Peripheral nerve lesions in Zimbabwe: a retrospective study

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A retrospective study of all individuals referred to the Clinical Neurophysiology Laboratory, Harare during a three year period and found to have electrophysiological evidence of peripheral nerve lesions is presented. One hundred and ninety-two patients had peripheral nerve lesions of which 64 were traumatic and 128 non-traumatic. The commonest cause of trauma was road traffic accidents and the nerves most frequently affected were ulnar, sciatic and radial. Ten individuals had iatrogenic nerve lesions following either surgical procedures or intramuscular injections.

Of the non-traumatic lesions carpal tunnel syndrome was diagnosed in 73% of cases. Women were more often affected than men. Many (59%) of the patients had unilateral symptoms but 20 of them had significantly reduced conduction velocity on the other side. In 18 individuals evidence of the "double crush" syndrome was found. Carpal tunnel syndrome was considered idiopathic in most cases.

Tardy ulnar palsy occurred in 14 cases and 11 patients had radial nerve palsy. The pattern of peripheral nerve lesions found in this report appears similar to that reported elsewhere.

Introduction

In 1994, a Clinical Neurophysiology Laboratory was set up in Harare, Zimbabwe, the first of its kind in the Central African Region. Patients are referred from both the public and private health sectors from throughout Zimbabwe (population 12.3 million, area 390,000km^2). Tests of nerve function carried out in this laboratory include sensory and motor nerve conduction measurements, electromyography and somatosensory evoked potentials. Their application has been reviewed previously[1].

Peripheral nerves are common sites of injury. The consequences can be debilitating to the individual in terms of loss of function with possibly a concomitant loss of livelihood. The management of such injuries depends on their extent and severity and whether recovery is likely. Neurophysiological (electrophysiological) tests are helpful in localising the injury, determining its pathophysiology (whether an axonal injury or a disturbance of the myelin sheath), assessing the severity of the resulting dysfunction and assessing the progress of recovery and re-innervation of the muscle[2].

The objective of this study was to analyse the distribution, presentation and causes of peripheral nerve lesions in patients referred to this Laboratory.

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Patients and methods

All patients referred over three years (1995-1997) and diagnosed, following clinical and neurophysiological evaluation, to have lesions of peripheral nerves were retrospectively reviewed from data stored on a computerised storage system.

Nerve lesions were considered to be complete if the nerve segment stimulated failed to elicit a motor response (a compound muscle action potential (CMAP) or a sensory nerve action potential) and if there was an absence of motor unit action potentials on the electromyogram in response to volitional activity[2]. Partial lesions were those in which a response (a CMAP or a sensory nerve action potential), albeit delayed or reduced in amplitude, was recordable. Neurapraxic injuries, where a segment of a nerve exhibits a conduction block but the segment distal to the lesion may conduct normally[3], were included in the partial lesions category. Tests were carried out at least one week following the injury during which time distal segments of a severed nerve would have degenerated[2]. Fibrillation potentials which are indicative of muscle denervation do not make an appearance until at least three weeks after trauma[2].

Results

During the study period, 235 patients presented to the Clinical Neurophysiology Laboratory with symptomatology suggestive of peripheral nerve lesions or with requests by referring clinicians to exclude nerve lesions. Of these, 192 patients (91 men and 101 women) were diagnosed to have peripheral nerve lesions. Most, 182 (95%) had lesions affecting only a single nerve but 10 patients had multiple nerve lesions. In 64 patients the lesions were traumatic and they were non-traumatic in the remaining 128 cases.

TRAUMATIC NERVE LESIONS

Men (mean age 34+/-16) were more affected than women (M:F;1.9:1). Table I lists the nerves affected. There was only a single case of bilateral lesions and that was in an elderly woman who was pulled out of a bath by her arms resulting in a bilateral ulnar nerve palsy. Ten individuals had iatrogenic nerve lesions following either surgical procedures or intramuscular injections (Table II). Commonest causes of trauma were road traffic accidents followed by accidents in the home (Table III). Miscellaneous causes included an individual who suffered median nerve damage during an episode of decompression sickness following deep water diving, an individual being restrained by wires and another being badly beaten with a truncheon across the upper arms.

In 44 individuals, the trauma resulted in partial lesions, and in the remaining 20 the lesion was considered to be complete. Of those with partial lesions, 14 had neurapraxia and they were reassured that full recovery was likely to take place (albeit over a period of a few weeks). Some degree of recovery was likely in a further 12 patients, while 10 patients were asked to return in three to six months time to assess the recovery process. Exploratory surgery was recommended in five cases and in the remaining three patients recovery or further recovery was thought unlikely in view of the time that had elapsed since the injury. Of the 10 that were asked to return, five were lost to follow up, four returned and showed improvement and one showed no improvement. In his case, surgery was recommended.

Of the 20 patients with complete lesions surgery was recommended in 11 patients, five were asked to return of whom only one returned with no signs of improvement and was recommended for surgery. The others were lost to follow up. In the remaining four cases recovery was thought unlikely.

Non-traumatic nerve lesions

The 128 patients (46 men and 82 women) in this category had no history of trauma immediately prior to onset of symptoms. The median nerve was the most commonly affected nerve, followed by the ulnar and the radial nerves (Table I). All the median nerve lesions, with the exception of one, involved compression of the nerve under the flexor retinaculum (carpal tunnel syndrome, CTS). Individuals with CTS are discussed separately.

Fifteen patients, 10 men and 5 women, had ulnar nerve lesions. In 14 the conduction velocity along the ulnar nerve was significantly slower across the elbow segment and was thus consistent with a diagnosis of tardy ulnar palsy[4]. This was unilateral in 13 and bilateral in one patient. Only two patients had a history of previous injury to the elbow. Exploration of the elbow and transposition of the nerve was recommended in 13 cases and one patient was asked to return for review. The one patient

TABLE I *Nerves affected by traumatic and non-traumatic lesions.*

Nerve	*Traumatic lesions*	*Non-traumatic lesions*
Ulnar	14	15
Sciatic	11	1
Radial	11	11
Median	9	94
Peroneal	7	
Tibial	1	
Sural	1	
Median/ulnar/radial	3	
Median/ulnar	2	
Median/radial	2	
Radial/ulnar	2	
Peroneal/tibial	1	1
Facial		5
Lateral femoral cutaneous		1

TABLE II *Iatrogenic nerve lesions*

Cause	*Nerve affected*	*Number*
Intramuscular injection into the gluteal muscle	Sciatic	4
Surgery for hip replacement	Sciatic	1
Open reduction for fractured hip	Sciatic	1
Delayed reduction for dislocated hip	Sciatic	1
Surgery to the calf	Sural	1
Surgery for exploration of the ulnar nerve	Radial	1
Application of tourniquet	Ulnar	1

TABLE III *Causes of trauma*

Cause		*Number*
Road traffic accidents		22
Accidents at home		10
Gunshot wounds		3
Occupational accidents		3
Stab wounds		3
Humerus fracture (cause unknown)		3
Miscellaneous:	Sports injury	1
	Tied with wire	1
	Dog bite	1
	Beaten on arms	1
	Decompression sickness	1
	Unknown	5

who did not have slowing across a specific segment of the ulnar nerve was nevertheless surgically explored and the ulnar nerve was transposed. His symptoms recurred after a few days and subsequent investigations revealed an underlying illness (HIV infection) which is known to cause a non-compressive mononeuropathy[5].

Eleven individuals (10 men and 1 woman) presented with an acute onset wrist drop and a radial nerve neurapraxia was diagnosed. The woman reported that she had fallen asleep on her arm but none of the men could think of any precipitating factors.

The facial nerve palsies of five individuals (2 men and 3 women) were considered to be idiopathic and typical of Bell's palsy. Recovery, with time, was expected to be complete. The fifth had a previous history of ear infection. His facial nerve was severely damaged and although there was ongoing re-innervation it was not possible to predict whether recovery would be complete.

Two other individuals also appeared to have peripheral nerve lesions following infections: one had a median nerve lesion at the level of the digits following gas gangrene of the hand; the other, a curious case, presented with progressive weakness and wasting of the left leg following a septic foot injury. There was no history of surgery for wound incision and drainage and neither had a tourniquet been applied. Nerve conduction studies demonstrated unequivocal damage to the sciatic nerve.

Carpal Tunnel Syndrome

Ninety-four individuals (23 men and 71 women) satisfied electrophysiological criteria for CTS, that is a difference of >0.4ms between the median palmar sensory nerve and the ulnar palmar sensory nerve latencies[6]. Seven patients were aged between 16 and 30 years, 24 were between 31 and 45 years and the majority (63) were older than 45 years. All the patients complained of pain and/or paraesthesia involving the radial three fingers which was worse at night. Patients reported a paraesthesia which did not always correspond to the median nerve distribution. Often the whole hand and sometimes the arm extending to the shoulder was painful. Nineteen patients complained of additional weakness of the hand. Symptoms had persisted for

over a year in 21 patients. Four patients presented with symptoms seemingly totally unrelated to their pathology: one woman complained of weakness of one arm and leg, another complained of pain in the breast and two men complained of pain in the legs. In none of these cases did neurological or neurophysiological investigations reveal any deficit other than the CTS. Of the 94 patients, 55 presented with symptoms affecting one hand. However, 20 of these had significant slowing of the median palmar sensory nerve in the other hand but were as yet asymptomatic. The other 39 had bilateral symptoms and bilateral CTS was confirmed. Eighteen patients had, in addition to CTS, electrophysiological evidence and clinical evidence of cervical root irritation, the "double crush" syndrome.

In 87 patients, the syndrome was considered idiopathic (although some women mentioned that they used their hands excessively in knitting or typing). Precipitating factors were found in some cases: three had diabetes mellitus[7], two had arthritis[8], one had hypothyroidism[9] and one was pregnant[10]. Two patients who presented with weakness and paraesthesia of the lower limbs were found to have a sensorimotor axonal polyneuropathy in addition to the carpal tunnel syndrome. This was characterised by absent sural responses and motor responses of greatly reduced amplitude. One woman had electrophysiological evidence of a widespread demyelinating polyneuropathy consistent with a diagnosis of chronic inflammatory demyelinating polyneuropathy (CIPD) underlying the CTS.

Surgical decompression of the median nerve was recommended in 83 cases. Those with minimal pathological slowing and milder symptoms were recommended steroid injections. Five individuals had already undergone surgical decompression but their symptoms had persisted. Reduced median nerve conduction velocity was apparent but without preoperative values it was not possible to ascertain whether there had been improvement or not. All were recommended to have further surgery.

Discussion

The distribution and frequency of peripheral nerve lesions in this survey cannot be taken as representative of actual population prevalence for several reasons such as the difficulties of referral over large distances, lack of familiarity with neurophysiological investigations by practitioners and economic hardships. All play a role in preventing referral. However, the pattern of peripheral nerve lesions found in this report appears to be similar to those observed in other countries where median nerve compression at the carpal tunnel was the most common entrapment neuropathy and ulnar nerve lesions the most common traumatic nerve palsy[11].

Despite years of awareness amongst nurse educators sciatic nerve injuries still occur after intramuscular gluteal injections. Three of the four patients were young children and were left with permanent neurological deficits.

More than 20% of patients with carpal tunnel syndrome had symptoms for longer than a year and 20% had weakness of the hand which may be irreversible even after surgical release[12]. It is clear that awareness amongst the medical community of this common problem could be improved in order to prevent morbidity and disability.

Carpal tunnel syndrome is reported to occur more frequently in polyneuropathies[13]. One of the individuals with a sensorimotor polyneuropathy was known to be HIV positive. The HIV status of the others was not known. The finding of carpal tunnel syndrome in patients with symptoms remote from the site of compression is interesting. Patients complained of chest and breast, or arm and leg pain on the affected side and only admitted to arm and hand pain on closer questioning. The cause of this radiation of pain in uncertain but, speculatively, may be non-organic amplification of symptoms.

In 43 patients with signs and symptoms of carpal tunnel syndrome the diagnosis was excluded on electrophysiological grounds. In 23 of these cervical root irritation was diagnosed (19 on electrophysiological grounds and 4 on clinical grounds). In the remaining 20 patients no definite diagnosis was reached after the neurological examination and neurophysiological tests proved normal. Somatization appeared to be responsible for the symptoms in 4 of the 20 patients.

In patients with traumatic nerve lesions, electrophysiological testing indicated that recovery was likely to occur in more than two thirds. This was based on finding incomplete nerve lesions or ongoing re-innervation of muscle. (We were, however, unable to verify this prediction because of poor follow up). This implies that surgical exploration may not be immediately indicated in this group. As regards complete nerve lesions, approximately a year from time of injury should be allowed for nerve regeneration and re-innervation to take place before informing the patient than any further recovery is unlikely.

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