Polyneuropathies

Peripheral neuropathy is common, often distressing and sometimes disabling or even fatal. The prevalence is about 24 per 1,000, rising with age to 80 per 1,000. Most polyneuropathies are chronic and usually develop over several months. Three main patterns of polyneuropathy can be distinguished and each has a different differential diagnosis:

- Acute symmetrical peripheral neuropathy.
- Chronic symmetrical peripheral neuropathy.
- Multiple mononeuropathy.

Both peripheral and cranial nerves are affected, either by axonal degeneration (nerve becomes electrically inert within one week) or demyelination, which initially leaves the axon intact and results in blockage or slowing of conduction.

Presentation

Patients with polyneuropathy may present with altered sensation, pain, weakness or autonomic symptoms. Acute symmetrical polyneuropathy (eg, Guillain-Barré syndrome) is uncommon.

Sensory neuropathy

- In sensory polyneuropathy, usually, the feet are affected first.
-Paraesthesiae, numbness, burning pain, and loss of vibration sense and position sense are prominent. Muscle wasting may occur.
- The sensory neuropathy may be subacute with ataxia caused by loss of sense of posture.

Autonomic neuropathy

- Polyneuropathy often affects the autonomic nervous system.
- Typical symptoms are constipation, loss of bowel or bladder control and orthostatic hypotension. The skin may become pale and dry and sweating may be reduced.

Hereditary polyneuropathy

Hereditary causes of polyneuropathy may also cause hammer toes, high arches and scoliosis.

Differential diagnosis

- Predominantly motor peripheral neuropathies include Guillain-Barré syndrome, Charcot-Marie-Tooth syndrome, porphyria, lead poisoning and diphtheria.
- Possible causes of painful peripheral neuropathy include alcoholic neuropathy, diabetic amyotrophy, porphyria, vitamin B1 deficiency or vitamin B12 deficiency and carcinoma.

The cause of chronic polyneuropathy is often unknown. The most common causes of peripheral neuropathy are:

- Diabetic neuropathy.
- Nutritional, including alcohol (with or without vitamin B1 deficiency), B12 deficiency.
Others causes include:

- Heavy metal poisoning - eg, lead, mercury.
- Infection - eg, HIV, leprosy, diphtheria, tetanus, botulism.\(^3\)
- Malignancy - eg, lung cancer, breast cancer, myeloma.
- Metabolic - eg, hypothyroidism, liver failure, chronic kidney disease, acute intermittent porphyria.
- Chronic vascular disease - eg, polyarteritis nodosa, systemic lupus erythematosus.\(^4\)
- Chronic inflammatory demyelinating polyneuropathy.
- Postinfective polyneuritis - eg, Guillain-Barré syndrome.
- Sarcoidosis.
- Drugs - eg, isoniazid, vincristine, phenytoin, nitrofurantoin, gold and excess amounts of vitamin B6 (pyridoxine).

Investigations\(^2\)

Initial tests include:

- Urine: glucose, protein.
- Haematology: FBC, erythrocyte sedimentation rate (ESR), vitamin B12, folate.
- Biochemistry: fasting glucose, renal function, liver function and thyroid function.

Further investigations will depend on the outcome of clinical assessment and initial investigation results:

- Neurophysiology testing with assessment of distal and proximal nerve stimulation; electrophysiological procedures are helpful in determining the pathological process which may be either an axonopathy, a myelinopathy or a neuronopathy.\(^5,6\)
- Immunology: antinuclear factor, anti-extractable nuclear antigen antibodies (anti-Ro, anti-La), antineutrophil cytoplasmic antigen antibodies.
- Urine: Bence-Jones protein.
- Cerebrospinal fluid: cells, protein, immunoglobulin oligoclonal bands.
- Immunology: anti-HIV antibodies, antineuronal antibodies (Hu, Yo), antigliadin antibodies, serum angiotensin-converting enzyme, antiganglioside antibodies, antimyelin-associated glycoprotein antibodies.
- Search for carcinoma, lymphoma or solitary myeloma.
- Molecular genetic tests - eg, for Charcot-Marie-Tooth syndrome.
- Nerve biopsy may be required.

Management

The initial assessment of the diagnosis and underlying cause is usually performed in secondary care. Re-referral may be indicated at a later stage if there is a significant deterioration in symptoms or an alteration in the presentation suggesting a further assessment is required.

Acute multiple mononeuropathy requires urgent assessment, as the most common cause is vasculitis. Prompt treatment with steroids, with or without cyclophosphamide, may prevent further irreversible nerve damage.

- Preventative and palliative treatments include foot care, weight reduction, sensible footwear and foot orthoses.
- Patients with severe leg weakness may need walking aids.
- Simple wrist splints can help weak wrist extension.
- Disabled patients require help from a multidisciplinary team including an occupational therapist and a physiotherapist.

Pharmacological

- Specific treatment depends on the cause.
- Good control of glucose and blood pressure in patients with diabetes may improve or at least slow the progress of neuropathy.
Chronic inflammatory demyelinative polyneuropathy is treatable with corticosteroids, intravenous immunoglobulin, plasma exchange and some immunosuppressant drugs. Multifocal motor neuropathy responds to intravenous immunoglobulin, and possibly immunosuppressant drugs but not to corticosteroids or plasma exchange. No specific treatment is available for chronic idiopathic axonal polyneuropathy. Painful neuropathy is difficult to treat. The most useful drugs are amitriptyline, duloxetine, gabapentin or pregabalin.

Complications

- With loss of sensation, recurrent injury to joints may lead to permanent joint destruction (Charcot joint).
- May lead to disability, social isolation or loss of independence, especially in the elderly.

Further reading & references

7. Said G; Chronic inflammatory demyelinating polyneuropathy. Neuromuscul Disord. 2006 May;16(5):293-303.

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