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Peripheral neuropathy after burn injury

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Abstract. – OBJECTIVES: Peripheral neuropathy is a well-documented disabling sequela of major burn injury. These lesions are associated with both thermal and electrical injuries that may be frequently undiagnosed or overlooked in clinical settings. The purpose of this study was to evaluate the prevalence of burn-related neuropathy in our database and to investigate the clinical correlates for both mononeuropathy and generalized peripheral polyneuropathy.

PATIENTS AND METHODS: Out of 648 burn patients, admitted to our clinic forty-seven burn patients with the diagnosis of peripheral neuropathy were evaluated retrospectively. The demographic and clinical data collected were gender, age, degree, site and percent surface area of burn, type of burn, and the results of electrodiagnostic examination, including electromyography and nerve conduction assessments and associated pathology if existed.

RESULTS: Peripheral neuropathy is the most frequent disabling neuromuscular complication of burn, that may be undiagnosed or overlooked. In current study, peripheral neuropathy associated with burn all of our patients were identified by electrodiagnostic study. After treatment in Burn Unit, clinical and electrodiagnostic studies were applied. Motor and sensory distal latencies were prolonged and sensory nerve action potentials reduced in amplitude.

CONCLUSIONS: The findings of our study have shown that polyneuropathies and axonal neuropathy were more frequent than mononeuropathy and demyelination.

Key Words:
Burn, Neuropathy, Risk factor, Electrodiagnostic study, Axonomtesis.

Introduction

Peripheral neuropathy is a well-documented disabling sequel of major burn injury. These lesions are associated with both thermal and electrical injuries It may be frequently undiagnosed or overlooked in clinical settings. The frequency varies widely, from 2% to 84% of patients, based on the methodology of each study[1-3]. Describing the cause is also difficult due to the complex metabolic nature of burn injury, subsequent use of neurotoxic antibiotics, and the numerous iatrogenic causes of neuropathy. The purpose of this study was to evaluate the prevalence of burn-related neuropathy in our database and to investigate the clinical correlates for both mononeuropathy and generalized peripheral polyneuropathy.

Patients and Methods

During the 6 year period from January 2004 to September 2010, 648 burn patients were admitted to the the Clinical Neurology Unit, Dicle University Hospital, Diyarbakir. Forty-seven burn patients with the diagnosis of peripheral neuropathy were evaluated retrospectively.

We excluded patients with preexisting neurological problems, including stroke, traumatic brain injury, peripheral neuropathy or other medical conditions that could lead to neuropathy (diabetes mellitus, collagen diseases, uremia, and alcohol abuse) and history of taking known neurotoxic drugs before admission, as well as patients with previous burns, contracture and skin loss.

The demographic and clinical data collected were gender, age, degree, site and percent surface area of burn, type of burn, the results of electrodiagnostic examination, including electromyography and nerve conduction assessments and associated pathology if existed.

Electrodiagnostic examination included electromyography and motor nerve conduction velocities (MNCV) of burn extremities. This was performed using the "a NIHON KOHDEN Measuring System Model MEB-9102K Power input 150 VA EP/EMG machine (Tokyo, Japan). Motor nerve conduction velocities were assessed using

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standard procedures with concentric needle electrodes. A pulse of 0.2 ms duration, at the rate of 1 per second at supramaximal intensity was used for conduction studies

Peripheral neuropathies were scored as demyelinating neuropathy, axonotmesis and entrapment neuropathy.

The criteria for demyelinating neuropathy were; a marked prolongation of terminal latency (more than 50% of normal control values), slow nerve conduction velocity (NCV) (more than 40% below the normal mean) with normal amplitude of compound muscle action potential (CMAP), abnormal shape of CMAP (multiple phases or prolonged time) and innervated muscles with the same neuropathic features as those described for axonal degeneration. The criteria for axonotmesis were ; a reduction in the amplitude of CMAP (two or more standard deviations below the mean of normal values) but with normal shape and duration, normal NCV, and distal latency or minimal disturbance. Also noted were denervation potential, such as fibrillation or positive sharp wave, increased amplitude duration of motor unit potentials, and a reduction of recruitment in the muscle innervated by the affected nerve.

Entrapment neuropathy was defined as focal slowing in a nerve across the site of entrapment by 20 per cent below the lower limit of control motor conduction value with a reduction of CMAP amplitude by -2 SD of the control value'.

Peripheral neuropathies were scored as mono- or polyneuropathy according to the number of involved nerves.

Statistical Analysis

Descriptive statistics, (e.g. mean, standard deviation, frequencies, percentages) were calculated. Results for quantitative variables are given as mean ± standard deviation and parametric tests (Student's *t* and Chi-square tests) were used to identify statistical differences. *p*-values below 0.05 were considered to indicate statistical differences.

Results

Fortty-nine of 648 patient charts were reviewed, two were excluded because of a lack of follow-up notes. There were 26 (55%) men and 21 (45 %) women. The mean age (±SD) of the patients was 35.60±19.23 (6-68) years. 15 (31.9%) patients had polyneuropathy and 32 (68.1%) had mononeuropathy**.** Patients with polyneuropathy frequently had axonotmesis. The proportion of patients whose burns resulted in axonotmesis was significantly greater than those resulting in demyelinating neuropathy for patients with larger area of burns.

The most frequent etiology of mononeuropathy was low-voltage electrical injury (n=16 50%). The clinical characteristics of the patients are summarized in Table I.

There were 27 (57%) burns caused by thermal reasons, and 20 (43%) burns caused by electricity. 16 (34%) were low voltage (< 1000 V) electrical injuries and 4 (8%) were high voltage (> 1000 V) ones. Thermal injury was the most frequent type of injury. Low voltage electrical injury was more frequent in males. The clinical characteristics of burn patients according to the cause are summarized in Table II.

The upper limbs were the most frequent site of burn (n=37, 79%). There were no significant gender differences among burn sites.

The mean total body surface area burned was 22.17±14.76% (4-54%). Injuries involving less than 10% of the body surface were observed in of 14 (29.78%) assessable patients, injuries involving 10-19% of body surface were observed in 11 patients (23.40%), 20-49% were in 19 patients (40.42%), and 50% or more were in 3 patients (6.38%).

Axonotmesis was present in 30 (63.8%), demyelinating in 14 (29.8%), entrapment neuropathy in three (6.4%) patients. Polyneuropathy was present in 16 patients (45.7%) and mononeuropathy in 13 (37.1%) (Figure 1). No significant statistical difference was found between electrodiagnostic results.

The proportion of neuropathy types (demyelinating versus axonotmesis) did not differ significantly between groups of patients with electrical and flame injuries (Figure 2).

Discussion

Peripheral neuropathy is the most frequent disabling neuromuscular complication of burn, that may be undiagnosed or overlooked[1-3].

In current study, peripheral neuropathy associated with burn all of our patients was identified by electrodiagnostic study. After treatment in Burn Unit, clinical and electrodiagnostic studies were applied. Motor and sensory distal latencies were prolonged and sensory nerve action potentials reduced in amplitude.

Table I. The clinical characteristics of our patients with polyneuropathy and mononeuropathy patients.

Clinical characteristics	Polyneuropathy n=15 (31.9%)	Mononeuropathy n=32 (68.1%)	p
Age (years; mean ± SD)	29.67 ± 20.24	38.38 ± 18.40	0.169
Gender			
Male	7 (46.7%)	19 (59.4%)	0.533
Female	8 (53.3%)	13 (40.6%)	
Cause of burn			
Thermal	13 (86.7%)	14 (43.7%)	0.010
Low voltage electrical	–	16 (50%)	0.001
High voltage electrical	2 (13.3%)	2 (6.3%)	0.583
Localization of burn			
Upper extremity	4 (26.7%)	19 (59.4%)	0.060
Lower extremity	7 (46.7%)	12 (37.5%)	0.711
Upper + Lower	4 (26.7%)	1 (3.1%)	0.030
Type of neuropathy			
Axonotmesis	14 (93.3%)	16 (50%)	0.004
Demyelination	1 (6.7%)	13 (40.6%)	0.020
Entrapment	–	3 (9.4%)	0.541
TBSA (%; mean ± SD)	37.07 ± 12.47	15.19 ± 9.77	0.000

TBSA: Total Body Surface Area.

Our study found a retrospective prevalence of 7% based on electrodiagnostic and clinical examination. This finding is inconsistent with other reported percentages of burn-related neuropathy. Marquez[3] in his retrospective chart review found 2%, where as a prospective study done by Henderson[4] reported that the frequency was 15%. The difference of prevalence of peripheral neuropathy in previous studies may be due to variations in methodology.

Selection of only symptomatic patients, such as those with muscle weakness or sensory loss, may lead to overlooking peripheral neuropathy in many patients which could have been detected through electrodiagnostic study. The small sample size in the studies may also be responsible for the different results[5,6].

The findings of our study have shown that polyneuropathies and axonal neuropathy were more frequent than mononeuropathy and demyelination.

Polyneuropathies were due to mainly thermal burns covering greater than 30% of the total body surface area and associated with axonotmesis than

Table II. The clinical characteristics of burn patients according to the cause.

Clinical characteristics	Thermal n=27 (57.4%)	Electrical injury n=20 (42.6%)	p value
Age (years; mean±SD)	36.30 ± 21.60	34.65 ± 15.96	0.765
Gender			
Male	15 (55.6%)	11 (55%)	1.000
Female	12 (44.4%)	9 (45%)	
Neuropathy			
Poly-	13 (48.1%)	2 (10%)	0.010
Mono-	14 (51.9%)	18 (90%)	
Localization of burn			
Upper extremity	5 (18.5%)	18 (90%)	0.000
Lower extremity	17 (63%)	2 (10%)	0.000
Upper + Lower	5 (18.5%)	–	0.063
Type of Neuropathy			
Axonotmesis	18 (66.7%)	12 (60%)	0.761
Demyelination	6 (22.2%)	8 (40%)	0.214
Entrapment	3 (11.1%)	–	0.251
TBSA (%; mean±SD)	30.78 ± 13.30	10.55 ± 6.17	0.000

TBSA: Total Body Surface Area.

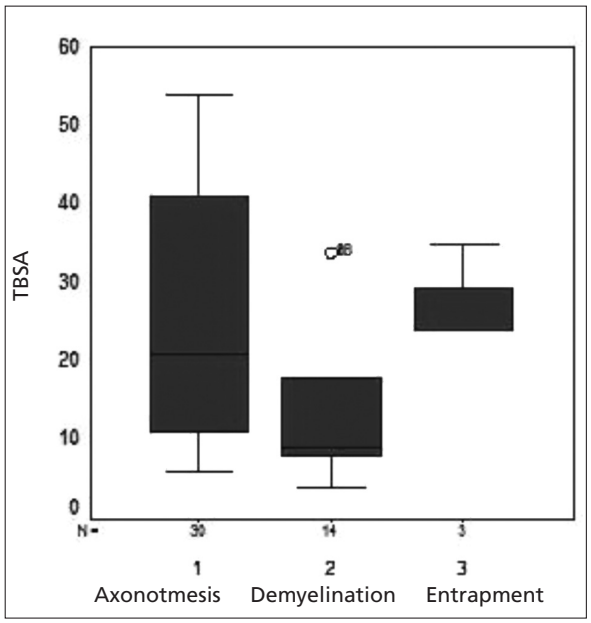

Figure 1. Burn percentages of total body surface area depending on the type of neuropathy.

with myelin sheath involvement. These findings were consistent with the literature[3,7,8].

The etiology of burn neuropathy is unclear. Direct thermal injury, vascular occlusion of the vasa nervorum, postinjury edema, compressive nerve entrapments are several possible mechanisms that have been suggested.

Experimental animal studies on burn, have shown that functional and morphological deficits in peripheral nerve axons after burn is seen. Degeneration of the neural plate disintegration of myelin sheath, and wallerian degeneration of axon have been demonstrated. Significant reduction in motor and sensory conduction velocities in electrodiagnostic studies and decreased mean caliber of large axons in histologic evaluation confirm the assessment[9,10].

Blood vessel embolism occur within the injured nerve due to protein denaturation and clumping within the vessel lumina. Increased platelet aggregation, accelerated fibrin deposition and clot formation may contribute to the occlusion. Alteration in the distribution of fluids and electrolytes, together with prevalent activation of inflammation lead to edema formation[11-15].

Entrapment neuropathy is seen in 3 (6.4%) patients of this study, all due to thermal reasons and presented itself as mononeuropathy. Entrapment neuropathy may occur because of nerve compression caused by postinjury edema poor positioning, bulky dressings, prolonged tourniquet times, scarring and contracture after the closure of the wound. Appropriate positioning of the patients to avoid nerve compression includes avoiding prolonged elbow flexion, frog legging (positioning the lower extremities in neutral rotation and knee extension), and prone positioning with arms overhead[16-18].

The prevalence electrical injury in current study was 3%, which was lower than the value stated by Hussmann et al[19]. Mononeuropathies were mainly due to electrical injury (n=18, 90%). Direct electrical injury to the nerve or neuropathy from postinjury edema may be the cause. Electrical injury has been shown to correlate with mononeuropathy and the results of current study is consistent with the literature[16]. Direct electrical injury to the nerve or neuropathy from postinjury edema may be potential cause.

The limitation of this study is retrospective design and the probability of false positive nerve conduction tests in patients with significant scarring[20].

Future electrodiagnostic studies for longer periods are necessary to clarify characteristics of this disorder and evaluate the underlying metabolic factors that contribute to neuropathy.

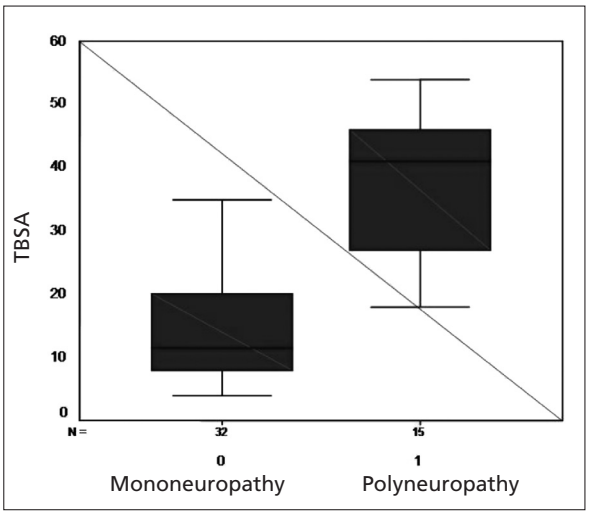

Figure 2. The percentages of patients with poly- and mononeuropathies

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