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Clinical Neurology

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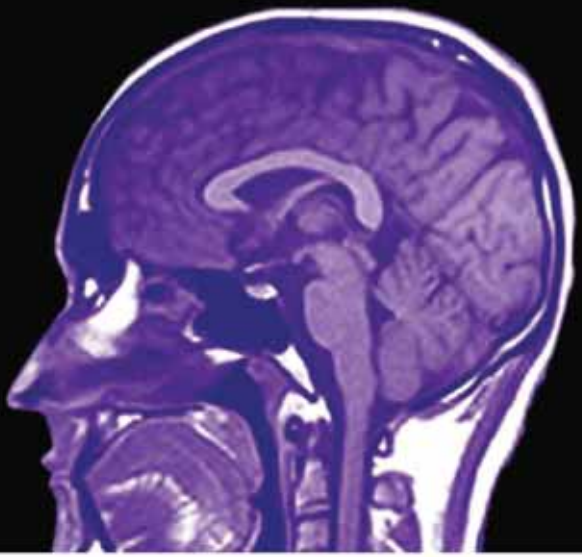

Forewords
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CHAPTER

18 Pins/Needles in Feet

Tingling, numbness, paresthesia, "jum jum" sensation are some of the common complaints in the OPD for which a quick prescription is doled out with a combination of B complex. A good history taking – the basics of clinical medicine – is imperative (Table 18.1).

Table 18.1: What is not peripheral neuropathy?

- ***Intermittent*** numbness, 'jum jum' sensation
- Numbness in one half of the body
- Numbness of one limb

Peripheral nerves have three components—sensory, motor, and autonomic and manifestations are positive and negative symptoms.

Sensory Symptoms

Positive sensory symptoms consist of pain, dysesthesia, paresthesia, tingling, burning, pricking. Once the sensory fibers are destroyed it leads to *negative symptoms* like numbness, loss of appreciation of touch, hot and cold, touch of the clothes the person is wearing, inability to appreciate the firmness of the ground. In advanced stages, it may lead to trophic ulcers. In patients who have disabling sensory symptoms like burning pain disturbing the sleep, are now happy that the pain has disappeared, *it may be good or bad,* good if the sensory signs are normal—which means restoration of nerve functions, bad if the patient has lost the ability to appreciate pain, touch and temperature all of which point to advancement of neuropathy.

Positive posterior column sensory symptoms consist of band-like constriction around the toe or the foot, the *negative symptom* is imbalance sensory ataxia—which worsen in the dark.

Motor Symptoms

The *positive motor symptoms* consist of twitching, fasciculations, myokymia. However all these are unusual in the common varieties of peripheral neuropathy. The *negative motor symptoms* consist of motor weakness, wasting in distal muscles with inability to perform finer movements of the hands, difficulty to grip slippers. If one is *aware* of slippers slipping off the feet it is motor weakness; if *unaware* it is sensory deficit.

Autonomic Symptoms

Impotence, bladder, bowel dysfunction, orthostatic hypotension (Table 18.2)

The clinical features also depend on the type of fiber involvement.

Table 18.2: Peripheral neuropathy—clinical features.

Clinical feature	*Irritative stage*	*Paralytic stage*
Sensory	Tingling Burning pain Dysesthesia Hyperesthesia Hyperalgesia	Numbness Loss of sensation—pain, touch, temperature
Posterior column	Band-like sensation across toe, leg	Sensory ataxia
Motor	Cramps, fasciculations	Weakness, atrophy
Autonomic dysfunction	Heat intolerance, sweating	Impotence, bladder, bowel dysfunction; Orthostatic hypotension
Ankle reflex	—	Absent

Small Fiber Neuropathy

(Involvement of unmyelinated or small myelinated fibers)

- ❑ Pain, touch, temperature impaired
- ❑ Preserved tendon reflexes

Large Fiber Neuropathy

(Involvement of large myelinated fibers)

- ❑ Joint position, vibration sense impaired
- ❑ Sensory ataxia
- ❑ Absent tendon reflexes

Diagnostic Approach to Peripheral Neuropathy

Is this Peripheral Neuropathy?

A clinical diagnosis of peripheral neuropathy is valid if any or all of the symptoms mentioned above are present.

This is further strengthened by physical signs of glove and stocking sensory disturbances (pin, cotton touch, sense of vibration) and weakness and wasting of distal muscles, if motor component is affected. The ankle jerk is usually absent.

Further diagnostic workup depends on the following observations. (Table 18.3).

Table 18.3: Pattern of involvement in neuropathy.

- ❑ Mononeuropathy/plexopathy/mononeuropathy multiplex
- ❑ Unilateral/bilateral; symmetrical/asymmetrical, proximal/distal
- ❑ Pure motor, pure sensory/mixed
- ❑ Upper limb predominance/lower limb predominance
- ❑ Associated autonomic or cranial nerve involvement
- ❑ Onset—acute/subacute/chronic; relapsing – remitting

The pattern has to be diligently looked for as it is a *gateway to the possible final diagnosis*.

The etiological causes are specific to the pattern of neuropathy.

The common type of neuropathy is bilateral symmetrical distal sensory/sensory motor neuropathy (Table 18.4).

Table 18.4: Clinical features of symmetric distal neuropathy.

- ❑ Symptoms—Tingling, numbness, burning paresthesia, hyperesthesia, motor weakness of hands and feet; bilateral symmetric distal
- ❑ Signs—Diminished sensations—pin, touch, temperature
- ❑ Diminished/absent ankle reflex
- ❑ Causes—Symmetric distal neuropathies are due to *several systemic disorders*. The commonest being diabetes (Table 18.5)

Table 18.5: Common systemic causes for peripheral neuropathy.

- ❑ Deficiency disorders—Vitamin B1, B6, B12
- ❑ Endocrine—Hypothyroidism
- ❑ Metabolic—Diabetes
- ❑ Toxic—Alcohol, heavy metals
- ❑ Drugs—INH, vincristine, digoxin, lithium
- ❑ Autoimmune disorders

The common variety of peripheral neuropathy is diabetic in origin, simply because diabetes is the common medical disorder affecting a large population. Diabetic neuropathy typically is bilateral, symmetrical and distal, affecting first the feet as the longest axons are in the lower limbs and then as the symptoms progress to mid calf region, distal part of upper limbs are affected.

***Case History*:** A 56-year-old female long standing diabetic, complained of burning paresthesia in the feet disturbing her sleep. Examination showed hyperesthesia over both feet and absent ankle jerks. There was no motor weakness.

Diagnosis: Diabetic sensory neuropathy.

Nerve conduction studies are superfluous and academic in nature.

***Case History*:** A 65-year-old female long standing diabetic complained of difficulty in walking due to imbalance, which became worse in the dark.

On probing she told that her slippers slip off without her knowledge. She did not have tingling, numbness or paresthesia at any time. On examination there were no motor or sensory signs, ankle reflex was absent bilaterally and here the focused neurological examination consists of elicitation of Romberg's sign, i.e. patient is asked to stand with feet together, when her eyes were open she was stable, but with eyes closed, she became unstable to the extent of falling, confirming sensory ataxia. In addition absence of vibration and joint position point to posterior column dysfunction.

***Diagnosis*:** Sensory ataxia due to diabetic sensory neuropathy (posterior column sensory dysfunction).

Message: Peripheral neuropathy can *manifest only* as sensory ataxia without paresthesia, loss of sensory functions to cotton touch, pin.

Certain clinical features distinguish neuropathy of systemic causes from local causes (Table 18.6).

Table 18.6: When is neuropathy not due to systemic cause?

- ❑ Unilateral, e.g. brachial plexopathy
- ❑ Bilateral but asymmetric
- ❑ Starting or predominantly in upper limbs
- ❑ Confined to one nerve (mononeuropathy)
- ❑ Proximal more than distal

In essence only distal symmetric distribution starting in lower limbs is of systemic origin.

Any other pattern deviating from this is due to other causes, e.g. vasculitis, infective, compressive, infiltrative. Diabetes causes symmetric neuropathy due to metabolic disorder, as well as myeloencephalopathy due to muscle complication.

Mononeuropathy

Here a single nerve trunk is affected. If acute it is due to a vascular cause and if subacute/chronic it is due to a compressive cause.

Diabetic vasculitic neuropathy causes mononeuropathy affecting ulnar, median, radial, lateral popliteal nerves.

Carpal Tunnel Syndrome

***Case History*:** A 60-year-old female non-diabetic complained of disturbed sleep in the night, waking up in the early hours with uncomfortable dysesthesias in her right hand which initially she attributed to sleeping with her head on the right hand. As the symptoms persisted she sought a consultation. First thing that strikes is that the patient has a positive sensory symptom that is localized to thumb and index finger. The anatomical localization is median nerve. Some patients have wasting of abductor pollicis brevis confirming the involvement of motor component of median nerve.

***Diagnosis*:** Right carpal tunnel syndrome—chronic compression of median nerve.

***Investigation*:** Median nerve conduction block, at carpal tunnel.

Foot drop can be due to an UMN lesion, LMN lesion at the level of root (L5-S1) due to disc prolapse or LMN lesion due to lateral popliteal nerve paralysis at the neck of fibula. Ankle jerk forms a crucial physical sign to differentiate the three. Foot drop with absent ankle jerk is a root lesion (L5-S1) as the reflex pathway travels through these roots. Foot drop with intact ankle jerk is seen in lateral popliteal nerve palsy as it supplies only the dorsi flexors of the ankle but does not participate in the reflex pathway. Foot drop with exaggerated ankle jerk is UMN lesion (Table 18.7).

Table 18.7: Differential diagnosis of foot drop.

	Lateral popliteal nerve palsy	*L5 radiculopathy*	*UMN—Spinal cord*
Distribution of weakness	Dorsiflexors of ankle	Dorsiflexors of ankle	Distal, entire limb
Tone	Hypo	Hypo	Hyper
Ankle jerk	Normal	Absent/hypo	Exaggerated
Plantar reflex	Flexor	Flexor	Extensor
Sensory findings	Normal/varying sensory loss dorsum of foot	May be impaired	Normal/impaired in other limb
Associated features	Nil	Backache along L5 distribution	Spasticity of ipsilateral limb

How to differentiate sensory symptoms of peripheral nerve localization from spinal cord localization?

As long as the symptoms are confined to the lower limbs, distally, consisting of numbness and tingling, it could mean involvement of peripheral nerve, root, or even spinal cord (sensory tracts). What definitely points to a peripheral origin is shock like sensation, burning pain aggravated by touching of the clothes so much so that they cannot wear socks or even cover themselves with a bed sheet. All these point to the involvement of peripheral sensory involvement. Characteristically the sensory symptoms ascend up to the mid-calf and then jump to the fingers and hand (length dependent neuropathy), evading thighs. This pattern is diagnostic of peripheral neuropathy. A situation where tingling continues to spread upward from the feet involving the entire lower limbs and the lower abdomen suggests spinal cord localization (Table 18.8).

Table 18.8: Differentiation of sensory symptoms of peripheral neuropathy and myelopathy.

Symptoms	*Peripheral neuropathy*	*Myelopathy*
❑ Tingling numbness	+	+
❑ Hyperalgesia	+	–
❑ Hyperesthesia	+	–
❑ Sensory symptoms	Distal	Entire lower limbs
❑ Ankle jerk	Absent	Brisk
❑ Plantar reflex	Flexor	Extensor

Investigations in peripheral neuropathy: The investigation of choice for the anatomical localization is nerve conduction studies (NCV).

Nerve conduction may be normal in frank neuropathies and abnormal in asymptomatic patients and also depends a lot on the experience of the operator (Table 18.9).

Table 18.9: Utility of nerve conduction studies.

- ❑ Confirming the diagnosis of neuropathy. In the process excluding the disorders of myoneural junction and muscle
- ❑ The type of neuropathy—sensory/motor or mixed
- ❑ The pathological type—demyelinating or axonal or conduction block
- ❑ Symmetric/asymmetric
- ❑ Mono/polyneuropathy

Pitfalls of Depending on Nerve Conduction Study

The patient may have typical burning dysesthesias in the feet, and one expects to find an absent ankle jerk, but surprisingly it may be in intact. Even the nerve conduction studies show normal results. One should not discard the diagnosis but bank on the symptoms. This is because the sensory symptoms are due to unmyelinated or small myelinated fibers which do not take part in the nerve conduction or in the ankle reflex pathways. However if the symptoms are due to posterior column dysfunction like sensory ataxia one would definitely expect absent ankle jerks and delayed conduction velocities. In diabetics and elderly patients the ankle reflex may be absent, even though there are no symptoms or signs of peripheral neuropathy. Nerve conduction studies may be normal in small fiber neuropathy and abnormal *in asymptomatic individuals* (diabetic, elderly).

Similarly, median nerve conduction block at the wrist (carpal tunnel syndrome) is often observed in asymptomatic individuals, hence the investigative reports are *relevant only if correlating with clinical diagnosis.*

INVESTIGATIONS FOR ETIOLOGICAL DIAGNOSIS

Basic—Hemogram, blood sugar, B12, folic acid, renal, hepatic profile.

Add on—Protein electrophoresis HIV, ANA, dsDNA, ACE,
Vasculitic profile: nerve biopsy in select cases (Table 18.10).

Table 18.10: Indications for nerve biopsy.

- ❑ Hansen's disease
- ❑ Vasculitic neuropathy
- ❑ Amyloid neuropathy
- ❑ Hereditary neuropathy

Psychogenic Causes

Intermittent fluctuating numbness of one half of the body particularly in women is a common psychogenic complaint. Sensory symptoms which are *transient, changing locations*, inconsistent and of *long duration* not *affecting daily* activities point to psychogenic origin. Elderly patients often complain of sensory symptoms in one limb or one half of body and reassurance that these symptoms are non specific and *will not lead to stroke* is more satisfying than a flurry of investigations, because they are worried that the sensory symptoms are the forerunner of stroke! I reassure them saying that "stroke strikes suddenly: and it does not send messages!" So note that any "jum jum" sensation is not neuropathy.

***Case History*:** A 30-year-old female complains of intermittent numbness of left lower limb of three months duration. MR lumbar spine showed L4, 5 disc prolapse and she was referred for opinion on possibility of surgical management.

Further history revealed that the numbness fluctuated more in the later part of the day and on probing further she mentioned that she also had other fluctuating symptoms like numbness of left upper limb and chronic headache.

Step 1: *Neurological deficit*—Total loss of sensation in the left upper and lower limb with normal motor power and gait.

Step 2: *Anatomical localization*—Total loss of sensation, means sensory nerve root affecting the entire lower and upper limb which is not possible with history of fluctuating symptoms.

Diagnosis is psychogenic—functional- which required no further investigation. Patient was referred to a psychiatrist.

Symptomatic Management of Sensory Neuropathy

The drugs used for positive sensory symptoms like burning paresthesia, disturbing pain, tingling are tricyclic antidepressants (amitriptyline) and antiepileptic drugs (carbamazepine, gabapentin).

These are NOT useful in negative sensory symptoms like numbness, loss of sensation.

HANSEN'S DISEASE

Hansen's disease being less common, is usually not thought of. However, it is an eminently treatable condition, so one should not miss it.

Hansen's disease is classified into tuberculoid, borderline, lepromatous. Neuropathy is commonly seen in tuberculoid. Lepromatous type is reflection of low immunity and bacilli can be detected in skin biopsy.

The diagnosis of Hansen's neuropathy can be confirmed by appropriate nerve biopsy which shows inflammatory changes with lepra bacilli. The treatment is for 6 months to 2 years.

***Case History*:** A 35-year-old male complained of loss of sensation over the dorsal aspect of right hand and middle finger. Nerve conduction done showed evidence of "carpal tunnel syndrome".

Clinical examination showed depigmented hypoesthetic large patch over the dorsum of the hand extending to the middle finger.

Diagnosis: "Hansen's disease". Nerve conduction showing carpal tunnel syndrome is *incidental* because the median nerve which traverses through the carpal tunnel does not supply the skin of dorsum of the hand!

Message: Investigation without a clinical diagnosis may take you up the wrong path!

***Case History*:** A 56-year-old male presented with history of numbness of right face of one year duration and numbness of the right foot since six months and inability to close right eye since three months. He was on antidepressants for a long period of time. He had already undergone extensive investigations including MR brain, cervical spine and LS spine all of which were normal. On examination he had partial LMN facial palsy with reduced pin sensation and greatly thickened great auricular nerve, ulnar nerve and right lateral popliteal nerve.

***Message*:** Partial facial palsy means one of the branches of facial nerve is affected and NOT the nerve trunk as in Bell's palsy. Hansen's disease does not involve the nerve trunk. Patchy sensory loss over face is suggestive of sensory nerve twigs involvement, NOT trigeminal nerve trunk.

***Diagnosis*:** Hansen's disease.

Thickened nerves are commonly seen in ulnar, lateral popliteal nerves. The thickened cutaneous nerves are observed in great auricular nerve, dorsal cutaneous branch of ulnar nerve and cutaneous branch of radial nerve.

Hansen's neuropathy is predominantly sensory or sensory motor and manifests as mononeuropathy/mononeuropathy multiplex.

Lateral Popliteal Nerve Palsy

***Case History*:** A 35-year-old male diabetic of 10 years duration had abrupt onset of difficulty in walking with left foot. On examination there was left foot drop with *intact ankle jerk* with no sensory impairment.

Step 1: Neurological deficit—Left ankle dorsiflexor paresis.

Step 2: Anatomical localization—Lateral popliteal nerve.

Step 3: Pathological diagnosis—Abrupt onset—vascular—lateral popliteal nerve infarct.

Step 4: Etiological diagnosis—Diabetic vasculitis.

***Diagnosis*:** Lateral popliteal nerve palsy.

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Clinical Neurology is on the decline with the availability of an array of investigative tools—CT, MR, EEG, ENMG etc. However, it is a mistaken belief and fond hope, that investigations will 'tell' the diagnosis. As there are many incidental findings in MR Brain (e.g. lunar infarct), MR spine (C5-6 & L 4-5 disc prolapse) etc., they may mislead the clinicians to wrong diagnosis and inappropriate treatment. Think of this—many common neurological diseases—headache, epilepsy, movement disorders (Parkinson's, tremors etc.) and dementia—are all clinical diagnosis—there are no investigations to confirm the diagnosis!

This book *Clinical Neurology Made Easy®* is to remind the clinicians to make an appropriate clinical diagnosis by following stepwise approach and then investigate accordingly.

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ISBN 978-93-5270-251-0