to a glucocorticoid injection.

Pain isolated to an area lateral to the hip joint usually reflects the presence of trochanteric bursitis.

Synovial fluid white count is >1000/μL, inflammatory arthritis or gout or pseudogout is likely.

Treatment

Treatment goal to alleviate pain and minimize loss of physical function.

Nonpharmacotherapy strategies

Full explanation of the condition, weight reduction, appropriate use of cane and other supports

Isometric exercises to strengthen muscles around affected joint.

Correction of Malalignment frontal plane (varusvalgus) surgically or with bracing.

Treatment - Pharmacological

Topical capsaicin cream may help relieve hand or knee pain.

Acetaminophen—commonly used analgesic.

NSAIDs, COX-2 inhibitors

Opioid analgesics- symptoms inadequately controlled or those who cannot undergo surgery.

Intraarticular glucocorticoids - symptomatic relief but short-lived.

ntraarticular hyaluronan

Treatment - Surgical

Joint replacement surgery

In patients with pain, stiffness and reduced function impact significantly on their quality of life and are refractory to non-surgical core and adjunctive treatments.

Knee Joint -Arthroscopic debridement and lavage.

Arthroscopic meniscectomy is for acute meniscal tears.

High tibial osteotomy.

Total knee or hip arthroplasty – Knee or Hip replacement.

Chapter 2.3

Rheumatoid Arthritis

Rheumatoid arthritis is a chronic inflammatory disease of unknown etiology marked by a symmetric, peripheral polyarthritis.

It is the most common form of chronic inflammatory arthritis often resulting in joint damage and physical disability.

A chronic **multisystem disease** of unknown etiology characterized by **persistent inflammatory synovitis**, usually involving peripheral joints **symmetrically**.

It is characterised by cartilaginous destruction, bony erosions, and joint deformity.

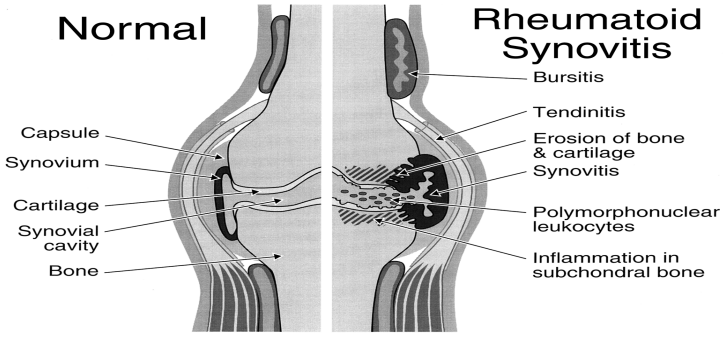


Fig. Showing Changes in Rheumatoid synovitis

Course of Disease – Importance of diagnosis

The clinical course is prolonged, with intermittent exacerbations and remissions.

Patients with RA have an increased mortality when compared with age-matched controls, primarily due to cardiovascular disease.

In RA patients are registered disabled within 3 years, around 80% are moderately to severely disabled within 20 years; and 25% will require a large joint replacement.

Functional capacity decreases most rapidly at the beginning of disease.

Incidence

RA occurs in 0.5–1.0% of the population

Women affected three times more often than men(3:1)

The incidence of RA increases between 25 and 55 years of age after which it plateaus until the age of 75 and then decreases.

Both **genetic** and **environmental factors** may play a role in initiating disease

Association with **HLA-DR4**

Cigarette smoking is a strong risk factor.

It can be triggered by an infectious agent.

Aetiology & Pathophysiology

RA is an autoimmune disease.

It is characterised by persistent cellular activation, autoimmunity and the presence of immune complexes at the site of articular and extraarticular lesions.

This leads to chronic inflammation, granuloma formation and joint destruction.

Earliest signs change is swelling and congestion of synovial membrane and underlying connective tissue.

It is characterised by infiltration of the synovial membrane with lymphocytes, plasma cells and macrophages.

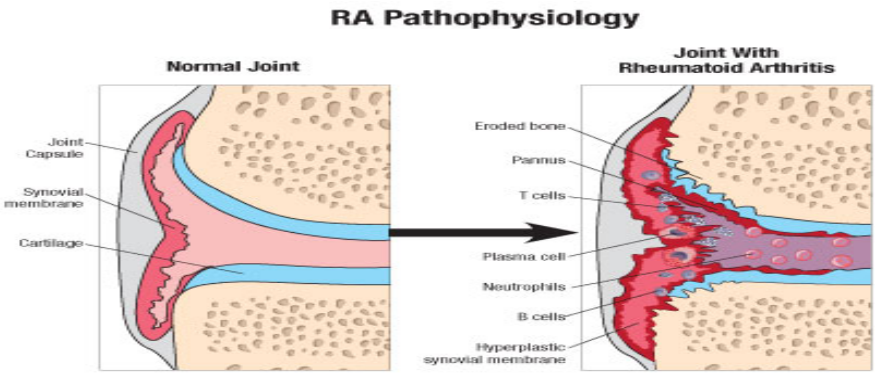
Pathophysiology

Activated T cells stimulate B cells to produce immunoglobulins including RF, and macrophages to produce proinflammatory cytokines.

It acts on endothelium, synovial fibroblasts, bone cells and chondrocytes to promote **swelling and congestion** of the **synovial membrane** and **destruction of bone**, cartilage and soft tissues

The B cells release immunoglobulins, including RF, which can form immune complexes leading to vasculitis.

Lymphoid follicles form within the synovial membrane.



Inflammatory granulation tissue (pannus) spreads over and under the articular cartilage, which is progressively eroded and destroyed.

Fibrous or bony ankylosis can occur.

Muscles adjacent to inflamed joints atrophy and infiltrated with lymphocytes.

Rheumatoid nodules consist of a central area of fibrinoid material surrounded by a palisade of proliferating mononuclear cells.

The extraarticular lesions can occur in the pleura, lung, pericardium and sclera.

Lymph nodes are often hyperplastic, showing many lymphoid follicles.

Immunofluorescence confirms RF antibody synthesis by plasma cells in synovium and lymph nodes.

Difference B/W RA & OA

Hallmarks of RA includes:

Cartilaginous destruction,

Bony erosions,

Joint deformity.

RF antibody, cytokinines

Hallmarks of OA includes:

Focal loss of articular hyaline cartilage

Proliferation of new bone

Remodelling of joint contour.

Clinical Features

The diagnosis can be established by careful history and physical examination.

RA is a **systemic disease** hence the clinical Features can be divided into:

Articular Manifestations

Extraarticular Manifestations

Articular Manifestations – The clinical hallmark of inflammatory joint disease is persistent sinovitis.

The history of symptoms persisting for more than 6 weeks is cut-off to exclude viral arthritis.

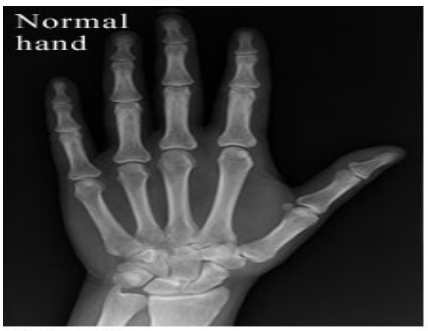


Figure 1

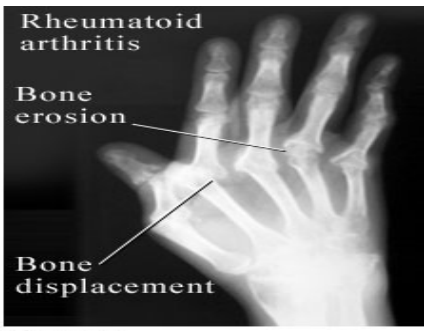


Figure 2

The presenting symptoms results from inflammation of the joints, tendons, and bursae.

Early morning joint stiffness lasting more than 1 hour that eases with physical activity.

Joints involvement: initially small joints of the hands, feet and wrist.

Large joint involvement, and systemic symptoms may also occur.

Symmetric distribution.

The initial pattern is monoarticular, oligoarticular (≤4 joints), or polyarticular (>5 joints).

Criteria for Diagnosis

Morning stiffness (> 1 hr)

Arthritis of three or more joint areas

Rheumatoid factor

Radiological changes

Arthritis of hand joints

Duration ≥ 6 wks

Symmetrical arthritis

Rheumatoid nodules

Sometimes RA has a very acute onset, with morning stiffness, polyarthritis and pitting oedema.

Once the disease process of RA is established, the wrists, metacarpophalangeal (MCP), and proximal interphalangeal (PIP) joints are the most frequently involved joint.

The joints are tender and swollen.

Test positive for serum rheumatoid factor (RF) or anti-CCP(cyclic citrullinated protein**)** antibodies, and have higher scores for physical disability.

Other joints are actively inflamed if they are tender on pressure, and have stress pain on passive movement or effusion/soft tissue swelling.

Calcaneovalgus (eversion) reflecting damage to the ankle and subtalar joint is often associated with loss of the longitudinal arch (flat foot).

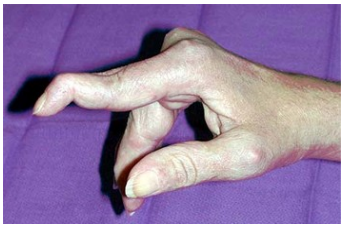
Deformities Rheumatoid Arthritis

Progressive destruction of tendons, joint capsule, and soft tissues may lead to chronic, irreversible deformities.

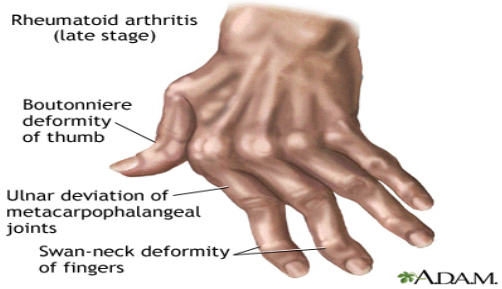
Flexor tendon tenosynovitis is a frequent hallmark of RA and leads to decreased range of motion, reduced grip strength, and "**trigger**" fingers.

Ulnar deviation results from subluxation of the MCP joints, with subluxation of the proximal phalanx to the volar side of the hand.

Swan neck deformity : Hyperextension of the PIP joint with flexion of the DIP joint.



Boutonnière deformity : flexion of the PIP joint with hyperextension of the DIP joint.



Z-line deformity : subluxation of the first MCP joint with hyperextension of the first interphalangeal (IP) joint.

Piano-key movement : Inflammation about the ulnar styloid and tenosynovitis of the extensor carpi ulnaris causes subluxation of the distal ulna.

Flat feet (pes planovalgus) : chronic inflammation of the ankle and midtarsal regions leads to flat feet.

Characteristic deformities develop in foot with long-standing disease.

In the foot, dorsal subluxation of the MTP joints may result in '**cock-up'** toe deformities.

Calcaneovalgus (eversion) reflecting damage to the ankle and subtalar joint is often associated with loss of the longitudinal arch (flat foot).

Extraarticular manifestations

Extraarticular manifestations may develop during the clinical course of RA, even prior to the onset of arthritis.

History of smoking,

Patients have history of early onset of significant physical disability, and test positive for serum RF.

Common menifestations: Subcutaneous nodules, secondary Sjögren's syndrome, pulmonary nodules, and anemia.

Reduced incidence of Felty's syndrome and vasculitis.

Constitutional : weight loss, fever, fatigue, malaise, depression and in severe cases, cachexia.

Nodules : Subcutaneous nodules generally firm; nontender; and adherent to periosteum, tendons, or bursae due to repeated trauma or irritation on forearm, sacral prominences.

Sjögren's Syndrome: presence of keratoconjunctivitis sicca (dry eyes) or xerostomia (dry mouth) in association with another connective tissue disease. It occurs in 10% of patients with RA.

Pulmonary : Most common pulmonary manifestation Pleuritis. It presents with pleuritic chest pain and dyspnea, as well as a pleural friction rub and effusion. Interstitial lung disease (ILD) may also occur with symptoms of dry cough and progressive shortness of breath. Pulmonary nodules may be solitary or multiple.

Cardiac Manifestations : clinical manifestations of pericarditis occur in 10% patients. Cardiomyopathy, another clinically important manifestation. Mitral regurgitation is the most common valvular abnormality in RA.

Vasculitis: Rheumatoid vasculitis typically occurs in patients with long-standing disease, a positive test for serum RF, and hypocomplementemia. The cutaneous signs include petechiae, purpura, digital infarcts, gangrene, and in severe cases large, painful lower extremity ulcerations.

Hematologic: normochromic, normocytic anemia often develops in patients with RA. The degree of anemia parallels the degree of inflammation, correlating with the levels of serum C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR).

Felty's syndrome: clinical triad of neutropenia, splenomegaly, and nodular RA and is seen in less than 1% of patients.

Lymphoma: There is two- to fourfold increased risk of lymphoma in RA patients. Most common type is is a diffuse large B cell lymphoma.

Neurological complications: Peripheral entrapment neuropathies results from compression by hypertrophied synovium or by joint subluxation. Median nerve compression in the carpal tunnel is an early presenting feature.

Cervical cord compression can result from subluxation of the cervical spine at the atlanto-axial joint.

Associated Conditions In addition to extraarticular manifestations, several conditions associated with RA contribute to disease morbidity and mortality rates.

Cardiovascular Disease: incidence of coronary artery disease and carotid atherosclerosis is higher in RA patients. Congestive heart failure occurs at an approximately twofold higher rate in patients with RA.

Associated Conditions

Osteoporosis: Osteoporosis is more common in patients with RA than an age- and sex-matched population, with prevalence rates of 20–30%. Chronic use of glucocorticoids and disability-related immobility also contributes to osteoporosis.

Hypoandrogenism: Men and postmenopausal women with RA have a lower mean serum testosterone, luteinizing hormone, and dehydroepiandrosterone levels than control populations.

Poor Prognosis

Poor prognosis is associated with:

Disability at presentation

Female gender

Involvement of MTP joints

Smoking and

Positive RF and anti-CCP.

Criteria for diagnosis of Rheumatoid Arthritis

Diagnosis of RA is made with four or more of the following:

Morning stiffness (> 1 hr)

Arthritis of three or more joint areas

Rheumatoid factor

Radiological changes

Arthritis of hand joints

Symmetrical arthritis

Duration ≥ 6 weeks

Rheumatoid nodules

Diagnosis of Rheumatoid Arthritis

The diagnosis of RA is based on clinical criteria and there is no single diagnostic test.

Swollen and tender joints in the upper limbs and knees, and combining this with the ESR and the patient's assessment of the general health on a visual analogue scale.

The higher the value, the greater the disease activity.

Investigations and Monitoring

To establish diagnosis

Clinical criteria

Acute phase response (APR)

Serological tests

X-rays

To monitor disease activity and drug efficacy:

Pain (visual analogue scale)

Early morning stiffness (minutes)

Joint tenderness

Joint swelling

DAS 28 score (Disease Activity Score 28)

APR (acute phase reactant)

To monitor disease damage:

X-rays

Functional assessment

To monitor drug safety:

Urinalysis

Biochemistry

Haematology

Investigations

ESR and CRP are usually raised (acute phase response), but may not be in patients with isolated small joint arthritis.

RF is non-specific; low titres are found in about 10% of the normal population

Plain X-rays of the hands, wrist and feet are useful. Osteoporosis is common during the early stages.

Ultrasound and MRI are more sensitive than X-rays at detecting early erosions.

In patients with Baker's cyst, Doppler ultrasound and an arthrogram may be required to establish the diagnosis.

Evaluation

History and physical examination with careful examination of all joints.

Rheumatoid factor (RF) is present in >66% of pts; its presence correlates with severe disease, nodules, extra-articular features.

Antibodies to cyclic citrullinated protein (anti-CCP) have similar sensitivity but higher specificity than RF.

Tendency for developing bone erosions is present most commonly in patients with aggressive disease.

Radiographs: juxta-articular osteopenia, joint space narrowing, marginal erosions.

Differential Diagnosis

Gout

SLE

Psoriatic arthritis

Infectious arthritis

Osteoarthritis

Sarcoid.

Treatment

Patient education on disease and joint protection.

Physical and occupational therapy consider

Assistive devices.

Aspirin or NSAIDs.

Intra-articular glucocorticoids.

Systemic glucocorticoids.

Disease-modifying antirheumatic drugs (DMARDs): e.g., methotrexate, sulfasalazine.

Surgery: in severe functional impairment due to deformity.

Chapter 2.4

Ankylosing Spondylitis

It is an inflammatory disorder of unknown cause that primarily affects the **axial skeleton**; peripheral joints and extraarticular structures.

It is a chronic inflammatory arthritis predominantly affecting the **sacroiliac joints** and spine, which can progress to bony fusion of the spine.

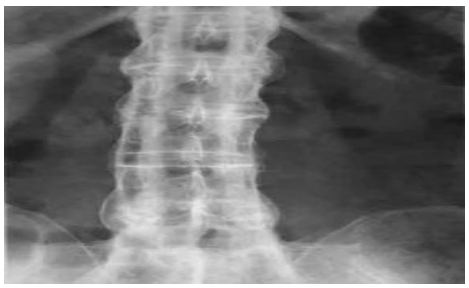


Fig. X Ray Showing Changes in ankylosing spondylosis

It belongs to the family of *spondyloarthritides.*

Spondyloarthritides also includes :

Reactive arthritis

Psoriatic arthritis

Enteropathic arthritis

Juvenile-onset spondyloarthritis

Undifferentiated spondyloarthritis.

Incidence

Ankylosing Spondylitis usually begins in the second or third decade.

Male-to-Female prevalence is between **2:1 and 3:1**.

It is commonly known as *axial spondyloarthritis.*

It has an association with the HLA B27 antigen.

Pathology

Sacroiliitis is often the earliest manifestation.

Knowledge of its pathology comes from both biopsy and autopsy studies.

There is inflammatory granulation tissue in the paravertebral connective tissue at the junction of annulus fibrosus and vertebral bone.

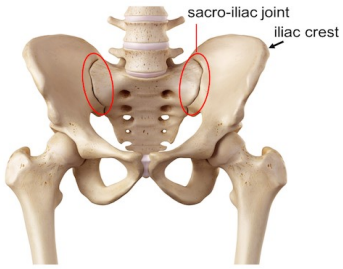


Fig showing the sacro iliac joint and iliac crest

The outer annular fibers are eroded and eventually replaced by bone, forming the beginning of a syndesmophyte.

Syndesmophyte then grows by continued endochondral ossification, ultimately bridging the adjacent vertebral bodies.

Ascending progression of this process leads to the **bamboo spine**.

Other lesions in the spine include :

Diffuse osteoporosis.

Erosion of vertebral bodies at the disk margin, "**squaring**" or "**barreling**" of vertebrae.

Inflammation and destruction of the disk-bone border.

Inflammatory arthritis of the apophyseal (facet) joints is common. Erosion of joint cartilage by pannus is followed by bony ankylosis.

Inflammation in the fibrocartilaginous enthesis.

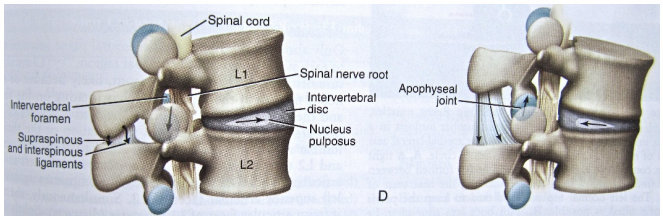


Fig showing Vertebrae

Clinical Manifestations

The course of the disease is **extremely variable**, ranging from the individual with mild stiffness and normal radiographs to the patient with a totally fused spine and severe bilateral hip arthritis, accompanied by severe peripheral arthritis and extraarticular manifestations.

The **constitutional symptoms** includes fever, fatigue, and weight loss.

Then Neurologic complications related to spina fracture or dislocation, atlantoaxial subluxation (can lead to spinal cord compression) and cauda equina syndrome.

The symptoms of the disease are usually first noticed in late adolescence or early adulthood.

The **initial symptom**:

Dull pain, insidious in onset, felt deep in the lower lumbar or gluteal region.

It is accompanied by low-back morning stiffness of up to a few hours duration that improves with activity and returns following inactivity.

Within a few months, the pain becomes persistent and bilateral.

Nocturnal exacerbation of pain often forces the patient to rise and move around.

Bony tenderness at costosternal junctions, spinous processes, iliac crests, greater trochanters, ischial tuberosities, tibial tubercles, and heels may accompany back pain or stiffness.

Hip and shoulder arthritis is considered part of the axial disease.

Severe isolated hip arthritis or bony chest pain may be the presenting complaint.

Arthritis of peripheral joints other than the hips and shoulders are asymmetric.

Extra-articular findings include acute anterior uveitis in up to 40% of patients, aortitis, aortic insufficiency, GI inflammation, cardiac conduction defects, amyloidosis, bilateral upper lobe pulmonary fibrosis.

Extra/juxta-articular pain: due to “enthesitis” i.e inflammation at insertion of tendons and ligaments into bone; frequently affects greater trochanter, iliac crests, ischial tuberosities, tibial tubercles and heels.

Chest pain occurs from involvement of thoracic skeleton and muscular insertions.

Physical Examination

Tenderness over involved joints

Diminished chest expansion

Loss of spinal mobility, with limitation of anterior and lateral flexion and extension of the lumbar spine and of chest expansion.

Pain in the sacroiliac joints may be elicited either with direct pressure or with stress on the joints.

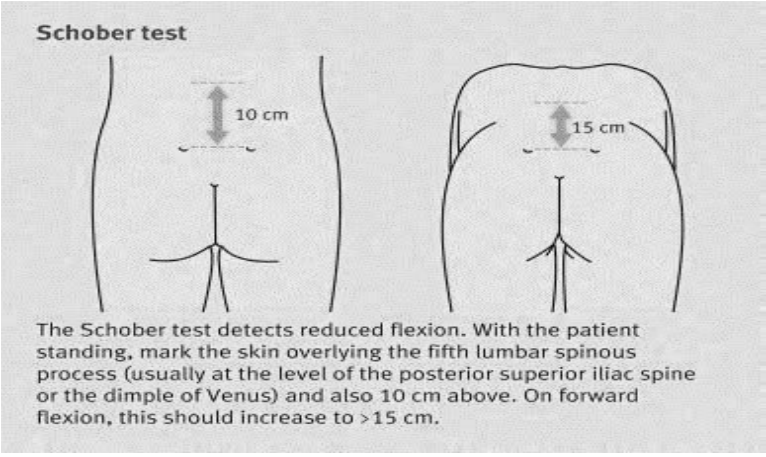
Diminished anterior flexion of lumbar spine (Schober test).

Schober test

It is a useful measure of lumbar spine flexion.

The patient stands erect, with heels together, and marks are made on the spine at the lumbosacral junction and 10 cm above.

The patient then bends forward maximally with knees fully extended, and the distance between the two marks is measured. This distance increases by ≥5 cm in the case of normal mobility and by <4 cm in the case of decreased mobility.



The Schober test detects reduced flexion. With the patient standing, mark the skin overlying the fifth lumbar spinous process (usually at the level of the posterior superior iliac spine or the dimple of Venus) and also 10 cm above. On forward flexion, this should increase to >15 cm.

Evaluation

ESR and C-reactive protein elevated.

Mild anemia.

Rheumatoid factor and ANA negative.

HLA-B27 is helpful in patients with inflammatory back symptoms but negative x-rays.

Radiographs: early may be normal. Sacroiliac joints are usually symmetric.Bony erosions with “pseudo widening” followed by fibrosis and ankylosis.

Differential Diagnosis

Osteoarthritis/spondylosis

Degenerative disk disease

Muscular strain

Fibromyalgia

Metabolic, infectious, or malignant causes of back pain.

Diffuse idiopathic skeletal hyperostosis.

Treatment

Exercise program to maintain posture and mobility.

NSAIDs first-line treatment.

TNF-modulatory agents to improve disease activity and reduce bone marrow edema.

Secukinumab, an IL-17A antagonist, has been found to reduce signs and symptoms.

Sulfasalazine, Methotrexate – DMARD's.

Intra-articular glucocorticoids.

Surgery for severely affected or deformed joints.

Chapter 2.5

Reactive Arthritis

It refers to acute nonpurulent arthritis complicating an infection elsewhere in the body.

The term has been used primarily to refer to spondyloarthritides following **enteric** or **urogenital infections.**

The association of acute arthritis with episodes of **diarrhea** or **urethritis** has been recognized.

During World Wars I and II the triad of **arthritis, urethritis, and conjunctivitis**, often with additional mucocutaneous lesions, became widely known.

Pathology

The clinical manifestations are triggered by:

Enteric infection : *Shigella*, *Salmonella*, *Yersinia*, and *Campylobacter* species;

Genital infection with *Chlamydia trachomatis*.

There is also evidence implicating *Clostridium difficile*, certain toxigenic *Escherichia coli*, and possibly other agents.

Synovial histology is similar to that of other SpAs. Enthesitis shows increased vascularity and macrophage infiltration of fibrocartilage.

Incidence

There is association of ReA with HLAB27

Age group is 18 - 40 years.

The gender ratio in ReA following enteric infection is nearly 1:1, whereas venereally acquired ReA occurs mainly in men.

It has become the most common rheumatic diseases in Africans in the wake of the AIDS epidemic.

Clinical Features

These constitute a spectrum that ranges from an isolated, transient monoarthritis or enthesitis to severe multisystem disease.

A careful history elicit evidence of an **antecedent infection** 1–4 weeks before onset of symptoms.

Constitutional symptoms are fatigue, malaise, fever, and weight loss.

The musculoskeletal symptoms are usually acute in onset.

Arthritis **is asymmetric and additive**, with involvement of new joints occurring over a few days to 1–2 weeks.

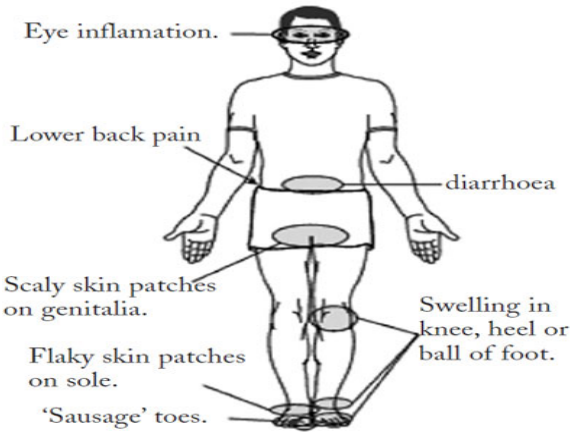


Fig Showing features of reactive arthritis

Joints of the **lower extremities**, especially the knee, ankle, and subtalar, metatarsophalangeal, and toe interphalangeal joints, are commonly involved.

The joints are usually quite painful, and tense with joint effusions.

Dactylitis, or "**sausage digit**" a diffuse swelling of a solitary finger or toe, is a distinctive feature.

Tendinitis and **fasciitis** are characteristic lesions, producing pain at multiple **insertion sites** (enthesis).

Sites : Achilles insertion, the plantar fascia, and sites along the axial skeleton.

Spinal, low-back, and buttock pain are quite common It caused by insertional inflammation, muscle spasm, acute sacroiliitis, orarthritis in intervertebral joints.

Urogenital lesions in **males, urethritis** occurs in both postvenereal and postenteric ReA. **Prostatitis** is also common. **In females**, **cervicitis or salpingitis** may be caused either by the infectious trigger or by the sterile reactive process.

Ocular disease : transient, asymptomatic conjunctivitis to an aggressive anterior uveitis and may result in blindness.

Mucocutaneous lesions :painless lesions on glans penis (circinate balanitis) and oral mucosa, **keratoderma blennorrhagica** cutaneous vesicles that become hyperkeratotic, most common on soles and palms.

Nail changes are common and consist of onycholysis.

Uncommon manifestations: pleuropericarditis, cardiac conduction defects, aortic regurgitation, neurologic manifestations, secondary amyloidosis.

Arthritis typically persists for 3–5 months, but recurrences of the acute syndrome are common.

Work disability or **forced change in occupation** is common in those with persistent joint symptoms.

Chronic heel pain is distressing.

Laboratory Findings

Pursuit of triggering infection by culture, serology, or molecular methods.

Rheumatoid factor and ANA negative.

Mild anemia, leukocytosis, elevated ESR.

HLA-B27 association showing a prevalence <50%. May be helpful in atypical cases and may have prognostic significance.

HIV screening should be performed in all patients.

Polymerase chain reaction (PCR) for chlamydial DNA in first-voided urine specimens may have high sensitivity.

Synovial fluid analysis—often very inflammatory; negative for crystals or infection.

Synovial fluid often contains giant macrophages (Reiter's cells).

Radiographs : In early or mild disease, radiographic changes are absent or confined to juxtaarticular osteoporosis.

Erosions and loss of joint space is seen with new periosteal bone formation, ossification of entheses, sacroiliitis (often unilateral).

Spurs at the insertion of the plantar fascia are common.

The syndesmophytes are nonmarginal; coarse, asymmetric, and "comma"-shaped, arising from the middle of a vertebral body, a pattern less commonly seen in primary AS.

Progression to spinal fusion is uncommon.

Differential Diagnosis

Septic arthritis (gram +/–),

Gonococcal arthritis,

Crystalline arthritis,

Psoriatic arthritis

Treatment

Most patients with ReA benefit from high-dose NSAIDs.

Indomethacin, is the initial treatment of choice.

Prompt antibiotic treatment of acute chlamydial urethritis may prevent subsequent reactive arthritis.

Tendinitis and other enthesitic lesions may benefit from intralesional glucocorticoids.

Skin lesions ordinarily require only symptomatic topical treatment.

Uveitis require therapy with ocular or systemic glucocorticoids.

Sulfasalazine may be beneficial to patients with persistent ReA .

Chapter 2.6

Psoriatic Arthritis

It is a chronic inflammatory arthritis that affects 5–42% of persons with psoriasis.

It refers to an inflammatory musculoskeletal disease that has both **autoimmune** and **autoinflammatory** features occurring in individuals with psoriasis.

It is seronegative.

Often involved the distal interphalangeal (DIP) joints of the fingers and the spine and sacroiliac joints.

It has features similar to those of AS and ReA.

Onset of psoriasis usually precedes development of joint disease.

Nail changes are seen in 90% of patients with psoriatic arthritis.

Six pattern of nail involvement : pitting, horizontal ridging, onycholysis, yellowish discoloration of nail margins, dystrophic hyperkeratosis, and combination of these findings.

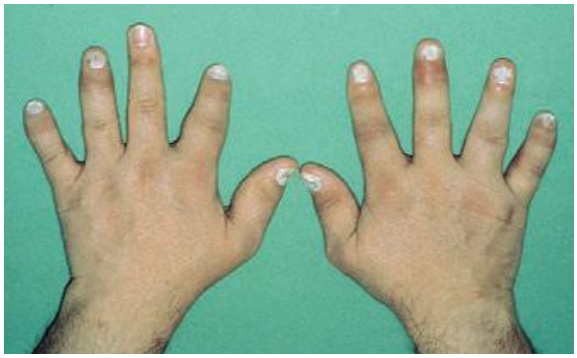


Fig : Psoriatic Nails

Patterns of Joint Involvement

There are five patterns of joint involvement

Asymmetric oligoarthritis: often involves distal interphalangeal/proximal interphalangeal (DIP/PIP) joints of hands and feet, knees, wrists, ankles; "**sausage digits**" may be present, reflecting tendon sheath inflammation.

Symmetric polyarthritis (40%): resembles rheumatoid arthritis except rheumatoid factor is negative, absence of rheumatoid nodules.

Predominantly DIP joint involvement (15%): high frequency of association with psoriatic nail changes.

Arthritis mutilans (3–5%): aggressive, destructive form of arthritis with severe joint deformities and bony dissolution.

Spondylitis and/or sacroileitis: axial involvement is present in 20–40% of pts with psoriatic arthritis; may occur in absence of peripheral arthritis.

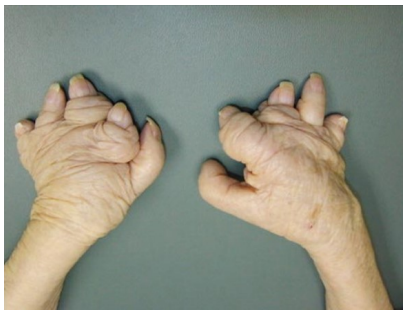


Fig: Arthritis Mutilans

Evaluation

Negative tests for rheumatoid factor.

Hypoproliferative anemia, elevated ESR.

Hyperuricemia may be present.

HIV infection should be suspected in fulminant disease.

Inflammatory synovial fluid and biopsy without specific findings.

Radiographic Findings

- Radiographic features include erosion at joint margin, bony ankylosis, tuft resorption of terminal phalanges, “pencil-in-cup” deformity (bone proliferation at base of distal phalanx with tapering of proximal phalanx), axial skeleton with asymmetric sacroiliitis, asymmetric nonmarginal syndesmophytes.

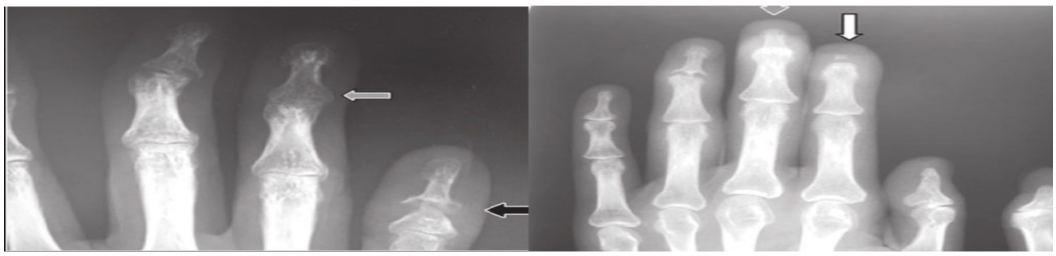


Fig: Showing radiographic features of erosion of join margins

Diagnosis (CASPAR Criteria)

Patient must have inflammatory articular disease (joint, spine, or entheseal) with ≥3 points from any of the following five categories:

Evidence of current psoriasis,a personal history of psoriasis, or a family history of psoriasis.

Typical psoriatic nail dystrophy observed on current physical examination.

A negative test result for rheumatoid factor

Either current dactylitis or a history of dactylitis recorded by a rheumatologist.

Radiographic evidence of juxtaarticular new bone formation in the hand or foot.

Treatment

Coordinated therapy is directed at the skin and joints.

Patient education, physical and occupational therapy.

TNF modulatory agents, infliximab.

NSAIDs.

Intraarticular steroid injections

Efficacy of gold salts and antimalarials

Sulfasalazine

Methotrexate

Leflunomide

Chapter 2.7

Gout

Gout is a form of inflammatory arthritis with high levels of **uric acid** in the blood.

The acid forms needle-like crystals in the joint.

It cause sudden, severe episodes of pain, tenderness, redness, warmth and swelling.

Hyperuricemia : Plasma and extracellular fluids become supersaturated with uric acid.

Normal plasma uric acid : 2.5-6 mg/dl in women, 3.4-7 mg/dl in men.

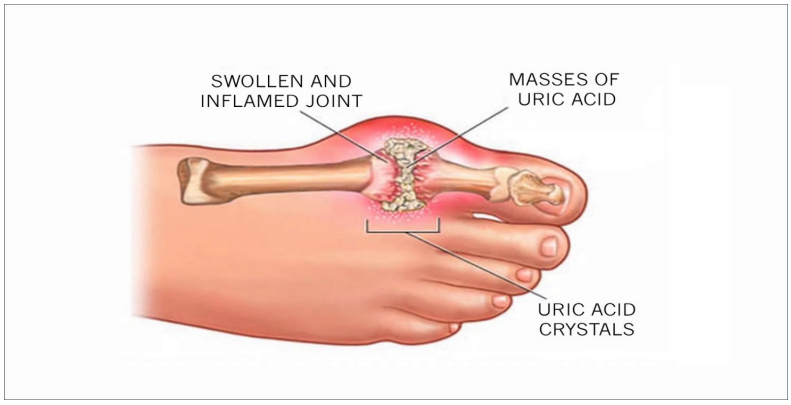


Fig: Showing uric acid crystal deposit in great toe

Gout is a metabolic disorder of purine metabolism.

It is characterized by recurrent attacks of :

Acute arthritis

Chronic deforming arthropathy

Formation of tophi

Systemic complication like renal failure.

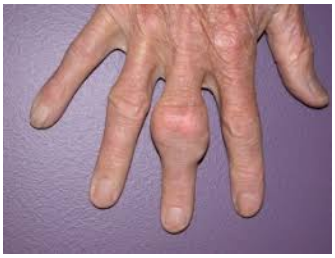


Fig: Showing Tophi

Incidence:

It affects middle aged to elderly men and postmenopausal women.

M: F ratio is 7:1 -9:1

Only around 5% of **hyperuricemic** patients develop gout.

Most women with gouty arthritis are **postmenopausal** and elderly, have osteoarthritis and arterial hypertension that causes mild renal insufficiency, and usually are receiving diuretics.

Pathogenesis

Uric acid is the end product of purine nucleotide degradation.

Its production is closely linked to pathways of purine metabolism, with the intracellular concentration of 5-phosphoribosyl-1-pyrophosphate (PRPP)

Uric acid is excreted primarily by the kidney through glomerular filtration, tubular secretion, and reabsorption.

Hyperuricemia arises with **overproduction** or **reduced excretion** of uric acid or a combination of the two.

Arthritis is caused due to deposition of **monosodium urate (MSU)** crystals in the synovium.

Polymorphonuclear leucocytes ingests crystals and release lysosomal enzymes which causes inflammation.

Acute arthritis : Crystals are demonstratable in the synovium and articular cartilage.

Chronic arthritis : Erosion of articular cartilage, proliferation of synovial membrane and pannus formation.- **secondary OA changes occurs**.

Tophi are nodular urate deposits in and around the joints in articular cartilage.

Histologically

Monosodium urate crystals are surrounded by mononuclear cell infiltration and foreign body giant cells which leads to:

Osteoarthritic changes,

Ankylosis of joints,

Tissue destruction.

Long standing cases develops multiple renal calculi, pyelonephritis, and atherosclerosis.

Clinical Features

Gout passes through 3 clinical stages :

- Asymtomatic hyperuricemia
- Acute gouty arthritis
- Chronic tophaceous gout.

Only one joint is affected initially, but polyarticular acute gout in subsequent episodes.

First joint : The metatarsophalangeal joint of the first toe is involved.

Inflamed Heberden's or Bouchard's nodes may be a first manifestation of gouty arthritis.

It begins at **night** with dramatic pain, swelling, warmth, and tenderness.

Attack will generally subside spontaneously after 3–10 days.

There are recurrent episodes with intervals of varying length with no symptoms between attacks.

Podagra : Painful affection of the foot occurring as a result of metatarsophalangeal arthritis.

Later other joints tarsal joints, ankles, and knees also are also involved.

These are associated with fever and other constitutional disturbances.

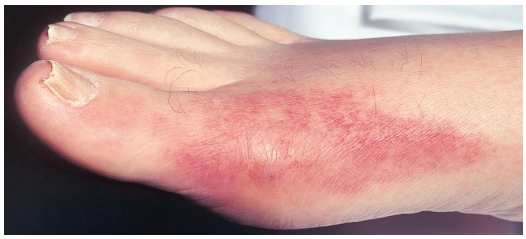


Fig: Showing Podagra

Precipitating Factors

Dietary excess : red meat, liver, pancreas, testes, peas, etc.

Trauma,

Surgery

Excessive ethanol ingestion

Hypouricemic therapy

Serious medical illnesses such as myocardial infarction and stroke.

Chronic Arthritis

Recurrent attacks presents with nonsymmetric synovitis.

It manifest only as periarticular tophaceous deposits in the absence of synovitis.

The tophi are soft and small but later becomes hard and may reach upto 7 cm in diameter.

The tophi may ulcerate discharging chalky material.

Common sites of tophi : around olecranon, ankles, tendo-achilles, helix of ear and other joints.

Complications

Renal damage : by obstruction of renal tubules by urate crystals, urate deposition in the renal parenchyma leading to renal failure. Major cause of death in gout.

Cardiovascular system : Hypertension and ischemic heart disease.

Gout is associated with insulin resistance.

Diagnosis

Synovial fluid analysis : demonstration needle-shaped MSU crystals.

Serum uric acid : elevated but normal levels do not rule out gout.

Urine uric acid: excretion of >800 mg/day in the absence of drugs suggests overproduction.

Screening for risk factors : urinalysis; serum creatinine, liver function tests, glucose and lipids; complete blood counts.

If overproduction is suspected, measurement of erythrocyte hypoxanthine guanine phosphoribosyl transferase (HGPRT) and 5-phosphoribosyl-1-pyrophosphate PRPP levels.

Joint x-rays: demonstrate cystic changes, erosions with sclerotic margins in advanced chronic arthritis.

If renal stones suspected, abdominal flat plate (stones often radiolucent), possibly IVP.

Chemical analysis of renal stones.

Differential Diagnosis

Septic arthritis

Reactive arthritis,

Calcium pyrophosphate dihydrate (CPPD) deposition disease,

Rheumatoid arthritis.

Treatment

Acute stage : symptomatic relief only since attacks are self-limiting and will resolve spontaneously.

Chronic Stage

Analgesia

NSAIDs

Colchicine

Intraarticular glucocorticoids

Systemic glucocorticoids

Uric acid–lowering agents

Chapter 2.8

Crystal Deposition Disease

These diseases are classified into 2 types:

Calcium Pyro-Phosphate Dihydrate (CPPD) Deposition Disease (Pseudogout)

Calcium Apatite Deposition Disease

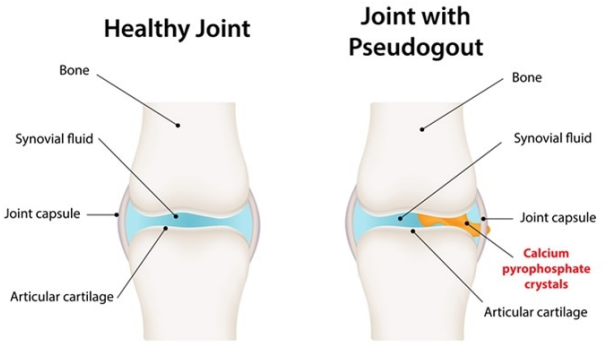


Fig: Showing CPPD Crystals in joints

Calcium Pyro-Phosphate Dihydrate (CPPD) Deposition Disease (Pseudogout)

CPPD disease is characterized by acute and chronic inflammatory joint disease, usually affecting older individuals.

The knee and other large joints are most commonly affected.

Crystals are thought **not to form in synovial fluid** but are **shed from articular cartilage** into joint space, where they are **phagocytosed** by neutrophils and incite an **inflammatory response**.

It is most common in the elderly, occurring in 10–15% of persons age 65–75 years and 30–50% of those >85 years.

CPPD is most often **idiopathic** but can be associated with other conditions like ageing.

Associated Conditions

Primary hyperparathyroidism

Hemochromatosis

Hypophosphatasia

Hypomagnesemia

Chronic gout

Postmeniscectomy

Epiphyseal dysplasias

Precipitating Factors

Trauma

Rapid diminution of serum calcium concentration, in severe medical illness or after surgery (especially parathyroidectomy).

Clinical Manifestations

It can be:

Asymptomatic

Acute

Subacute

Chronic

Acute synovitis superimposed on chronically involved joints.

Acute CPPD arthritis was termed **pseudogout** because of its striking similarity to gout.

Acute CPPD arthritis (pseudogout) : knee is most frequently involved, **but polyarticular** in two-thirds cases. Other sites wrist, shoulder, ankle, elbow, hands. and temporomandibular joint.

The involved joint is **erythematous, swollen, warm**, and **painful**.

Most patients have evidence of **chondrocalcinosis** i.e Calcium deposits in articular cartilage.

Chronic arthropathy : progressive degenerative changes in multiple joints; can resemble osteoarthritis (OA).

Joint distribution sites including knee, wrist, metacarpophalangeal (MCP), hips, and shoulders.

Symmetric proliferative synovitis : seen in familial forms with early onset; clinically similar to RA.

Intervertebral disk and ligament calcification with restriction of spine mobility

Spinal stenosis

Rarely periarticular **tophus-like nodules**.

Low-grade fever and, on occasion, temperatures as high as 40°C (104°F).

The leukocyte count in synovial fluid in acute CPPD can range from several thousand cells to 100,000 cells/μL, the predominant cell being the neutrophil.

Diagnosis

Synovial fluid analysis—demonstration of CPPD crystals, typical rhomboid or rodlike crystals (generally weakly positively birefringent or nonbirefringent with polarized light).

Radiographs or ultrasound demonstrate punctate and linear radiodense deposits within fibrocartilaginous joint menisci or articular hyaline cartilage, chondrocalcinosis and degenerative changes.

Secondary causes of CPPD deposition disease in patients <50 years old.

Differential Diagnosis

OA

RA

Gout

Septic arthritis.

Treatment

NSAIDs

Intraarticular injection of glucocorticoids.

Colchicine

Hydroxychloroquine, or even methotrexate may be helpful

Progressive destructive large-joint arthropathy may require joint replacement

Calcium Apatite Deposition Disease

Apatite is the primary mineral of normal bone and teeth.

Abnormal accumulation occurs in areas of tissue damage (**dystrophic calcification**), **hypercalcemic** or **hyperparathyroid states** (metastatic calcification), and certain conditions of unknown cause.

30–50% of patients with **osteoarthritis** have apatite microcrystals in their synovial fluid.

Apatite **aggregates** are commonly present in **synovial fluid** in an extremely **destructive chronic arthropathy** of the elderly that occurs most often in the shoulders (**Milwaukee shoulder**), hips, knees, and erosive osteoarthritis of fingers.

Periarticular or articular deposits is associated **with acute reversible inflammatio**n to **chronic damage** to the joint capsule, tendons, bursa, or articular surfaces.

Joint destruction is associated with **damage to cartilage and supporting structures**, leading to instability and deformity.

Clinical manifestations include **asymptomatic radiographic abnormalities**, **acute synovitis, bursitis, tendinitis, and chronic destructive arthropathy**.

Symptoms range from **minimal to severe pain** and **disability** leading to joint replacement surgery.

There is acute or subacute worsening of joint **pain and swelling.**

Diagnosis

The synovial fluid leukocyte count in apatite arthritis is usually low (<2000/μL) despite dramatic symptoms, with predominance of mononuclear cells.

It depends upon **identification of crystals** from synovial fluid or tissue.

Individual crystals are **very small** and can be seen only by **electron microscopy** or **x-ray diffraction studies**.

Radiographic appearance resembles CPPD disease.

Treatment

Treatment of apatite arthritis or periarthritis is nonspecific.

Acute attacks of bursitis or synovitis is self-limiting, resolving in days to several weeks.

NSAIDs

Repeated aspiration,

Rest of affected joint

Intra or periarticular injection of glucocorticoid.

Chapter 2.9

Fibromyalgia

It is a common disorder **characterized by**:

Chronic widespread musculoskeletal pain

Aching

Stiffness

Paresthesia

Disturbed sleep

Easy fatigability

Multiple tender points.

FM affects around 2% of the population.

Common in women than in men, with a ratio of 9:1.

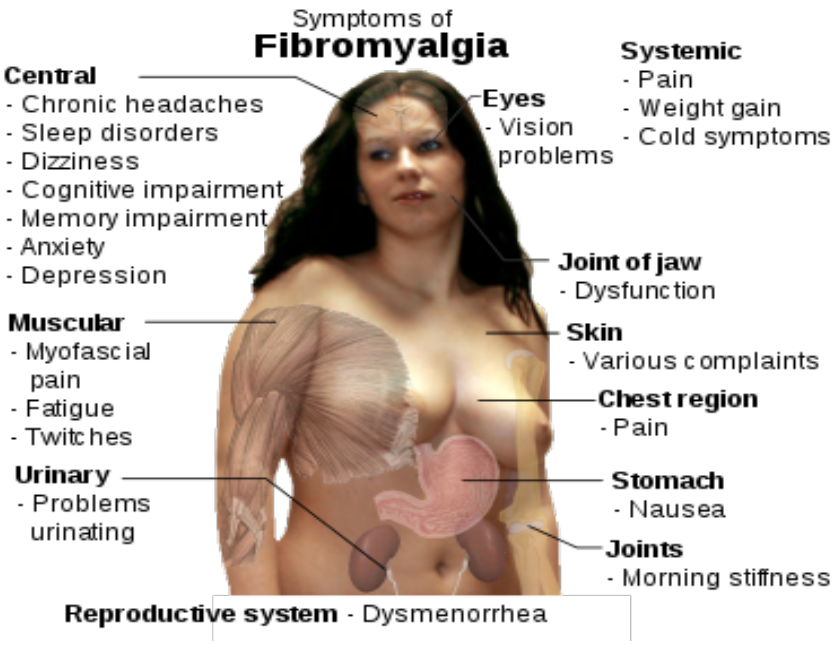


Fig: showing symptoms of fibromyalgia.

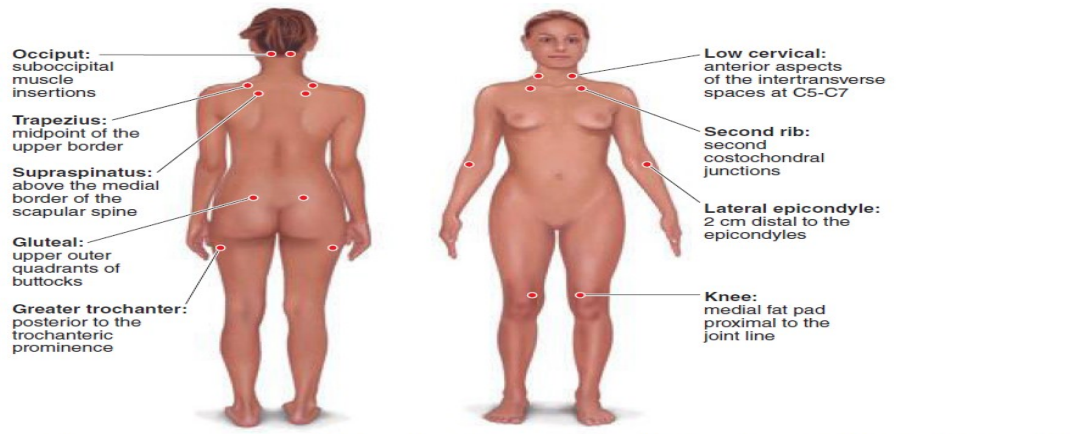
Clinical Manifestations

Patients present pain all over.

Pain both above and below the waist on both sides of the body and involves the axial skeleton (neck, back, or chest).

The pain attributable to FM is poorly localized, difficult to ignore, severe in its intensity, and associated with a reduced functional capacity.

For diagnosis, pain should be present on most days for at least 3 months.



Tender-point assessment in patients with fibromyalgia. *(Figure created using data from F Wolfe et al: Arthritis Care Res 62:600, 2010.)*

Fig: **Tender points of Fibromyalgia-** When you press on these **spots**, they feel **sore**

Neuropsychological Symptoms : fatigue, stiffness, sleep disturbance, cognitive dysfunction, anxiety, and depression.

Fatigue is highly prevalent. Pain, stiffness, and fatigue often are worsened by exercise.

The sleep complaints include difficulty falling asleep, difficulty staying asleep, and early-morning awakening.

Symptoms of anxiety and depression are common.

Headaches, facial or jaw pain, regional myofascial pain particularly involving the neck or back, and arthritis. Visceral pain involving the gastrointestinal tract, bladder, and pelvic or perineal region is often present.

Comorbid Conditions : chronic musculoskeletal, infectious, metabolic, or psychiatric conditions.

Psychosocial Considerations : Symptoms of FM often have their onset and are exacerbated during periods of high-level real or perceived stress.

Functional Impairment : physical, mental, and social domains.

Differential Diagnosis

Musculoskeletal pain is a common complaint, the differential diagnosis of FM is broad.

Inflammatory : rheumatoid arthritis, spondyloarthritides, systemic lupus erythematosus, Sjögren's syndrome.

Infectious : Hepatitis C, HIV infection, Lyme disease.

Endocrine : Hypo or hyperthyroidism,Hyperparathyroidism

Psychiatric Disease : Major depressive disorder

Lab Investigations

Routine : Erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP)

Complete blood count (CBC)

Thyroid-stimulating hormone (TSH)

Guided by History and Physical Examination : Complete metabolic panel, Antinuclear antibody (ANA)

Anti-SSA (anti–Sjögren's syndrome A) and anti-SSB

Rheumatoid factor and anti–cyclic citrullinated peptide (anti-CCP)

Creatine phosphokinase (CPK)

Viral and bacterial serologies

Spine and joint radiographs

Treatment

Understanding the symptoms.

Explaining the genetics, triggers, and physiology of FM.

Education regarding expectations for treatment. The focus on improved function and quality of life rather than elimination of pain.

Pregabalin, duloxetine, and milnacipran have shown benefit for fibromyalgia.

Tricyclics for sleep disorder.

Antidepressants and anxiolytics.

Chapter 2.10

Osteoporosis

Osteoporosis is defined as a reduction in the strength of bone that leads to an increased risk of fractures.

Loss of bone tissue is associated with deterioration in skeletal microarchitecture.

The World Health Organization (WHO) defines osteoporosis as a bone density that falls 2.5 standard deviations (SD) below the mean for young healthy adults of the same sex—also referred to as a **T-score**.

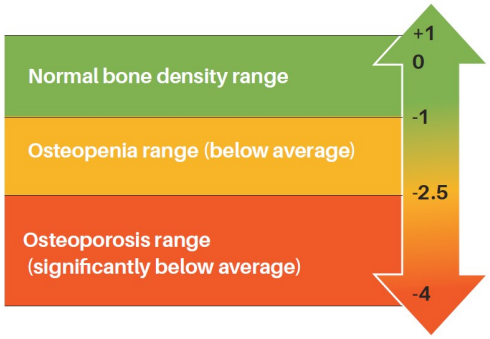


Fig: Showing bone density range

The most common sites for osteoporosis-related **fractures** are :

Vertebrae,

Hip,

Distal radius (Wrist Joint)

Hip fractures are associated with significant morbidity (thromboembolism) with 5–20% mortality rate within a year.

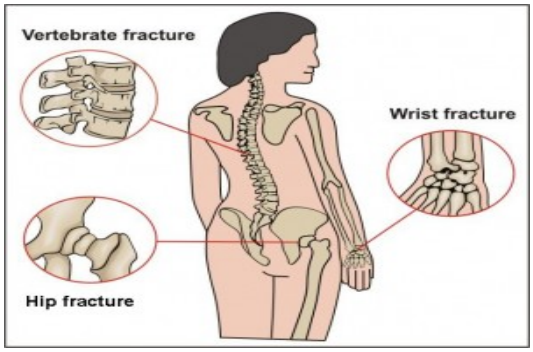


Fig: Showing common sites of fracture in osteoporosis

Etiopathogenesis

Bone Remodeling : Bone remodeling has two primary functions:

Repair microdamage within the skeleton to maintain skeletal strength.

To supply calcium from the skeleton to maintain serum calcium.

Bone remodeling is regulated by several circulating hormones, including estrogens, androgens, vitamin D, and parathyroid hormone (PTH), etc.

Calcium Nutrition: Peak bone mass may be impaired by inadequate calcium intake during growth it contributes to relative secondary hyperparathyroidism and an increase in the rate of bone remodeling to maintain normal serum calcium levels.

Vitamin D: Vitamin D insufficiency leads to compensatory secondary hyperparathyroidism and is an important risk factor for osteoporosis and fractures.

Estrogen Status : Estrogen deficiency causes bone loss by two distinct mechanisms:

Activation of new bone remodeling sites.

Exaggeration of the imbalance between bone formation and resorption.

Physical activity: prolonged bed rest or paralysis, results in significant bone loss.

Chronic Disease : Various genetic and acquired diseases are associated with an increase in the risk of osteoporosis.

Medications: *Glucocorticoids* are the most common cause of medication-induced osteoporosis.

Cigarette Consumption: Cigarette smoking also produces secondary effects that modulate skeletal status.

Clinical Features

Patients with multiple vertebral crush fractures have loss of height.

Kyphosis, and secondary pain from altered biomechanics of the back.

Thoracic fractures can be associated with restrictive lung disease.

Lumbar fractures are sometimes associated with abdominal symptoms or nerve compression leading to sciatica.

Lab Investigations

Dual-energy x-ray absorptiometry (DEXA) for measuring bone density.

Complete blood count,

Serum and 24-h urine calcium, 25(OH)D level

Renal and hepatic function tests.

Thyroid-stimulating hormone (TSH),

Urinary free cortisol,

Parathyroid hormone (PTH),

Serum and urine electrophoresis,

Testosterone levels (in men).

Treatment

Management of acute fractures

Modifying risk factors

Smoking cessation and reduced alcohol intake.

Offending drugs should be discontinued.

Exercise program should be instituted.

Treating any underlying disorders that lead to reduced bone mass.

Oral calcium (1–1.2 g/d of elemental calcium in divided doses) and vitamin D (400–800 IU/d).

Chapter 2.11

Systemic lupus Erythematosus

It is an autoimmune disease in which organs and cells undergo damage initially mediated by tissue-binding autoantibodies and immune complexes.

T- and B-cell hyperactivity, production of autoantibodies with specificity for nuclear antigenic determinants.

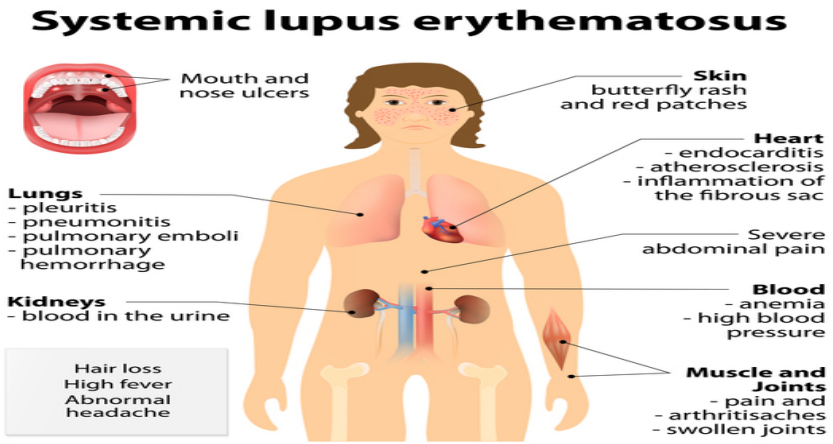


Fig: showing symptoms of SLE

Pathophysiology

The cause of SLE is incompletely understood.

Genetic factors play an important role.

It is associations with multiple polymorphisms in the HLA locus on chromosome 6. mutations in complement components C1q, C2, and C4.

TREX1, ITGAM, IRF5 and STAT4, BLK gene are associated.

The characteristic feature of SLE is the production of autoantibodies.

Patients with SLE have defects in apoptosis or in the clearance of apoptotic cells, which causes inappropriate exposure of intracellular antigens on the cell surface, leading to polyclonal B and T cell activation and autoantibody production.

Environmental factors that cause flares of lupus, such as **ultraviolet (UV) light, pregnancy and infections**, increase oxidative stress and stimulate apoptosis.

Etiopathogenesis

90% of pts are women,

Peak age at onset is between 20 and 30 years.

Usually of child-bearing age.

More common in blacks than whites.

Characterized by periods of exacerbation and relative quiescence.

It can involve any organ system and have a wide range of disease severity.

Clinical Manifestations

Constitutional: fatigue, fever, malaise, weight loss.

Cutaneous : rashes (especially malar **butterfly rash**), photosensitivity, vasculitis, alopecia, oral ulcers which may or may not be painful.

Raynaud's phenomenon is common and may antedate other symptoms by months or years.

Arthritis : inflammatory, symmetric, non erosive. Migratory arthralgia and early morning stiffness, tenosynovitis and small joint synovitis.

Hematologic: anemia, neutropenia, thrombocytopenia, lymphadenopathy, splenomegaly, venous or arterial thrombosis.

Cardiopulmonary : pleuritis, pericarditis, myocarditis, endocarditis.

Patiens are also at increased risk of **myocardial infarction** usually due to accelerated atherosclerosis.

Nephritis: classification is primarily histologic (Glomerulonephrit) is, characterised by heavy haematuria, proteinuria and casts, the main determinants of prognosis.

GI : peritonitis, vasculitis

Neurologic: organic brain syndromes, seizures, psychosis, cerebritis

Systemic: Fatigue, malaise, fever, anorexia, weight loss

Musculoskeletal

Arthralgias/myalgias

Nonerosive polyarthritis

Hand deformities

Myopathy/myositis

Ischemic necrosis of bone

Cutaneous

Photosensitivity

Malar rash

Oral ulcers

Alopecia

Discoid rash

Vasculitis rash

Other (e.g., urticaria, subacute cutaneous lupus)

Hematologic

Anemia (chronic disease)

Leukopenia (<4000/μL)

Lymphopenia (<1500/μL)

Thrombocytopenia (<100,000/μL)

Lymphadenopathy

Splenomegaly

Hemolytic anemia

Neurologic

Cognitive disorder

Mood disorder

Headache

Seizures

Mono-, polyneuropathy

Stroke,

Acute confusional state or movement disorder

Aseptic meningitis, myelopathy

Cardiopulmonary

Pleurisy, pericarditis, effusions

Myocarditis, endocarditis

Lupus pneumonitis

Coronary artery disease

Interstitial fibrosis

Pulmonary hypertension, ARDS, hemorrhage

Shrinking lung syndrome

Renal

Proteinuria ≥500 mg/24 h, cellular casts

Nephrotic syndrome

End-stage renal disease

Gastrointestinal

Nonspecific (nausea, mild pain, diarrhea)

Abnormal liver enzymes

Vasculitis

Thrombosis

Venous

Arterial

Ocular

Sicca syndrome

Conjunctivitis, episcleritis

Vasculitis

Investigations

History and physical examination

At least 4 of the 11 factors must be present.

Presence of ANA is a cardinal feature.

Laboratory assessment includes: CBC, ESR, ANA and ANA subtypes antibodies, serum immunoglobulins, VDRL, lupus anticoagulant, urinalysis, etc.

Appropriate radiographic studies

ECG

Consideration of renal biopsy if evidence of glomerulonephritis.

Aims of Management

To educate the patient about the nature of the illness,

To control symptoms and to prevent organ damage.

Goals are to control acute, severe flares and to develop maintenance strategies.

Management consists of:

Patients is advised to avoid sun and UV light exposure.

Cardiovascular risk factors- hypertension and hyperlipidaemia are controlled.

Disease restricted to skin and joints are managed with analgesics and/or NSAIDs.

Life-threatening disease affecting the kidney, CNS or cardiovascular system requires high-dose steroids and immunosuppressives.

Chapter 2.12

Systemic Sclerosis

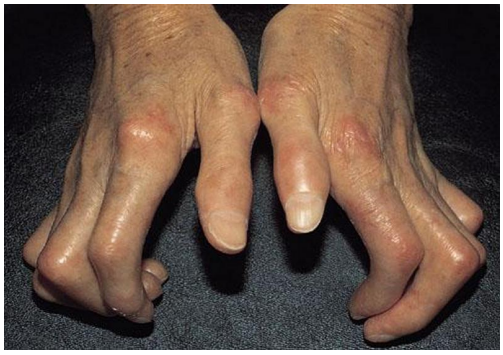


Fig Showing systemic sclerosis changes

Systemic sclerosis is an autoimmune rheumatic disease characterised by excessive **production and accumulation of collagen**, called fibrosis, in the skin and internal organs and by injuries to small arteries.

It is a multisystem disorder characterized by **thickening of the skin** (scleroderma) and distinctive involvement of **multiple internal organs** (chiefly GI tract, lungs, heart, and kidney).

Age incidence : fourth and fifth decades.

M:F – 1:4/ More common in black compared to white

Pathogenesis

Exact pathogenesis is unclear.

There is genetic associations with alleles at the HLA locus.

It involves **immunological mechanisms**, T lymphocytes infiltrate leading to vascular endothelial damage and activation of fibroblasts.

Fibroblasts increase production of extracellular matrix in the dermis, primarily type I collagen.

This results in symmetrical thickening, tightening and induration of the skin

Arterial and arteriolar narrowing occurs due to intimal proliferation and vessel wall inflammation.

Endothelial injury causes release of vasoconstrictors and platelet activation which results in further ischaemia, and exacerbate the fibrotic process.

Clinical Features

Cutaneous: edema followed by fibrosis of the skin (chiefly extremities, face, trunk); telangiectasis; calcinosis; Raynaud's phenomenon.

Arthralgias : morning stiffness and flexor tenosynovitis.

GI: esophageal hypomotility; intestinal hypofunction, gastric antral vascular ectasia (GAVE)

Pulmonary: interstitial lung disease, pulmonary arterial hypertension, alveolitis

Cardiac: pericarditis, cardiomyopathy, conduction abnormalities

Renal: hypertension; renal crisis/failure

Types

Two distinct subtypes :

Diffuse cutaneous Systemic Sclerosis*:* 30% of cases

Limited cutaneous Systemic Sclerosis **:** 70% of cases

The prognosis in DCSS is poor, with a 5-year survival of approximately 70%.

Diffuse cutaneous Systemic Sclerosis

There rapid development of symmetric skin thickening of proximal and distal extremity, face, and trunk.

At high risk for development of visceral disease early in course.

Limited cutaneous Systemic Sclerosis

It has long-standing Raynaud's phenomenon before other features appear.

Skin involvement limited to fingers (sclerodactyly), extremity distal to elbows, and face.

Generally associated with better prognosis.

Associated with pulmonary arterial hypertension.

CREST syndrome (calcinosis, Raynaud's, esophageal dysmotility, sclerodactyly, telangiectasias).

Investigations

The ESR is elevated.

Raised levels of IgG.

ANA is positive in about 70%.

Anticentromere antibodies in patients with CREST syndrome.

Radiographs: CXR, barium swallow if indicated, hand x-rays may show distal tuft resorption and calcinosis.

Others : Echo, PFT, skin biopsy.

Treatment

Education regarding warm clothing, smoking cessation, antireflux measures.

Calcium channel blockers (e.g., nifedipine) useful for Raynaud's phenomenon.

ACE inhibitors: for controlling hypertension and limiting progression of renal disease.

Antacids, H_2 antagonists, omeprazole, are useful for esophageal reflux.

D-Penicillamine to reduce skin thickening and prevent organ involvement.

Cyclophosphamide: improves lung function.

Chapter 2.13

Sjögren's Syndrome

Fig: showing clinical picture of sjogrens syndrome

It is a chronic, slowly progressive autoimmune disease characterized by lymphocytic infiltration of the exocrine glands resulting in xerostomia and dry eyes.

One-third of patients present with systemic manifestations.

Small number of patients develop malignant lymphoma.

It presents alone (primary Sjögren's syndrome) or in association with other autoimmune rheumatic diseases (secondary Sjögren's syndrome).

Incidence & Prevalance

Female-to-Male ratio - 9:1

Middle-aged women (40 – 50 Yrs)

It can occur at any age, including childhood.

The prevalence of primary Sjögren's syndrome is 0.5–1%.

30% of patients with autoimmune rheumatic diseases suffer from secondary Sjögren's syndrome.

Pathogenesis

It is characterized by both lymphocytic infiltration of the exocrine glands and B lymphocyte hyperreactivity.

The sera contain autoantibodies to non-organ-specific antigens.

The major infiltrating cells in the affected exocrine glands are activated T and B lymphocytes. T cells predominate in mild lesions, whereas B cells are dominant in more severe lesions.

Glandular epithelial cells undergo apoptotic death by signals provided from T cells.

Infiltrating lymphocytes not only provides apoptotic messages to epithelial cells but are also resistant to apoptosis.

Clinical Features

Constitutional: fatigue

Sicca symptoms: KCS and xerostomia

Dryness of other surfaces: nose, vagina, trachea, skin.

Extraglandular features: arthralgia/arthritis, Raynaud's, lymphadenopathy, interstitial pneumonitis, vasculitis (usually cutaneous), nephritis, lymphoma

Majority of patients have symptoms related to diminished lacrimal and salivary gland function.

The initial manifestations can be mucosal or nonspecific dryness, and may take 8–10 years to full-blown development of the disease.

Xerostomia : dryness of oral symptom, difficulty in swallowing dry food, an inability to speak continuously, a burning sensation, an increase in dental caries, atrophy of the filiform papillae.

Enlargement of the parotid or other major salivary glands occurs in two-thirds of patients.

Ocular symptoms: sandy or gritty feeling under the eyelids. There is burning, decreased tearing, redness, itching, eye fatigue, and increased photosensitivity.

Keratoconjunctivitis sicca: destruction of corneal and bulbar conjunctival epithelium.

Other Exocrine Gland

Diminished secretion of:

Upper and lower respiratory tree - dry nose, throat, and trachea (xerotrachea).

Exocrine glands of the gastrointestinal tract -esophageal mucosal atrophy, atrophic gastritis, and subclinical pancreatitis.

External genitalia : Dyspareunia

Dry skin

Extraglandular (systemic) manifestations

Associated with rheumatoid arthritis

It consists of easy fatigability, low-grade fever, Raynaud's phenomenon, myalgias, and arthralgias.

Pulmonary involvement: Dry cough, small airway disease.

Renal involvement - interstitial nephritis manifested by hyposthenuria(increased osmolarity) and renal tubular dysfunction with or without acidosis.

Vasculitis affects small and medium-sized vessels, manifested by purpura, recurrent urticaria, skin ulcerations.

Lymphoma is a well-known manifestation presents later in the illness.

It is manifested by:

Persistent parotid gland enlargement,

Purpura,

Leukopenia,

Cryoglobulinemia,

Ectopic germinal centers in minor salivary gland biopsy.

Diagnosis

Presence of autoantibodies (ANA, RF, anti-Ro/SS-A, anti-La/SS-B).

ESR; CBC; renal, liver, and thyroid function tests.

Mild normochromic, normocytic anemia.

Ocular studies: to diagnose and quantitate KCS; Schirmer's test, Rose Bengal staining.

Oral examination: unstimulated salivary flow, dental examination.

Labial salivary gland biopsy: demonstrates lymphocytic infiltration and destruction of glandular tissue.

Treatment

Regular follow-up with dentist and ophthalmologist.

Dry eyes: artificial tears, ophthalmic lubricating ointments.

Xerostomia: frequent sips of water, sugarless candy.

Sicca manifestations: Pilocarpine or cevimeline.

Arthralgias: Hydroxychloroquine.

Glucocorticoids: in treatment of extraglandular manifestations.

Chapter 2.14

Homoeopathic Management in case of Locomotor Disease

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.

Although constitutional treatment is advised in the patients, but there are certain specific medicines which offers instant relief in painful conditions, stiffness and limitation of movement. Some homoeopathic medicines can be given on the therapeutic basis some of which are discussed below.

Arnica: Useful for chronic arthritis with a feeling of bruising and soreness. The painful parts feel worse from being moved or touched. After traumatic injuries, soreness after overexertion, pain in back and limbs, as if bruised or beaten.

Aurum metallicum: tendency to feel depressed from joint complaints.

Belladonna: Shooting pains along limbs. Joints swollen, red, shining, with red streaks radiating. Tottering gait. Shifting rheumatic pains.

Bryonia: Acts on all serous membranes, pain is a stitching, tearing, worse by motion, better rest.

Calcarea carbonica: Deep aching arthritis involving node formation around the joints. Inflammation and soreness worse from cold and dampness.

Causticum: Useful when deformities develop in the joints, in a person with a tendon problems, muscle weakness, and contractures.Chronic rheumatic, arthritic and paralytic affections, tearing, drawing pains, with deformities about the joints, progressive loss of muscular strength.

Colchicum : affects synovial membranes of joints, joints stiff and feverish; shifting rheumatism; pains worse at night, Knees strike together, can hardly walk.

Calcarea fluorica: Helpful when arthritic pains improve with heat and motion. Joints become enlarged and hard, and nodes or deformities develop.

Dulcamara: Indicated if arthritis flares up during cold damp weather. The person gets chilled and wet.

Kali bichromicum: This is useful when arthritic pains alternate with asthma or stomach symptoms. Pains may suddenly come and go, or shift around.

Kali carbonicum: Arthritis with great stiffness and stitching pains, worse in the early morning hours and worse from cold and dampness.

Kalmia latiflora: Useful for intense arthritic pain that flares up suddenly. The problems start in higher joints and extend to lower ones..

Ledum Pal: Rheumatism begins in lower limbs and ascends.

Mag Carb: Tearing in shoulders as if dislocated. Right shoulder painful, cannot raise it.

Mag Phos: Neuralgic pains relieved by warmth.

Nux Vom: Cracking in knee-joints during motion. Drags his feet when walking. Sensation of sudden loss of power of arms and legs in the morning.

Pulsatilla: Hip-joint painful, Knees swollen, with tearing, drawing pains. Boring pain in heels toward evening. Applicable when rheumatoid arthritis pain is changeable in quality, or when the flare-ups move from place to place.

Rhododendron: Strongly indicated if swelling and soreness flare up before a storm, continuing until the weather clears. Rheumatic tearing in all limbs, especially right side; worse, at rest and in stormy weather. Stiffness of neck. Pain in shoulders, arms, wrists; worse when at rest.

Rhus toxicodendron: Useful for rheumatoid arthritis, with pain and stiffness that is worse in the morning and worse on first motion, but better from continued movement. pains and stiffness in joints. Pains tearing in tendons, ligaments, and fasciae. Pain < in wet rainy weather and after rain; at night, during rest; better motion.

Ruta graveolens: Arthritis with a feeling of great stiffness and lameness, worse from cold and damp and worse from exertion.

Sulphur : Stiffness of knees and ankles. Cannot walk erect; stoop-shouldered. Rheumatic pain in left shoulder.

Bibliography

1.Elsevier Publication, Harrison Principles of Internal Medicine 19th Edition by Kasper, Fauci, Hauser, Longo, Jameson, Loscalzo

2. Elsevier Publication Harrisons Manual of Internal Medicine 19th Edition, by Kasper, Fauci, Hauser, Longo, Jameson, Loscalzo

3 Elsevier Publication Davidson's Principles & Practice of Medicine 23rd Edition by Stuart H Ralston,Ian D Penman, Mark WJ Strachan, Richard P Hobson.

4. Textbook of Medical Psychiatry Paul Summergrad, M.D., David A. Silbersweig, M.D., Philip R. Muskin, M.D., M.A., and John Querques, M.D.

5. Oxford Textbook of Rheumatology by Richard A. Watts, Philip G. Conaghan, Christopher Denton, Helen Foster, John Isaacs, and Ulf Müller-Ladner

7. B Jain Publication Boericke New Manual of Homoeopathic MateriaMedica with repertory, by WilliaimBoericke 9th Edition

8.B Jain Publication Allen Key notes by H.C.Allen, 10th Edition

Contact Us:

University Campus Address:

Jayoti Vidyapeeth Women's University

Vadaant Gyan Valley, Village-Jharna, Mahala Jobner Link Road,

Jaipur Ajmer Express Way, NH-8, Jaipur- 303122, Rajasthan (INDIA)

(Only Speed Post is Received at University Campus Address, No. any Courier Facility is available at Campus Address)

Pages	: 137
Book Price	: ₹ 150/-

Women University Press

Jayoti Publication Desk

Year & Month of Publication- 3/4/2021