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Assessment of Recovery from Burn Related Neuropathy by Electrodiagnostic Testing

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Abstract

The purpose of this study was to investigate the recovery of burn related neuropathies by electrodiagnostic testing. Burn patients who presented to an American Burn Association verified burn center were interviewed and examined for clinical evidence of peripheral neuropathies by a physiatrist. Patients whom consented to participate were tested for electrodiagnostic evidence of peripheral neuropathy. Repeated studies were performed to assess for evidence of recovery.

A total of 370 patients were screened. 36 (9.73%) patients had clinical evidence of neuropathy. Eighteen male patients with a mean total body surface area burn of 42% had nerve conduction studies performed. Etiologies of the injuries included 8 flame, 8 electrical and 3 others. Seventy three nerve conduction studies were performed and 58 of the tests were abnormal. The most commonly affected nerve was the median sensory (10). For patients with repeated tests, the mean time between tests was 169 days (SD 140 days). There was a significant difference between the initial and follow up test (McNemar's change test p=0.009). In subset analysis of motor and sensory abnormalities, there was no significant difference (p=0.07). The most common neuropathy identified in this cohort was the median sensory. Overall, there was improvement in the nerve conduction abnormalities examined. This study suggests that the prognosis for recovery after burn related neuropathy is good.

Keywords

burn; neuropathy; nerve conduction studies; prognosis

Introduction

Burn related peripheral neuropathies are a well recognized complication of burn injuries. Burn related peripheral neuropathies are associated with both high and low voltage electrical injuries, a history of alcohol abuse, injuries requiring intensive care unit stay and increasing age. [1] The incidence of burn related peripheral neuropathy has been estimated between 11 and 41%. [1,2] The clinical and electrodiagnostic pattern of burn related peripheral neuropathy has been described as similar to that seen in mononeuritis multiplex.[3] Mononeuritis multiplex is an asymmetric disorder involving both motor and sensory peripheral nerves including pathology at sites not normally associated with compression. Mononeuritis multiplex is associated with several disorders usually related to vasculitis such as rheumatoid arthritis, diabetes and systemic lupus erythematosus. [4] In these other conditions leading to mononeuritis multiplex, controlling the underlying inflammatory disorder may result in improvements in the neuropathy. [5,6]

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Despite evidence that burn related peripheral neuropathies are prevalent in burn survivors, the prognosis for recovery has not been well described. This study describes the pattern of natural recovery from burn related peripheral neuropathies as evaluated by serial nerve conduction studies.

Methods

A consultant physiatrist screened 370 potential subjects from all admissions to an American Burn Association verified burn center. Subjects were assessed for symptoms and signs of peripheral neuropathies including anaesthesia, paraesthesias, and muscle weakness. Interviews and physical examinations were performed in an inpatient burn treatment center. Inclusion and exclusion criteria are summarized in table 1. Informed consent for electrodiagnostic testing was obtained from nineteen subjects and eighteen underwent electrodiagnostic testing. Nerve conduction tests were performed at the time of hospital discharge by a consultant physiatrist with training in electrodiagnostic medicine. Patients were asked to return post discharge for repeat nerve conduction studies. The nerve conduction study data was compared to population normal values and dichotomized as normal if between two standard deviations from the normal amplitude and latency or abnormal if less than two standard deviations from the acceptable normal values. [7–21] Follow up tests were examined for electrodiagnostic evidence of improvement in latency, amplitude or both. Alpha was set at 0.05. The overall effect was tested using McNemar's change test; a paired statistical test of changes in proportions. Data was entered in to an Excel spreadsheet and analyzed using the Simple Interactive Statistical Analysis program.

Results

A total of 370 subjects were screened. Thirty-six subjects (10.2%) were identified with clinical evidence of peripheral neuropathy. Nineteen male patients with a mean age of 33 years and an average total body surface area (TBSA) burn of 42% consented to the serial electrodiagnostic examinations One patient was excluded by an investigator because of a change in medical status between consent and testing. The etiologies of injury included eight flame, seven electrical, one grease, one chemical and one case of hot fume burns. A total of 73 nerve conduction tests were performed. Fifty-eight tests were abnormal. The most frequently abnormal nerve tested was the median sensory nerve (10), followed by the ulnar sensory nerve (9). In all patients with clinical symptoms, there was electrodiagnostic evidence of neuropathy. All patients except for one had both sensory and motor nerve function involvement in at least two peripheral nerves. The one patient who had an isolated median motor axonal neuropathy had an electrical injury including a contact point in the same extremity.

Of the original subjects, eight completed at least one follow up examination. Repeat data was collected on 45 nerves from these subjects. Four subjects refused the repeated tests because they found them too painful or felt they were unnecessary, 3 patients could not be contacted for a repeat test and the remaining subjects did not show for their follow up testing. For patients with serial tests, the mean length of time between the initial injury and first nerve conduction tests was 169 days (SD 140 days). The timing of the initial and follow up tests is summarized in table 2. In total nerve conduction tests there was a significant difference between the initial and follow up test between abnormal to normal. This was demonstrated by a significant result from McNemar's change test ($p=0.009$ with Yate's continuity correction for small sample size), which is a paired non parametric test. Table 3 summarizes the initial and follow up nerve conduction data, normal values and classification of each nerve as normal, abnormal or improved for those subjects with repeated studies. In subset analysis of motor and sensory abnormalities, there was not a significant difference noted between initial and follow up tests ($p=0.07$), which may have been due to the small sample sizes in some cells. None of the patients

studies with clinical and electrodiagnostic evidence of neuropathy had an intervening surgical procedure such as a nerve decompression surgery.

Discussion

The findings of this study are consistent with other reported incidences of burn related neuropathy of approximately 10% of people admitted to hospital with burn injury.[1,22] This study also considered the electrodiagnostic evidence for recovery by nerve conduction studies and in a small sample of subjects, the prognosis for spontaneous natural recovery appears positive.

The involvement of both sensory and motor peripheral nerves in all extremities is similar to the electrodiagnostic pattern seen in some disorders associated with mononeuritis multiplex such as Churg-Strauss syndrome and Wegener's granulomatosis.[23] However, this study did not include any nerve biopsies to examine any cellular inflammatory infiltrate around the vasa nervorum. Additionally, there was a trend towards improvement, which would not be typical in mononeuritis multiplex associated with vasculitidies.[24]

A potential source of error in the study exists because the subjects studied had burn and electrical injuries in the affected and studied extremities that included varying amounts of scarred skin. Furthermore, all of the subjects with repeated nerve conduction studies had at least one surgical procedure in the region associated with neuropathy prior to their first test (Table 2). It is possible that a peripheral nerve could be damaged through direct trauma during surgery or through compression during tourniquet use. Furthermore, skin thickness negatively correlates with amplitude in nerve conduction studies, which may have lead to false positive nerve conduction tests in patients with significant scarring.[25]

Additionally, the attrition of patients because of refusing repeated studies may limit the representativeness of the data for recovery, but reinforces that the nerve conduction studies are time consuming and potentially uncomfortable.

However, burn related neuropathies from both thermal and electrical injury are well recognized complications of burn injury.[1–3,26–30] There appear to be both systemic and local effects of burn injury on peripheral nerve as demonstrated in rat models where axonal patterns of neuropathy in limbs distant to the burn injury have been described.22 Furthermore, early excision of the burn wound appears to ablate these neurologic changes in these rodent models.[31] This pattern of neuropathy has also been observed in humans with severe inflammatory response syndrome associated with a wide variety of conditions from cardio-vascular surgery to chronic respiratory diseases.[32]

In conclusion, burn related neuropathy was identified in 10% of subjects in this sample. There was electrodiagnostic evidence of natural recovery from burn related neuropathies from both flame and electrical etiologies. Further studies considering both the electrodiagnostic characteristics and cellular response to injury will be helpful in understanding this disorder.

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Table 1

Patient Enrollment Criteria

Inclusion	Exclusion
• Major burn requiring hospitalization • Clinical signs / symptoms of neuropathy • Signed informed consent	• History of alcohol abuse • History of pre-morbid diabetes mellitus, uremia, neuropathy or myopathy • History of taking known neurotoxic drugs prior to admission • Skin loss due to Stevens- Johnson Syndrome, toxic epidermal necrolysis or necrotizing fasciitis

Table 2

Subject ID	Region with neuropathy burned?	Surgery in region with neuropathy?	TBSA burned	Etiology	Days until first test	Days between first and follow up test
A	Yes	Yes. Tangential excision, autograft	31	Flame	52	157
B	Yes	Yes. Tangential excision, heterograft, autograft	32	Electrical	60	247
C	Yes	Yes. Saphenous cut down, tangential excision, autograft	63	Flame	71	337
D	Yes	Yes. Tangential excision, autograft	81	Chemical	397	310
E	Yes	Yes. Tangential excision and autograft	24	Grease	329	477
F	Yes	Yes. Excision and autograft	89	Flame	209	365
G	Yes	Yes. Fasciotomy, suture closure, no graft	27	Electrical	24	96
H	Yes	Yes. Tangential excision, autograft	90	Flame	210	180

Table 3

Subject ID	Clinical features	Nerve tested	Values at first test (ms=milliseconds, uV= microVolts, mV=milliVolts)	Values at second test	Limits of normal	Classification of nerve on first test	Classification of nerve on follow up test
A	Right fourth and fifth digit numbness, weakness in grip Resolved at follow up test						
		Right median sensory	Peak latency 3.7 ms Peak to peak amplitude 27.2 uV	Peak latency 3.2 ms Peak to peak amplitude 27.4 uV	Peak latency <4.0 ms Peak to peak amplitude >27 uV	Normal	Normal
		Left median sensory	Peak latency 3.9 ms Peak to peak amplitude 26.7 uV	Peak latency 3.5 ms Peak to peak amplitude 32.1 uV	Peak latency <4.0 ms Peak to peak amplitude >27 uV	Abnormal	Normal
		Right ulnar sensory	Peak latency 3.5 ms Peak to peak amplitude 9.7 uV	Peak latency 3.4 ms Peak to peak amplitude 12.7 uV	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal: improved
		Left ulnar sensory	Peak latency 3.8 ms Peak to peak amplitude 7.5 uV	Peak latency 3.0 ms Peak to peak amplitude 23.6 uV	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal: improved
		Right median motor	Onset latency 4.2 ms Amplitude 8.2 mV	Onset latency 3.7 ms Amplitude 13.6 mV	Onset latency <4.7 ms Amplitude >3.0 mV	Normal	Normal
		Left median motor	Onset latency 4.2 ms Amplitude 3.2 mV	Onset latency 3.75 ms Amplitude 11.9mV	Onset latency <4.7 ms Amplitude >3.0 mV	Normal	Normal
		Right ulnar motor	Onset latency 3.85 ms Amplitude 3.9 mV	Onset latency 2.95 ms Amplitude 6.9 mV	Onset latency <3.6 ms Amplitude >7.4 mV	Abnormal	Abnormal: improved
		Left ulnar motor to abductor digiti minimi	Onset latency 2.75 ms Amplitude 8.1 mV	Onset latency 2.60 ms Amplitude 10.4 mV	Onset latency <3.6 ms Amplitude >7.4 mV	Normal	Normal
B	Left arm numbness below elbow and in hand						
		Left median motor	Not recordable	Onset latency 6.45 ms Amplitude 8.3 mV	Onset latency <4.7 ms Amplitude >3.0 mV	Abnormal	Abnormal: improved
		Left ulnar motor	Not recordable	Not recordable		Abnormal	Abnormal: no change
		Left median sensory	Not recordable	Peak latency 5.2 ms Peak to peak amplitude 17 uV	Peak latency <4.0 ms Peak to peak amplitude >27 uV	Abnormal	Abnormal: improved
		Left ulnar sensory	Not recordable	Not recordable		Abnormal	Abnormal: no change
C	Both feet numb Ankle dorsiflexion weakness						
		Right superficial radial sensory	Peak latency 3.5 ms Baseline to peak amplitude 7.8 uV	Test not performed	Latency <3.3 ms Amplitude >13 uV	Abnormal	Cannot classify: excluded from change analysis
		Right sural sensory	Peak latency 3.8 ms Peak amplitude 5 uV	Peak latency 3.88 ms Peak amplitude 3.17 uV	Latency <3.75 ms Amp > 5	Abnormal	Abnormal
		Left sural sensory	Not recordable	Peak latency 6.78 ms Amplitude 1.59 uV	Latency <3.75 ms Amplitude > 5 uV	Abnormal	Abnormal: improved
		Right superficial peroneal sensory	Not recordable	Not recordable	Latency <3.0 ms Amplitude >4 uV	Abnormal	Abnormal
		Left superficial peroneal sensory	Not recordable	Not recordable	Latency <3.0 ms Amplitude >4uV	Abnormal	Abnormal
		Right peroneal motor to tibialis anterior	Onset latency 2.5 (fibular head) Base to peak amp 1.32 m	Onset latency 3.19 ms Onset to peak amplitude 3.75 mV	Onset latency <4.9 msec Amplitude >1.7 mV	Abnormal	Abnormal: improved
		Left peroneal motor to tibialis anterior	Onset latency 2.34 (fib head) Base to peak amp 2.23 mV	Not recordable	Onset latency <4.9 msec Amplitude >1.7 mV	Normal	Abnormal
		Right tibial motor	Onset latency 4.4 ms Amplitude 0.49 mV	Onset latency 4.22 ms Amplitude 1.63 mV	Onset latency <2.9 ms Amplitude >6.3 mV	Abnormal	Abnormal: improved
D	Numbness on dorsal and plantar left foot						

Subject ID	Clinical features	Nerve tested	Values at first test (ms=milliseconds, uV= microVolts, mV=milliVolts)	Values at second test	Limits of normal	Classification of nerve on first test	Classification of nerve on follow up test
	Weakness ankle dorsiflexion and flexor hallicus longus						
		Left sural sensory	Not recordable	Not recordable	Latency <3.75 ms Amplitude > 5uV	Abnormal	Abnormal
		Left superficial peroneal sensory	Not recordable	Not recordable	Latency <3.0 ms Amplitude >4 uV	Abnormal	Abnormal
		Left peroneal motor to extensor digitorum brevis	Onset latency 4.2 ms Baseline to Peak Amplitude 2.5 mV	Onset latency 4.2 ms Baseline to Peak amplitude 4.7	Onset latency <6.4 ms Amplitude >1.1 mV	Normal	Normal
		Left tibial motor	Onset latency 3.45 ms Baseline to peak amplitude 3.1 mV	Onset latency 4.5 ms Baseline to peak amplitude 3.0 mV	Onset latency <2.9 ms Amplitude >6.3 mV	Abnormal	Abnormal
E	Not noted						
		Left median sensory	Not recordable	Peak latency 4.78 ms Amplitude 27.87 uV	Peak latency <4.0 ms Peak to peak amplitude >27 uV	Abnormal	Abnormal: improved
		Left median motor	Onset latency 4.95 ms Amplitude 0.9 mV	Onset latency 4.73 ms Amplitude 12.16mV	Onset latency <4.7 ms Amplitude >3.0 mV	Abnormal	Abnormal: improved
F	Bilateral ankle dorsiflexion weakness, bilateral hand intrinsic weakness						
		Right median sensory	Not recordable	Not recordable	Peak latency <4.0 ms Peak to peak amplitude >27 uV	Abnormal	Abnormal
		Left median sensory	Not recordable	Not recordable	Peak latency <4.0 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal
		Left ulnar sensory	Not recordable	Not recordable	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal
		Right ulnar sensory	Not recordable	Not recordable	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal
		Right median motor	Not recordable	Not recordable	Onset latency >4.7 ms Amplitude >3.0 mV	Abnormal	Abnormal
		Left median motor	Not recordable	Not recordable	Onset latency <4.7 ms Amplitude >3.0 mV	Abnormal	Abnormal
		Right ulnar motor	Not recordable	Not recordable	Onset latency <3.6 ms Amplitude >7.4 mV	Abnormal	Abnormal
		Right peroneal motor to extensor digitorum brevis	Not recordable	Not recordable	Onset latency <6.4 ms Amplitude >1.1 mV	Abnormal	Abnormal
G	Parasthesias left hand digits 2–5						
		Left median sensory	Not recordable	Onset 3.10 ms Peak to peak amplitude 10.6	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal: Improved
		Left median motor	Onset latency 4.05 Amplitude 6.2 mV	Onset latency 3.90 Amplitude 11.0 mV	Onset latency <4.7 ms Amplitude >3.0 mV	Normal	Normal
H	Parasthesias right upper and lower extremity						
		Right median motor	No response	No response	Onset latency <4.7 ms Amplitude >3.0 mV	Abnormal	Abnormal
		Left median motor	Latency 4.3 msec Amplitude 1.9 mV	No response	Onset latency <4.7 ms Amplitude >3.0 mV	Abnormal	Abnormal
		Right ulnar motor	No response	Latency 4.4 msec	Onset latency <3.6 ms	Abnormal	Abnormal: improved

Subject ID	Clinical features	Nerve tested	Values at first test (ms=milliseconds, uV= microVolts, mV=milliVolts)	Values at second test	Limits of normal	Classification of nerve on first test	Classification of nerve on follow up test
				Amplitude 4.4 mV	Amplitude >7.4 mV		
		Right radial motor	No response	No response	Latency <1.72 ms Amplitude >4.24 mV	Abnormal	Abnormal
		Right peroneal motor to extensor digitorum brevis	No response	No response	Onset latency <6.4 ms Amplitude 1.1 mV	Abnormal	Abnormal
		Right peroneal motor to tibialis anterior	Latency 8.5 ms Amp 0.175 mV	No response	Onset latency <4.9 msec Amplitude >1.7 mV	Abnormal	Abnormal
		Right tibial motor to abductor hallicus	No response	No response	Onset latency <2.9 ms Amplitude >6.3 mV	Abnormal	Abnormal
		Right sural sensory	No response	No response	Latency <3.75 ms Amplitude > 5uV	Abnormal	Abnormal
		Right ulnar sensory	Latency 2.2 msec Amplitude 13.7 uV	No response	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Normal	Abnormal
		Right median sensory	No response	No response	Peak latency <2.5 ms Peak to peak amplitude >13 uV	Abnormal	Abnormal