



Research Article

Phenotypes and Electrophysiological Features of Hereditary Neuropathy with Liability to Pressure Palsies (HNPP) According to Physical Activities: A Single Military Hospital Experience

Eun Jin Kim*

Abstract

Background: HNPP typically presents as focal nerve palsy with complete or near-complete recovery, often involving upper extremity weakness due to brachial plexus involvement during military service. This study aimed to analyze the clinical features and neurophysiological examination results of HNPP occurring in the Korean military to determine whether the clinical presentations and electrodiagnostic patterns differ according to the trigger activity and to obtain useful information for the early diagnosis of HNPP.

Methods: Sixty-four patients with HNPP were included over an 11-year period, and medical records and electrophysiological tests were analyzed.

Results: Thirty-five patients presented with isolated compressive neuropathy and 18 patients presented with brachial plexopathy. Analysis of phenotypes according to triggering activity revealed that 13 of 16 patients with symptom onset after military training showed a type of isolated compressive neuropathy and that 11 of 20 patients with symptom onset after push-ups or after backpack carriages showed a type of brachial plexopathy. Six patients experienced symptoms following sleep, while four reported symptoms after trauma. Atypical features were observed in 11 cases (17.2%), including 3 lower lumbosacral radiculopathy-like presentation. Abnormal findings were observed in 91.2% of distal latency of the peroneal motor nerve.

Conclusions: The phenotype varied according to the physical activity that triggered HNPP onset in soldiers. These results are expected to help in the early diagnosis of HNPP in soldiers or those who develop it after physical activity.

Keywords: Neurogenetics; Neuropathy; Peripheral Neuropathology; Genetic disorder; Elbow; Knee; Shoulder

Introduction

Hereditary neuropathy with liability to pressure palsies (HNPP) is an autosomal dominant genetic disorder (PMP 22 gene) characterized by recurrent acute painless palsy, with the wrist, elbow, knee, and shoulder being the most susceptible areas [1-3]. Study on soldiers with HNPP has shown that the most common symptom is proximal upper extremity weakness due to brachial plexus injury, and brachial plexus involvement is relatively more common among soldiers compared to the reported 10%–30% occurrence in the general HNPP population [4,5]. In particular, soldiers have been reported to develop brachial plexus injuries after training and subsequently be diagnosed

Affiliation:

Department of Physical Medicine and Rehabilitation, The Armed Forces Capital Hospital, Seongnam-si, Gyeonggi-do, South Korea

*Corresponding Author:

Eun Jin Kim, Department of Physical Medicine and Rehabilitation, The Armed Forces Capital Hospital, Seongnam-si, Gyeonggi-do, South Korea.

Citation: Eun Jin Kim. Phenotypes and Electrophysiological Features of Hereditary Neuropathy with Liability to Pressure Palsies (HNPP) According to Physical Activities: A Single Military Hospital Experience. *Journal of Orthopedics and Sports Medicine*. 7 (2025): 103-109.

Received: February 18, 2025

Accepted: February 24, 2025

Published: March 14, 2025

with HNPP [6]. Previous studies reported that push-ups were the most common physical activity associated with brachial plexus injuries in military patients with HNPP [7,8].

Interest in HNPP occurring in soldiers was focused on injury to the brachial plexus, but clinically, it shows various clinical features depending on the physical activity that causes symptoms. Studying the clinical features and electrophysiologic findings of HNPP is crucial for early diagnosis. Since HNPP presentation varies with the affected nerve, it's essential to examine phenotypes and electrodiagnostic patterns based on triggering activities. However, there is a lack of studies on the clinical presentation of HNPP in military personnel and the physical activities that trigger it.

This study aimed to analyze the phenotype of HNPP in soldiers according to the triggering activity and analyze the characteristics of the electrophysiological test to obtain information that will help diagnose HNPP early.

Methods

Study subjects

This retrospective study included patients diagnosed with HNPP via electrophysiologic and genetic testing at the Armed Forces Capital Hospital in South Korea from 2012 to 2022. The diagnosis criteria for HNPP were limited to patients who met clinical and electrodiagnostic criteria [9] and had abnormalities confirmed by genetic testing.

Data collection

The clinical characteristics, neurophysiological test, and genetic test results of the subjects were collected by searching medical records for 11 years.

Statistical analysis

The SPSS (version 11.0) statistical program was used for statistics, and the Student's T-test was applied for statistical comparison of the left and right nerve conduction study results.

Ethical consideration

The institutional review board of the Armed Forces Capital Hospital approved the study (AFCH 2023-05-001).

Results

Sixty-four patients were enrolled after checking their medical records, and the average age of the patients was 22.2 (18–44) years, with one of them being female. The ranks of the patients were 24 private second class soldiers, 19 private first class soldiers, 11 corporals, 5 sergeants, 4 noncommissioned officers (1 sergeant major, 2 master sergeants, and 1 sergeant first class), and 1 warrant officer.

The average height of patients was 174.7 (164–186)

cm, the average weight was 70.0 (52–100) kg, and the average body mass index was 28.5 (15.4–55.6) kg/m². The departments that the patients first visited included neurology (22 patients), orthopedic surgery (18 patients), rehabilitation medicine (15 patients), and neurosurgery (9 patients).

The timing of symptom onset in the patients was an average of 5 months prior to the initial consultation day, and five patients had symptoms prior to enlistment. There were 20 patients whose symptoms began within 1 month of enlistment, 17 whose symptoms began within 6 months of enlistment, and 6 whose symptoms began within 1 year of enlistment. Five patients had symptoms that started 1 year after enlistment, and 10 patients could not remember when the symptom started.

Fifty-four patients had symptoms in the upper extremity, 21 on the right, 23 on the left, and 9 bilaterally. The lower extremities were involved in nine patients, with five patients experiencing symptoms on the right side, two on the left side, and two bilaterally. Two patients had all four limbs involved. The most common initial symptom reported by patients was muscle weakness (38), followed by paresthesia (17), tingling (14), pain (10), and claw hand (2).

Twelve patients had a family history of acute nerve palsy. Twenty-nine patients had a history of nerve palsy, of whom two underwent surgical treatment for nerve compression (median nerve decompression and/or ulnar nerve decompression).

The patients were followed up for an average of 8.8 months, and 48 patients were discharged with mental and physical disabilities because they were unable to continue their military service due to physical problems. Table 1 summarizes the basic information and clinical presentation of the patients.

Triggering activities and phenotypes

The most common triggering activity for symptom onset was military training, including physical activities such as fitness training and basic military training such as shooting and obstacle course training, reported by 16 people. Thirteen patients reported symptom onset after push-ups, 7 after carrying backpacks, 6 upon waking up, 4 after trauma (shoulder dislocation, fracture, etc.), and 18 did not report any specific triggering activity.

The authors categorized patients into five phenotypes based on clinical presentation, referring to existing studies [2,3,10]: 1) isolated compressive neuropathy, 2) brachial plexopathy, 3) polyneuropathy, 4) radiculopathy, and 5) multiple mononeuropathy.

The most common phenotype was isolated compressive neuropathy in 35 patients, followed by brachial plexopathy in 18, polyneuropathy in 5, radiculopathy in 4, and multiple mononeuropathy in 1. Among 35 patients with isolated

Table 1: Demographic and clinical findings of 64 patients with HNPP.

Findings	
Male/Female	63 / 1
Average age (years old)	22.2 (18-44)
Rank	
Sergeant first class/Master sergeant/ Sergeant major/Warrant officer	1/2/1/1
Enlisted soldiers (PV1/PFC/Corporal/ Sergeant)	59 (24/19/11/5)
Physical condition	
Average height (cm)	174.7 (164-186)
Average weight (kg)	70.0 (52-100)
Average BMI (kg/m ²)	28.5 (15.4-55.6)
Department of initial consultation	
Neurology	22
Orthopedic Surgery	18
Rehabilitation Medicine	15
Neurosurgery	9
Average symptom duration (months)	5 (0-72)
Symptom onset before enlistment	5
Symptom onset within 1 month of enlistment	20
From 1 month to 6 months after enlistment	17
From 6 months to 1 year after enlistment	6
Others	15
Location (Side)	
Upper (Right/Left/Both)	53 (21/23/9)
Lower (Right/Left/Both)	9 (5/2/2)
Both Upper and Lower	2
Presenting manifestations	
Muscle weakness	38 (59.4%)
Sensory deficit	17 (26.6%)
Paresthesia	14 (21.9%)
Pain	10 (15.6%)
Claw hand	2 (3.1%)
Family History of nerve palsy	12 (18.8%)
Previous event of nerve compression	29 (45.3%)
History of surgery for nerve compression	2 (3.1%)
Discharge from military service due to physical disabilities	48 (75.0%)

compressive neuropathy, the ulnar nerve was most commonly involved in 14 patients, followed by the musculocutaneous nerve in 8, the radial nerve in 6, the peroneal nerve in 5, and the axillary and median nerves in 1 each.

The analysis of phenotypes according to the triggering activities showed the type of isolated compressive neuropathy in 13 out of 16 patients with symptom onset after military training (physical training and basic military training). The specific neural phenotypes of isolated compressive neuropathy included ulnar and musculocutaneous nerves in four patients each, radial and peroneal nerves in two patients each, and the axillary nerve in one patient. Of the remaining three, two had brachial plexus abnormalities and one had a type of radiculopathy.

In patients with symptom onset after push-ups or backpack carriages, the phenotype was most often brachial plexopathy (7 of 13 patients and 4 of 7 patients, respectively). Of the remaining six patients whose symptoms started after push-ups, two had a type of radial nerve, one had the musculocutaneous nerve, two had polyneuropathy, and one had radiculopathy. Of the remaining three patients whose symptoms started after backpack carriages, two had musculocutaneous nerve disorders and one had polyneuropathy.

In the six patients with symptom onset after sleep, radial nerve palsy and brachial plexus palsy were present in two patients each and musculocutaneous nerve palsy and peroneal nerve palsy were present in one patient each. In the 18 patients with symptom onset without any specific triggering activity, ulnar nerve involvement was the most common phenotype in 10 patients (Table 2).

Atypical clinical presentation

Three patients with a Guillain–Barre syndrome-like presentation were diagnosed with Guillain–Barre syndrome after presenting with chief complaints of decreased muscle strength or sensation in the upper extremities that occurred an average of 5.3 months before the hospital visit, with two of them treated with intravenous immunoglobulin (IVIG) without improvement.

Of the three patients who presented to the neurosurgery department with chief complaints of radiating pain to the lower extremities accompanied by lower back pain that occurred an average of 4.5 months before the hospital visit and showed a lumbosacral radiculopathy-like presentation, one patient had persistent pain after surgical treatment (partial hemilaminectomy and microdiscectomy) for herniated intervertebral disc.

Three patients who presented with tingling sensations in the upper extremities that began on average 28.7 months before the hospital visit were found to have chronic sensory neuropathy. Two patients presented with chronic ulnar

Table 2: Triggering activities and presenting phenotypes.

Triggering Activities N (%)		Military training	Push-ups	Backpack carriages	Sleep	Trauma	Unknown
Presenting Phenotype N (%)		16 (25.0)	13 (20.3)	7 (10.9)	6 (9.4)	4 (6.3)	18 (28.1)
Isolated compressive neuropathy	35 (54.7)	13	3	2	4	1	12
Ulnar nerve	14	4					10
Musculocutaneous nerve	8	4	1	2	1		
Radial nerve	6	2	2		2		
Peroneal nerve	5	2			1	1	1
Axillary nerve	1	1					
Median nerve	1						1
Brachial plexopathy	18 (28.1)	2	7	4	2	1	2
Polyneuropathy	5 (7.8)		2	1		1	1
Radiculopathy	4 (6.3)	1	1			1	1
Multiple mononeuropathy	1 (1.6)						1
Sensory polyneuropathy	1 (1.6)						1

neuropathy, characterized by persistent symptoms lasting for more than 12 months.

Electrodiagnostic study

After analyzing the nerve conduction studies of 64 patients diagnosed with HNPP, 47, 8, 7, and 2 were found to have sensorimotor polyneuropathy, sensorimotor polyneuropathy combined with brachial plexopathy, brachial plexopathy, and bilateral peroneal neuropathy, respectively.

When electromyography results were analyzed according to the triggering activities, 6/16, 5/13, and 2/5 patients had brachial plexus injuries or accompanying brachial plexus injuries after military training, push-ups, and backpack carriages, respectively. The electrodiagnostic patterns for each trigger are shown in Table 3.

We analyzed in detail the nerve conduction study results of 41 patients who underwent neurophysiological testing at our center. The tests were performed at a mean of 7.3 (1–44) months after symptom onset, and the results were compared with those of previous investigations of nerve conduction studies in patients with HNPP (Table 4) [2,3,10,11].

Delayed distal motor latency or no response of the peroneal nerve was observed in 91.2% of patients with HNPP, and delayed distal motor latency or no response of the median nerve was observed in 85.7%. In addition, a motor nerve conduction study revealed focal slowing across the elbow in the segment or no response of the ulnar nerve in 85.5% of patients.

There was no statistically significant difference between the left and right motor and sensory nerve conduction studies (Tables 5 and 6). Abnormal spontaneous activity was observed in 27 of the 36 patients who underwent needle electromyography.

Genetic study

As a results of test for the PMP22 gene, deletions were observed in 61 patients and mutations were observed in 3 patients. The mutations include a pathogenic variant (c.79-2A>C) in one patient at the exon 3 acceptor splice site, a splice site variant (c.179-1G>C) in one patient at the intron 3 site, and a splice site variant (c.78+1G>C) in one patient at the intron 2 site.

Table 3: Triggering activities and electrodiagnostic patterns.

Triggering Activities N (%)		Military training	Push-ups	Backpack carriages	Sleep	Trauma	Unknown
Electrodiagnostic Patterns N (%)		16 (25.0)	13 (20.3)	7 (10.9)	6 (9.4)	4 (6.3)	18 (28.1)
SMDPN [※]	47 (73.4)	9	8	5	4	3	18
SMDPN + Brachial plexopathy	8 (12.5)	4	3		1		
Brachial plexopathy	7 (10.9)	2	2	2	1		
Mononeuropathy	2 (3.1)	1				1	

※ SMDPN, Sensorimotor demyelinating polyneuropathy

Table 4: Results of the nerve conduction studies compared with previous studies.

Number of patients (Range of Age)		This study		Oliveira et al. [10]	Luigetti et al. [11]	Kumar et al. [13]	Mouton et al. [2]
		41 (19-44)		33 (6-77)	73 (17-84)	18 (3-73)	99 (14-84)
		Mean ± SD (Range)	Abnormal* (%)	Abnormal* (%)	Abnormal* (%)	Abnormal* (%)	Abnormal* (%)
Motor nerve							
Median	Distal motor latency (ms)	5.1 ± 1.4 (3.4 ~9.9)	85.7	80.7	90	81	89~100
	CMAP (mV)	9.9 ± 4.2 (3.0 ~22.9)	11.7	8.8			
	MCV (m/s)	50.7 ± 8.0 (37.1 ~67.5)	40	33.9	23	42	37-54
	Focal slowing (wrist)					81	
Ulnar	Distal motor latency (ms)	3.5 ± 0.8 (2.5 ~5.7)	37.3	57.6	74	59	55~94
	CMAP (mV)	9.6 ± 3.3 (0.7 ~20.5)	4	0			
	MCV (m/s)	51.5 ± 9.6 (30.6 ~80.8)	35.1	35.5	9	41	22-40
	Focal slowing (elbow)	37.0 ± 14.8 (17.8 ~90.0)	85.5	98.3	85	30	40-87
Tibial	Distal motor latency (ms)	4.8 ± 1.2 (3.5 ~7.5)	31.3	30.2			43-77
	CMAP (mV)	12.0 ± 5.0 (5.2 ~24.8)	9.4	3.8			
	MCV (m/s)	41.9 ± 8.0 (31.7~52.1)	25.8	35.8			
Peroneal	Distal motor latency (ms)	7.0 ± 2.0 (4.2 ~12.7)	91.2	57.6	59	58	70-91
	CMAP (mV)	4.0 ± 2.1 (0.3 ~8.5)	23.5	30.5			
	MCV (m/s)	39.4 ± 8.4 (28.6 ~48.6)	51.6	55.1	18	52	70-84
Sensory nerve							
Median	Distal latency (ms)	3.8 ± 1.1 (2.3 ~ 8.3)	52.6	82.8			
	SNAP (µV)	15.3 ± 10.1 (2.5 ~47.3)	61.8	56.9	36	67	52-75
Ulnar	Distal latency (ms)	3.6 ± 0.9 (1.9 ~6.6)	51.4	89.6			
	SNAP (µV)	14.1 ± 9.3 (2.0 ~46.6)	62.2	58.6		80	44-93
Sural	Distal latency (ms)	3.7 ± 0.7 (2.3 ~5.0)	61.3	61.7			
	SNAP (µV)	10.4 ± 7.4 (0.3 ~36.9)	22.6	21.7	77	76	45-90
Superficial peroneal	Distal latency (ms)	4.0 ± 1.1 (2.2 ~ 7.5)	66.7	43.6			
	SNAP (µV)	7.8 ± 3.1 (3.4 ~13.3)	40	33.3			

*Abnormal, including no response

Table 5: Results of the motor nerve conduction studies.

	Right			Left			p-value
	N	Mean±SD (Range)	Abnormal† (%)	N	Mean±SD (Range)	Abnormal† (%)	
Distal Motor Latency (ms)							
Median nerve	39	5.1 ± 1.4 (3.5 ~9.9)	84.6	38	5.2 ± 1.6 (3.4 ~9.7)	86.8	0.88
Ulnar nerve	38	3.4 ± 0.8 (2.5 ~5.7)	34.2	37	3.6 ± 0.8 (2.5 ~5.6)	40.5	0.497
Tibial nerve	21	4.7 ± 1.2 (3.5~7.5)	23.8	11	5.0± 1.3 (3.9 ~ 6.9)	45.5	0.459
Peroneal nerve	22	6.9 ± 2.2 (4.2~12.7)	86.4	12	7.1 ± 2.1 (5.1 ~ 10.4)	100	0.724
Compound Motor Action Potential (CMAP) (mV)							
Median nerve	39	10.0 ± 4.4 (3.2 ~22.9)	10.3	38	9.9 ± 4.1 (3.0 ~20.4)	13.2	0.892
Ulnar nerve	38	9.8 ± 3.6 (0.7 ~20.5)	2.6	37	9.5 ± 3.3 (1.2 ~ 17.7)	5.4	0.688
Tibial nerve	21	12.7 ± 5.5 (5.2 ~24.8)	9.5	11	10.8 ± 4.6 (5.6 ~19.0)	9.1	0.301
Peroneal nerve	22	4.1 ± 2.1 (0.3 ~8.5)	22.7	12	3.8 ± 2.2 (1.1 ~ 7.3)	25	0.65
Nerve Conduction Velocity (m/s)							
Median nerve	37	51.3 ± 9.9 (38.6 ~67.5)	40.5	38	50.2 ± 9.5 (37.1 ~63.2)	39.5	0.414
Ulnar nerve (wrist-BE)	37	51.9 ± 12.0 (31.3 ~80.8)	37.8	37	50.2 ± 10.1 (30.6 ~59.6)	32.4	0.367
Ulnar nerve (BE-AE)	31	39.9 ± 16.8 (19.8 ~90.9)	80.6	31	34.0 ± 13.4 (17.8 ~ 71.4)	90.3	0.107
Tibial nerve	20	42.2 ± 9.5 (35.9~52.1)	25	11	41.3 ± 12.0 (31.7 ~ 46.9)	27.3	0.575
Peroneal nerve (ankle-BFH)	20	39.7 ± 9.7 (28.6 ~48.6)	45	11	38.8 ± 11.8 (30.3 ~ 47.4)	63.6	0.65

† Abnormal, including no response

Table 6: Results of the sensory nerve conduction studies.

Nerve	Right			Left			p-value
	N (NR*)	Mean±SD (Range)	Abnormal† (%)	N (NR*)	Mean±SD (Range)	Abnormal† (%)	
Latency (ms)							
Median	38 (3)	3.8 ± 1.1 (2.3 ~8.3)	52.6	38 (1)	3.9± 1.1 (2.6 ~ 6.7)	52.6	0.702
Ulnar	36 (5)	3.5 ± 1.0 (1.9 ~5.5)	52.8	38 (2)	3.6 ± 0.8 (2.3 ~6.6)	50	0.635
Sural	20 (1)	3.5 ± 0.8 (2.3 ~4.9)	55	11	3.9 ± 0.8 (2.9 ~5.0)	81.8	0.106
Superficial peroneal	20 (6)	4.0 ± 1.2 (3.1 ~7.5)	60	10 (1)	4.1 ± 1.5 (2.2 ~ 5.7)	80	0.722
Sensory Nerve Action Potential (µV)							
Median	38 (3)	15.7 ± 10.2 (2.5 ~38.9)	60.5	38 (1)	14.9 ± 10.1 (3.2 ~47.3)	63.2	0.765
Ulnar	36 (5)	14.4 ± 8.8 (2.7 ~46.6)	55.6	38 (2)	13.8 ± 9.8 (2.0 ~44.0)	68.4	0.795
Sural	20 (1)	10.2 ± 6.2 (1.9 ~26.2)	20	11	10.7 ± 9.4 (0.3 ~36.9)	27.3	0.853
Superficial peroneal	20 (6)	8.0 ± 3.2 (4.0 ~13.3)	45	10 (1)	11.5 ± 3.4 (3.4 ~12.9)	30	0.74

*NR, No Response; † Abnormal, including no response

Discussion

HNPP often occurs during military service, and it is more prevalent in soldiers than in the general population due to its predominance in males and the high level of physical activity. In a Korean study, 31.3% of HNPP patients reported symptoms of onset during military service [9]. Similarly, Finnish military personnel experienced brachial plexus palsy at a rate of 53.7 per 100,000, with 7% diagnosed with HNPP [4]. This suggests a prevalence of at least 3.8 per 100,000 for brachial plexus-related HNPP in Finnish soldiers. Another Korean study found 78 HNPP cases in military personnel over 10 years, with a prevalence of 3.1 per 100,000 [12].

Although previous studies have focused on brachial plexus injuries for HNPP occurring in soldiers, 54.7% of patients were presenting with various forms of mononeuropathy and 28.1% presenting with brachial plexopathy in this study. Having a good understanding of the phenotype associated with the triggering activity of HNPP can aid clinicians in considering HNPP as a diagnosis in patients presenting with abnormal neurological symptoms based on their history. The most common triggering activity of mononeuropathy was military training. We found that if the onset occurred after a shoulder-intensive activity, such as push-ups and backpack carriages, the phenotype was often manifested in the form of brachial plexus injury.

In this study, most patients with HNPP had weakness due to a typical presentation of focal nerve palsy, but 17.2% of them exhibited an atypical clinical presentation [2,11]. Two of the three patients with a Guillain–Barre-like presentation, which is a relatively well-known atypical clinical presentation of HNPP, were misdiagnosed with Guillain–Barre syndrome and received immunotherapy, but the symptoms did not improve, thereby diagnosis of HNPP.

Although not reported in previous studies, we observed

a lumbosacral radiculopathy-like presentation as an atypical clinical presentation of HNPP. In patients who complained of lower back pain and radiating leg pain, herniations of lumbar discs were observed in radiology tests, and they were diagnosed with lumbosacral radiculopathy. One of the three patients underwent lumbar fusion, but the symptoms did not improve and the patient was diagnosed with HNPP on further thorough examination.

A better understanding of the atypical clinical presentation of HNPP will also help in early diagnosis, as atypical presentations of HNPP can lead to delayed diagnosis or misdiagnosis as other conditions, potentially resulting in unnecessary surgery.

Previous studies and studies involving military populations have reported the most abnormalities in motor nerve conduction studies of the median nerve in HNPP [1-3,10,11]. However, when we analyzed the nerve conduction studies of 41 patients with HNPP who underwent electrophysiologic studies, it was found that the peroneal motor nerve had the highest incidence of abnormalities including no response, with 91.2% of distal latency, in contrast to previous studies [3,10,11]. The reason for most patients complaining of upper extremity symptoms but exhibiting more abnormalities in the distal latency of the peroneal motor nerve can be attributed to the fact that soldiers spend significant time walking due to marching or training, which often stimulates the lower extremity nerves, and that soldiers wear combat boots that can compress the nerves in the lower leg.

In addition, axonal involvements were frequently observed, with abnormal spontaneous activity observed in 75% of patients who underwent needle electromyography. This may be because in HNPP patients with genetically vulnerable nerves, minor trauma during physical activities such as military training or drills can lead to rapid demyelination or focal axonal damage.

Based on the findings of this study, there are several considerations when clinically suspecting HNPP in soldiers and performing electrophysiologic study. First, even if the patient complains of symptoms in the upper extremities, the inclusion of motor nerve conduction studies of the peroneal nerve may help in the early diagnosis of HNPP. Second, it is important to consider that in physically active soldiers, HNPP is often accompanied by focal axonal damage.

The results of this study showed that special activities play significant a role in the manifestation of symptoms in soldiers with HNPP, and that different nerves are affected depending on the triggering activity, leading to a variety of phenotypes.

This study has limitations. First, its retrospective design and reliance on medical records may introduce bias and limit generalizability. Second, as the study focused on soldiers from one country, the findings may not apply to soldiers elsewhere with different equipment and training. Lastly, limiting the diagnostic criteria to genetically confirmed cases may result in different clinical and electrodiagnostic features compared to other studies.

Conflict of Interest

There are no conflicts of interest to declare.

Authorship Statement

The author solely conceived, designed, collected data, analyzed, and wrote the manuscript. The author is the guarantor of this work and takes full responsibility for the overall content and the integrity of the data presented.

Funding

This study was not funded by any external sources.

References

1. Dubourg O, Mouton P, Brice A, et al. Guidelines for diagnosis of hereditary neuropathy with liability to pressure palsies. *Neuromuscul Disord* 10 (2000): 206-208.
2. Mouton P, Tardieu S, Gouider R, et al. Spectrum of clinical and electrophysiologic features in HNPP patients with the 17p11.2 deletion. *Neurology* 52 (1999): 1440-1446
3. Kumar NJ, Cole J, Parry GJ. Variability of presentation in hereditary neuropathy with liability to pressure palsy results in underrecognition. *Ann N Y Acad Sci* 883 (1999): 344-350
4. Mäkelä JP, Ramstad R, Mattila V, et al. Brachial plexus lesions after backpack carriage in young adults. *Clin Orthop Relat Res* 452 (2006): 205-209
5. Pareyson D, Scaiola V, Taroni F, et al. Phenotypic heterogeneity in hereditary neuropathy with liability to pressure palsies associated with chromosome 17p11.2-12 deletion. *Neurology* 46 (1996): 1133-1137.
6. Horowitz SH, Spollen LE, Yu W. Hereditary neuropathy with liability to pressure palsy: fulminant development with axonal loss during military training. *J Neurol Neurosurg Psychiatry* 75 (2004): 1629-1631
7. Kim K-E. Characteristic features of hereditary neuropathy with liability to pressure palsy (HNPP) presenting with brachial plexopathy in soldiers. *J Neurol Sci* 346 (2014): 174-177
8. Kim K-Y, Yeom J. Proximal arm weakness is the most common presentation in young Korean soldiers diagnosed as having hereditary neuropathy with liability to pressure palsy (HNPP). *J R Army Med Corps* 162 (2016): 352-354.
9. Kim SM, Choi BO, Sunwoo IN, et al. Clinical, electrophysiological and pathological characteristics in hereditary neuropathy with liability to pressure palsies (HNPP) with chromosome 17p11.2-p12 deletion. *J Korean Neurol Assoc* 19 (2001): 251-259
10. Oliveira APM, Pereira RC, Onofre PT, et al. Clinical and neurophysiological features of the hereditary neuropathy with liability to pressure palsy due to the 17p11.2 deletion. *Arq Neuropsiquiatr* 74 (2016): 99-105.
11. Luigetti M, Del Grande A, Conte A, et al. Clinical, neurophysiological and pathological findings of HNPP patients with 17p12 deletion: a single-centre experience. *J Neurol Sci* 341 (2014): 46-50
12. Kim K-E, Kim EJ, Kim KD, et al. Prevalence of neuromuscular diseases in young South Korean males; a Korean military manpower administration and medical command data-based study. *J Clin Neurol* (2023).



This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC-BY\) license 4.0](https://creativecommons.org/licenses/by/4.0/)