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ASSESSMENT OF PERIPHERAL NERVE INJURIES BY ELECTRONEUROMYOGRAPHY AFTER KAHRAMANMARAŞ EARTHQUAKES (ON FEBRUARY 6, 2023)

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Abstract

Objectives: This study aimed to assess the peripheral nerve injuries among survivors after the two consecutive Kahramanmaraş Earthquakes with magnitudes of 7.7 Mw and 7.6 Mw.

Materials and Methods: In the study, the medical records and electroneuromyographic findings of 94 earthquake victims who referred to Ankara City Hospital and underwent electroneuromyography analysis were analyzed.

Results: Peripheral nerve damage was most common in the lower extremities. The presence of crush syndrome was closely associated with peripheral nerve damage (p=0.049).

Conclusion: Peripheral nerve injuries require special attention as they may cause serious disability.

Keywords: Earthquake, electroneuromyography, peripheral nerve injury

Introduction

On February 6, 2023, two earthquakes, the first with a magnitude of 7.7 Mw in Kahramanmaraş Pazarcık at 04:17 and the second with a magnitude of 7.6 Mw in Elbistan approximately nine hours later, caused massive destruction over an area of 108,812 km^2 encompassing 11 provinces. According to AFAD reports, there were 50783 fatalities, 115,353 injuries, and 37,984 collapsed buildings.[1,2]

There were a high number of casualties and injured people. The first of the two earthquakes occurred in the early morning hours while most people were asleep, and the second occurred approximately 9 hours later and was unexpected. Although subsequent earthquakes of lesser intensity are expected, the second earthquake was more severe, affecting many of the people who survived the first one while they were entering their damaged houses to collect their belongings.

Reaching the earthquake survivors underneath the building wreckage also took longer than expected due to the size of the affected area and the extent of the quake damage. Survivors were trapped under debris for more than 296 hours after the earthquake. Being trapped under debris for a prolonged period may have increased the risk of complications.

Survivors extracted from debris entrapment in the acute period were referred to 3rd-level hospitals to undergo initial interventions for critical and urgent medical conditions such as traumatic fractures, compartment syndrome, organ injuries, crush syndrome, and head trauma (skull fracture). However, the earthquakes also significantly damaged regional hospitals. The surviving patients who were referred to Ankara City Hospital—approximately 600 km from the earthquake epicenter—were treated in the relevant departments and underwent treatment for peripheral nerve injuries in our electrophysiology laboratory four days after the earthquake. While head trauma, visceral organ injuries, fractures, crush syndrome, and organ failures were the focus of treatment and care services due to their tendency to result in patient death in the initial days and weeks after the earthquake, peripheral nerve injuries became one of the primary reasons for post-survival disabilities as time progressed.

The aim of the current study was to assess the demographic characteristics and early electroneuromyography (ENMG) findings of earthquake survivors diagnosed with peripheral nerve injury.

Materials and Methods

The current research is an observational descriptive study approved by the ethics committee of Ankara City Hospital (04.26.2023/E1-23-3481). Written consent was obtained from all participants. There were no patients with impaired consciousness that would prevent confirmation. The consent of 9 pediatric patients included in the study was obtained from their legal guardians. Following the earthquake, 94 patients transferred to Ankara City Hospital and treated in the relevant clinics were referred to the ENMG laboratory to diagnose their peripheral nerve injuries. These patients were also followed up in orthopedic, plastic surgery, internal medicine, and general intensive care clinics for several complaints, such as multiple traumas, compartment syndrome, and crush syndrome. The researchers reviewed the medical records of patients already referred for electrodiagnostic examination because they had motor loss and/or sensory symptoms in their extremities. A Keypoint (Alpine, Denmark) device was used for the ENMG examination.

Statistical analysis

The data were evaluated in the statistical package program IBM SPSS Statistics Standard Concurrent User V 26 (IBM Corp., Armonk, New York, USA). Descriptive statistics were given as the number of units (n), percentage (%), mean ± standard deviation (m±SD), median (M), minimum (min) and maximum (max) values. The normal distribution of the data of numerical variables was evaluated using the Shapiro-Wilk normality test. In comparisons regarding the number of damaged nerves, the Mann-Whitney U test was used since the data did not have a normal distribution. Relationships between variables were examined with Spearman's rho coefficient since the data did not provide a normal distribution. A value of $p<0.05$ was considered statistically significant.

Results

The 94 patients with extremity trauma were aged 7 to 66 years, with a mean age of 33 years. There were 45 male patients and 49 female patients. The average time spent under the rubble was 28 hours. Eleven people underwent electrodiagnostic examination within the first 20 days after the earthquake. Then, the number of cases examined in the first, second, and third months after the earthquake was 49, 17, and 17, respectively. The time between the ENMG test request and the testing date ranged from 1 to 12 days. The average number of damaged nerves detected by the ENMG test in patients was 3. The number of associated injuries, such as fractures, compartment syndrome, and crush syndrome, accompanying peripheral nerve damage, are summarized in Table 1. Sixteen of 44 patients with fractures underwent surgery. Compartment syndrome was present in 59 of 51 patient extremities. Fasciotomy was performed on 46 extremities of 37 of these patients. Thirty-three of the patients with crush syndrome needed hemodialysis.

Table 1. Demographic characteristics and injuries accompanying peripheral nerve damage

	$\bar{x}$±SD	*n (%)*
Age (year)	33±13.27	
Gender, Female **Male**		49 (52.13) 45 (47.87)
Time spent under the rubble (hours)	28.27±26.66	
Damaged nerve	3.04±2.21	
Concomitant injury		
Fracture		44 (46.80)
Compartment syndrome		51 (54.25)
Crush syndrome		48 (51.06)
Multiple injuries		44 (46.80)

Statistics are given as a number (n), percentage (%), $\bar{x}$: mean, SD: Standard deviation.

Considering 94 patients, there were nerve injuries in 129 extremities, 48 and 81 of which were upper and lower, respectively. There were also 114 peripheral nerve injuries, 35 localized in the upper extremity and 79 in the lower extremity, 33 plexus injuries—15 were brachial, 18 were lumbosacral—and six multiple cervical root avulsions. Table 2 displays the distribution of the injured nerves, crush syndrome, compartment syndrome, and their relationship with fracture. No relationship was detected between the number of damaged nerves and the duration of stay under the collapse (r=0.115, p=0.268).

When compared to the group in terms of the presence of crush syndrome, compartment syndrome and fracture, the presence of crush syndrome seems to be closely related to peripheral nerve damage (p=0.049). They are summarized in Table 3.

Although there were suggestive symptoms of peripheral nerve injury, the ENMG analysis of 36 extremities, 24 of which were lower and 12 of which were upper, could not be performed for the reasons listed in Table 4.

Table 2. Distribution of the injured nerves and concomitant other factors

Peripheral nerve injury	n	Partial axonal	Total axonal injury n	Crush syndrome n/%	Compartment syndrome (fasciotomy) n, %	Fracture n, %	Multiple concomitants n, %	Time spent under the rubble (hours) m (min-max)
Median nerve	8	4	4	2/25	5(4) 62.50%	0	1 12.50%	20 (5-49)
Ulnar nerve	13	8	5	7/53.84	6(4) 46.15	3 23.07	4 30.76	26 (1-49)
Radial nerve	11	5	6	5/45.45	5(3) 45.45	3 27.27	3 27.27	17 (1-49)
Axillary nerve	3	3	0	2/66.66	1 33.33	1 33.33	1 33.33	24 (4-40)
Femoral nerve	1	1	0	1/100	1 100		1 100	13
Obturator nerve	1	1	0	0/0	1(1) 100	1 100	1 100	12
Sciatic nerve	27	21	6	22/81.48	11(10) 40.74	4 14.81	10 37.03	31 (5-160)
Peroneal nerve	38	28	10	13/34.21	8 (6) 21.05	5 13.15	5 13.15	27 (1-96)
Tibial nerve	12	8	4	3/25	4(4) 33.33	2 16.66	0 0	12 (1-80)
Brachial plexus	15	12	3	8/53.33	5(4) 33.33	1 6.66	4 26.66	40 (1-160)
Lumbosacral plexus	18	11	7	12/66.66	4(3) 22.22	5 27.77	4 22.22	25 (2-72)
Cervical root avulsion	6	6	0	3/50	0	1 16.66	1 16.66	49 (2-160)

Table 3. Relationship of nerve injury to other accompanying injuries

Number of Damaged Nerves			
	$\bar{x}$±SD	*z*	*p value*
Victims with Crush syndrome	3.56±2.61	1.961	**0.049**
Victims without Crush syndrome	2.50±1.54		
Victims with Compartment syndrome	3.27±2.37	1.089	0.276
Victims without Compartment syndrome	2.76±1.99		
Victims with Fracture	3.36±2.43	1.256	0.209
Victims without Fracture	2.76±1.98		
Victims with Multiple injury	3.31±2.41	1.099	0.272
Victims without Multiple injuries	2.80±2.01		

$\bar{x}$: mean, SD: Standard deviation, *z*: Mann Whitney U test

Table 4. Reasons for exclusion

Edema	2 (5.55%)
Fasciotomy	23 (63.88%)
Plaster-external fixator	10 (27.77%)
Pain-intolerance	1 (2.77%)

Statistics are given as number (percentage %) values.

Discussion

The cross-sectional data gathered from 94 earthquake survivors through testing in the electrophysiology laboratory of the city hospital may add to the sparse literature on the electrophysiological findings of post-disaster peripheral nerve injuries; hence, it potentially put forth an approach for assessing peripheral nerve injuries. ENMG is an auxiliary diagnostic method delivering reliable data on the presence or absence of peripheral nerve injury, its severity, and extent, if any.

Peripheral nerve injuries may ensue from various types of traumatization, including lacerations, crushing, stretching, angulation, increased compression on the nerve, or prolonged external pressure for hours. Acute pressures may result in paranodal, segmental demyelination.[3] Demyelination also leads to conduction block, manifesting itself by weakness and sensory loss. Experimental studies of compression-based nerve injury revealed that the intensity and incidence rate of conduction block varies depending on the severity and duration of the compression. Such electrophysiological variations may be identifiable within 24 hours of injury.[4] Axonal injury may occur if the pressure and crushing are severe and lengthy or if the entire nerve is under compression. Hence, under pressure, the intraneural venous blood flow of the nerve is impaired, vascular permeability increases, the blood-nerve barrier becomes permeable, and endoneurial edema ensues. Nerve fiber dysfunction is triggered by an altered metabolic environment and increased intrafascicular pressure. This context leads to axon transport distortion. In acute compressive neuropathies, motor fibers are significantly more affected since they are less resistant than sensory nerves. Thin myelinated and unmyelinated fibers are affected when the pressure is so severe that it induces axonal degeneration of the thick myelinated nerves. Then, in addition to muscle weakness, severe sensory and autonomic dysfunction manifests.[3] When the axon is disrupted by severe crushing or acute stretching, Waller degeneration begins distally within 24 hours, and the axon and myelin sheath are fragmented. While these changes are concurrent, the distal part of the nerve still retains its ability to conduct impulses for the first few days.[5]

In addition to being trapped under building debris in earthquakes, some other reasons—regardless of severe earthquake damage—such as being trapped under overturned objects, being stuck between household items,

or stepping on broken and sharp objects on the floor while attempting to leave the house without shoes, [six] and pulling out extremities that have remained under the weight for hours and have become anesthetized[7] may typically result in peripheral nerve injury. It is also viable to consider long bone fractures and joint dislocations among the reasons. Even if there is no external pressure in a narrow space beneath the wreckage for an extended period, retaining a compelling flexion-extension posture that stretches the nerve may also result in nerve injury.[8]

In the aftermath of the 1999 Marmara Earthquake, Uzun et al. reported that the lower extremities of 80 patients were more severely injured than the upper extremities. They further noted that distal peripheral nerves in the same patient group were more injured than proximal peripheral nerves.[7] Another study focusing on surviving children in the earthquake documented that lower extremity distal peripheral nerve injuries were prevalent among children, similar to the adult group. The incidences of peripheral nerve injury and total axonal damage in the first ENMG analysis were higher in the pediatric age group than in the adult group.[9] Salimi et al. [six] also indicated that lower extremity injuries were common in people trapped under debris as a result of the Bam earthquake; however, fracture-based nerve injuries were more frequently humeral shaft fractures. They also reported that survivors sustained radial nerve injury more frequently than ordinarily predicted humeral shaft fractures. These significant injury rates might most likely be due to fractures sustained during rescue from wreckage or improper patient handling.[6]

In their research, in which they analyzed the incidence of peripheral nerve injury in 523 patients after the Wenchuan Earthquake, He et al.[8] scrutinized the variables associated with the nerve injury score. They revealed that the incidences of muscle, tissue, nerve, and vascular injury, compartment syndrome, and crush injury corresponded with nerve injury scores, whereas there was no relationship between open injury and nerve injury scores. They further reported the prevalence of sciatic nerve injury due to prolonged stay in passive body positions, stretching the nerve in a narrow space without direct external mechanical forces. This extensive case study disclosed that earthquake pressure injuries typically result from direct nerve compression, thereby causing nerve deformation. Direct nerve compression by an external object or compression or twisting of the nerve by a broken bone is among the causes of direct nerve compression. Apart from localized compression injuries that occur due to direct nerve compression, disruption of blood flow to the nerve due to crush and compartment syndrome or soft tissue injuries causing damage to the nerve vascular bed has also been reported among the causes of nerve injury.[8]

In their study in which they examined peripheral nerve palsy in 25 patients after the 1995 Hanshin-Awaji Earthquake, Yoshida et al.[10] reported that crush syndrome, compartment syndrome, prolonged nerve compression, and staying in a compelling posture in which the joint is in the forced flexion-extension and compressing the nerve were among the causes of nerve injury.

In our study, we detected single or multiple nerve injuries in 129 extremities of 94 patients. There were also 26 fractures and one joint dislocation coexisting with the nerve injury. Sixteen of the 26 fractured extremities—segmental fractures—were treated with surgery. Of the 129 extremities with nerve injury, 59 had compartment syndrome, and 51 of them were associated with nerve damage. Forty-six of the 59 extremities with compartment syndrome were treated with fasciotomy. There were fractures and compartment syndrome in ten extremities. Half of them, on the other hand, were patients with crush syndrome. Leaving aside the femoral and obturator nerve injuries, of which we saw one case each, multiple co-occurrences of crush syndrome, compartment syndrome and fractures were most present in individuals with ulnar nerve injuries in the upper extremity and in those with sciatic nerve injuries in the lower extremity. Peripheral nerve injury was detected more frequently in the lower extremities. The frequency of ulnar nerve injury in the upper extremity and peroneal nerve injury in the lower extremity was high.

The brachial plexus was the source of 15 of the 48 upper extremities with peripheral nerve injuries, corresponding to a high rate of approximately 1/3 (31.25%). The brachial plexus is highly vulnerable to neural injuries due to its superficial location and close proximity to skeletal structures. For instance, it could sustain traction injuries, become trapped and crushed between the clavicle and the ribs under it, or sustain damage from bone fragments, displaced muscles, or hematomas.[3] There was lumbosacral plexus injury in 18 (22.22%) of the 81 lower extremities with peripheral nerve injury. Traumatic injury to the lumbosacral plexus may occur in individuals with pelvic junction fractures, especially those with sacrum fractures.[3] Considering their relationship with the fracture, only one case of the 15 brachial plexus injuries was related to a clavicle fracture. Only five of the 18 individuals with lumbosacral plexopathy had a fracture of the sacrum. More than half of the patients with both brachial and lumbosacral plexus injuries were patients with crush syndrome (53.33% and 66.66%, respectively), most of whom also developed compartment syndrome.

Peroneal and sciatic injuries were the two most common nerve injuries identified in the lower extremity. Plexus damage was more common in the upper extremity, followed by ulnar nerve damage. In patients with crush syndrome, peripheral nerve damage was most frequently detected in the sciatic nerve. Approximately 81.48% of sciatic nerve injuries were concomitant with crush syndrome. Sciatic nerve damage was also seen most frequently in patients with compartment syndrome. In 31.81% of sciatic injuries, crush syndrome was accompanied by compartment syndrome, and in 9.09%, crush syndrome was accompanied by fracture. The ulnar nerve was most commonly affected in the upper extremity, and half of these patients also suffered coexisting crush syndrome, compartment syndrome, and fracture.

Peripheral nerve injuries may ensue concomitantly with fractures, fracture dislocations, and dislocations alone. Considering all injuries, five fibular, two tibial, a radial, a brachial plexus, and three lumbosacral plexus injuries were directly and solely related to fractures. However, since the study excluded some fractures and extremities

due to plaster, external fixation, and other factors, it is difficult to infer that these coexistences are distinct from those typically anticipated. The examination findings strongly suggested a potential peripheral nerve injury, even without the results of the electrodiagnostic analysis on 36 extremities. Open fasciotomy and the presence of a plaster cast-external fixator were the most typical reasons restricting ENMG analysis.

As mentioned above, sensorial nerves are more pressure-resistant than motor fibers. Sensory nerve injuries identified in the participant patients were concomitantly present with moderately severe motor nerve injury. The opposite also holds almost the same ratios, indicating that it is associated with the fact that crush injuries in participant patients were severe enough to affect the sensory and motor fibers significantly. Four patients had nearly all axonal injuries in the motor fibers that did not interfere without interfering with the sensory nerve. The first of these cases was about the radius fracture affecting the deep branch of the radial nerve; however, the other three were about tibial shaft fractures affecting the deep fibular nerve. Their sensory nerves were undamaged, whereas isolated motor nerves were injured.

Considering the case series in our study, the findings indicated that earthquake-based peripheral nerve injuries were not associated with direct nerve injury or deformation but were associated with crush and compartment syndromes. While the nerves of the bodily organs underneath the wreckage initially remain under pressure due to external forces, there is a potential for ischemic injury to the nerve due to hypoperfusion of the neural tissue if crush-related compartment syndrome develops. In addition to direct neural trauma, peripheral nerve damage is more likely to occur in the presence of rhabdomyolysis, compartment syndrome, and crush syndrome.[8,10] Compartment syndrome is a condition in which the increase in hydrostatic pressure in the osseous compartment induces muscle and nervous tissue hypoperfusion, leading to cellular anoxia, ischemia, and cell death.[11] Muscle infarction and nerve injury ensue at high compartment pressures. Compartment syndrome may result in rhabdomyolysis, and conversely, rhabdomyolysis may result in compartment syndrome.[12] Fasciotomy is a radical compartment syndrome treatment. It restores circulation with decompression, potentially inhibiting kidney failure and irreversible neurological injury by minimizing the risk of necrosis developing in the muscle. Routinely performed fasciotomies in the early period can reduce the muscle mass subject to necrosis, the severity of renal failure, the risks of peripheral neuropathy and ischaemic contracture, but they potentially increase the risk of infection.[13]

The literature review revealed that peripheral nerve injury is common in patients with rhabdomyolysis and crush syndrome.[8,10,14,15] Consequently, numerous interrelated issues that contribute to, trigger, and exacerbate each other will lead to multifactorial nerve injuries, making it challenging to identify the primary cause. Our results similarly revealed that peripheral nerve damage was significantly more common in patients with crush syndrome.

Peripheral nerve injuries have a considerable impact on long-term quality of life despite not being a life-threatening complication of earthquake-related traumas. Most cases assessed in the current study were associated with compartment and crush syndromes. However, this outcome is most likely due to more severe cases being referred to our hospital. The fact that the patients had multiple traumas, needed intensive care, and were coping with more critical health issues might have delayed their ENMG analyses. However, serial ENMG analyses within the first month may be helpful for patients requiring very early and early treatment. These are patients with clean incisions and neurotmesis. Although it is more beneficial to wait for other cases with neuropraxia and axonotmesis, serial ENMG examinations are crucial because rehabilitation is needed in this period, and close monitoring is required to see whether recovery, degeneration or regeneration develops. It is essential to immediately mobilize all available resources in post-disaster periods and focus on measures to prevent disability and increase the likelihood of survival.

Peripheral nerve injuries in earthquake victims are mostly seen in the distal nerves of the lower extremities and are often accompanied by crush syndrome. They require special attention as they may cause serious disability.

Ethical Considerations: This study was approved by the Ethics Committee of Ankara City Hospital (04.26.2023/E1-23-3481).

Conflict of Interest: The authors declare no conflict of interest.

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