Peripheral nerve entrapment syndromes of the upper limb

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Abstract

Peripheral nerve entrapment syndromes are a common condition in the upper limb resulting from the persistent application of pressure to a nerve. The symptoms include pain, numbness, tingling, muscle weakness and atrophy. The distribution of symptoms depends upon the nerve affected. This article discusses the aetiology, epidemiology and pathogenesis of peripheral nerve entrapment in the context of the upper limb, as well as the principles of diagnosis and management. Common examples of nerve entrapment are described in greater depth.

Keywords Carpal tunnel syndrome; cubital tunnel syndrome; double crush phenomenon; nerve compression; nerve decompression; peripheral nerve entrapment; thoracic outlet syndrome

Definition

Peripheral nerve entrapment syndrome (or peripheral entrapment neuropathy) is a neurological condition caused by direct pressure on an individual nerve along its course. Symptoms include pain, tingling, numbness, muscle weakness and atrophy. Surgical treatment in these cases is directed at relieving pressure on the nerve, but may not always relieve all the symptoms.

Aetiology

Entrapment neuropathy generally refers to the situation where the patient's own anatomy contributes to compression of a nerve. These neuropathies can be broadly classified into two categories dependent on the duration of the compression, that is, acute or chronic. In acute cases the nerve compression is the result of a discrete injury such as crush injury or a haematoma, whereas in chronic cases the compression occurs gradually over time as a result of increased pressure at sites of anatomic narrowing through which the nerve passes. Chronic entrapment neuropathies can be caused by a discrete tumour (lipoma, ganglion or neurofibroma) or alternatively there may be expansion of tissues around a nerve in a space where there is a little room to accommodate such expansion, as is often the case in carpal tunnel syndrome. This may be due to weight gain, peripheral oedema (especially in pregnancy) or to a specific condition such as acromegaly, hypothyroidism or scleroderma.

Some conditions cause nerves to be particularly susceptible to compression, such as diabetes, where neural blood supply is already compromised thus rendering the nerve more susceptible to minor degrees of compression. Inherited conditions such as hereditary neuropathy with liability to pressure palsies (HNPP) are much rarer, but can lead to multiple entrapment neuropathies which are difficult to treat.

Epidemiology

Carpal tunnel syndrome is the most common entrapment neuropathy, with a lifetime risk in the Western world approaching 10%. It is more common with increasing age, and overall prevalence is slightly higher in women (3%) compared to men (2%). Incidence is approximately 1 in 1000 per year. Peak prevalence is in females over the age of 55.

Ulnar neuropathy is the second most common entrapment neuropathy and is more common in men than women. Incidence is 21–25 cases per 100,000 population.

Pathology and pathogenesis

There is increasing agreement that the pathophysiology of compressive neuropathy is based upon a vascular aetiology. Mechanical obstruction of the venous return from the nerve, due to positional changes or local anatomy, leads to venous congestion, circulatory compromise and relative anoxia. Secondary small vessel and capillary dilation results in endothelial oedema and further compromise of venous return. Persistent endothelial oedema and disruption to the intraneural microcirculation leads to fibroblast proliferation and fibrosis. Fibrosis acts as a barrier to the exchange of vital nutrients and oxygen within the nerve, causing impaired axonal transport, segmental demyelination and deterioration in the function of the nerve. Ultimately this leads to irreversible damage and loss of motor endplates, causing muscle atrophy.

The double crush theory[1] predicts that a minor compressive lesion at one point along a peripheral nerve lowers the threshold for occurrence of symptomatic compression at another site, secondary to internal derangement of nerve cell metabolism. This is because the normal physiology of a nerve relies upon the unimpeded conveyance of an impulse and the exchange of nutrients along the entire length of the axons.

Factors associated with entrapment neuropathy are:

- vascular
  - diabetes, microcirculatory disease
- Inflammatory
  - synovitis, rheumatoid arthritis
- trauma
  - supracondylar fracture of the humerus, dislocation of the lunate
- anatomical
  - anomalous muscles, fascial bands, anomalous vascular plexus
- metabolic
  - pregnancy, hypothyroidism

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- iatrogenic
  - injections, haematoma
- tumour
  - ganglion, lipoma, sarcoma.

Principles of diagnosis

The symptoms and signs encountered depend upon which nerve is affected, and where along its length it is affected. Sensory disturbance is usually the first symptom noticed, particularly tingling or fizzing, followed or accompanied by reduced sensation or compete numbness. Muscle weakness is usually noticed later, and is associated with muscle atrophy. Sympathetic loss is also present (loss of sweating for instance) but this is rarely noticed by the patient.

An entrapment neuropathy can usually be diagnosed confidently on the basis of symptoms and signs alone. Reproducing symptoms via provocative manoeuvres or diagnosis deficits in power or sensibility can be accomplished in the clinic or with the aid of hand therapists. Two-point discrimination testing, Semmes-Weinstein monofilament testing, and grip strength measurements are simple and inexpensive means of evaluation.

Electrodiagnostic studies (such as electromyography or nerve conduction studies) may be a useful adjunct to history and examination for diagnosing the presence and level of nerve compression especially in patients presenting with unclear symptoms and sign. However, these studies are highly operator dependent and results do not always correlate with severity of symptoms or clinical outcome. Nerve conduction studies are particularly useful for confirming diagnosis in equivocal cases, quantifying severity of compression and ruling out involvement of other nerves (suggesting a mononeuritis multiplex or polyneuropathy). They may also be used to verify progression or resolution in neurophysiology following surgical decompression. Nerve conduction studies showing prolonged sensory and motor latencies, and decreased amplitude of nerve conduction potentials are highly suggestive of a compressive or entrapment neuropathy.

Radiological investigation is not usually indicated, but may be helpful if a tumour or other locally compressive mass lesion is suspected.

Principles of management

When compression neuropathy is secondary to a contributing condition, treatment should first be directed at this condition. For example, if weight gain is the underlying cause, weight loss is appropriate. Compression neuropathy during pregnancy often resolves after parturition, in which case simple supportive measures during pregnancy (nocturnal splints for instance) may be all that is required.

Physiotherapy, rest, behaviour modification and splintage all have a role in the management of nerve compression. If the cause is inflammatory then there may also be a role for steroid injection to reduce inflammation while nerve recovery occurs.

Some entrapment neuropathies are amenable to surgical decompression. Carpal tunnel syndrome and cubital tunnel syndrome are two common examples. Whether or not surgery is appropriate for any given case depends upon severity of the symptoms, risks of the proposed surgery and prognosis if left untreated. After surgery, symptoms may resolve completely, but if the compression was sufficiently severe or prolonged then the nerve will not recover completely and some symptoms will persist.

Drug treatment may be appropriate to treat an underlying condition, such as an appropriate pharmacologic agent to relieve peripheral oedema, or for the relief of severe neuropathic pain (tricyclic antidepressants such as amitriptyline or γ-aminobutyric acid analogues such as gabapentin or pregabalin).

Common compressive neuropathies

A list of the most common entrapment neuropathies in the upper limb is seen in Table 1.

Examples of entrapment neuropathies in the upper limb

Nerve involved	Syndrome	Where entrapped
Brachial plexus	Thoracic outlet syndrome	Interscalene space (cervical rib, cervical band, scalene anomalies, 1st rib masses, suprapleural membrane, Pancoast tumour)
Median nerve	Pronator syndrome	Ligament of Struthers, bicipital aponeurosis, pronator teres, flexor digitorum superficialis arcade
	Anterior interosseous syndrome	Pronator teres, flexor digitorum superficialis, Ganzer muscle
	Carpal tunnel syndrome	Carpal tunnel
Ulnar nerve	Cubital tunnel syndrome	Cubital tunnel, Arcade of Struthers,
	Guyon's canal syndrome	Guyon's canal
Radial nerve	Radial tunnel syndrome	Radiocapitellar joint, supinator, Arcade of Frohse, Leash of Henry, tendinous edge of extensor carpi radialis brevis muscle
	Posterior interosseous syndrome	
	Wartenburg's syndrome	Superficial distal forearm (distal brachioradialis)

Table 1