



The effects of head posture on nerve conduction studies in patients with ankylosing spondylitis

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Abstract

Ankylosing spondylitis (AS) is a chronic inflammatory disease which effects cervical posture of patients. The aim of this study was to evaluate AS patients according to the degree of cervical disorder and was evaluate them electrophysiologically, functionality, and disease parameters. Our study comprised 64 AS patients and 30 healthy controls. The head posture of patients was evaluated by craniovertebral angle (CVA) measurement. Nerve conduction of bilateral median, radial, ulnar, and medial antebrachial cutaneous (MAC) nerves were studied in all patients. The most important nerve conduction differences in AS patients who have severe forward head posture (FHP) were decrease in sensory nerve action potential (SNAP) amplitude and compound muscle action potential amplitudes of median nerves, a decrease in the SNAP amplitude of ulnar nerves, a delay in the F response latency of ulnar nerves, and prolongation in the SNAP latency of the MAC nerve. The FHP disorder that develops in AS patients may have electro physiological effects, similar to those of thoracic outlet syndrome. In addition, the functional status of these patients is worsened as severity of FHP increases.

Keywords Electrophysiology · Ankylosing spondylitis · Neural conduction · Posture

Introduction

Ankylosing spondylitis (AS) is a chronic inflammatory disease of axial skeleton [1]. Sacroiliitis that develops due to inflammation and joint surface fusion, spinal stiffness, and rigidity constitute the major clinical manifestation of the disease [2]. In the early period, postural changes and kyphotic deformity may develop in AS patients, and these changes may become more evident over time [3]. Forward head posture (FHP) caused by spinal kyphosis in AS patients is one of the most important complications affecting daily life activities and quality of life [4, 5]. Examined biomechanically, in AS patients, FHP shifts the center of gravity of the body forward and downward in the sagittal plane [5].

In addition to many studies investigating the involvement of the central nervous system (multiple sclerosis, cauda equina syndrome, and focal epilepsy), there are few that evaluated peripheral nervous system involvement in patients

with AS [6–9]. In these patients, nerve congestion caused by arthritis or tenosynovitis, amyloidosis, autoimmunity, and side effects of drugs used have been implicated in peripheral nerve involvement [6, 7, 10]. In one of the studies in the area, somatosensory evoked potential (SEP) responses in peripheral nerves were evaluated in AS patients, and in a later study, peripheral neuropathies were investigated in the same patient group [11, 12].

To the best of our knowledge, no research has been conducted to evaluate the effect of head posture in AS patients on the peripheral nerves in the upper extremity. The aim of this controlled study was to evaluate the electrophysiological findings of the upper extremity peripheral nerves in patients with AS and investigate the relationships between head posture and electrophysiological data and disease-related parameters.

Patients and methods

We evaluated 82 AS patients aged 20–55 years that fulfilled the modified New York criteria for AS who underwent non-steroidal anti-inflammatory drug and sulfasalazine treatment [13]. The medical files were examined, and the patients with diabetes, hypothyroidism, hyperthyroidism, cancer, chronic

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alcohol use, chronic kidney disease, diagnosed cervical root compression, cervical surgery or trauma history, idiopathic scoliosis, history of torticollis, central nervous system disease, and history of drug use that may cause neuropathy and those that did not undergo electrophysiological studies for any reason were excluded from the study. In view of these criteria, the head posture of AS patients was evaluated by craniocervical angle (CVA) measurement. Cervical bidirectional graphy was used to confirm that there were no anatomic variations that could cause compression of the peripheral nerves. All patients included in the study were photographed, and the angle between the relative line horizontally drawn from the C7 cervical vertebra and the relative line passing through the tragus and C7 was measured. A CVA of 53.2–56.8 was considered normal, 46.9–49.1 indicated mild FHP, and 40.7–43.2 moderate–severe FHP [14]. The patients with AS were divided into three groups according to the severity of FHP. Eighteen patients who could not tolerate electrophysiological studies or who were found to have peripheral neuropathy or Martin Gruber anastomosis on electrophysiological examination or presented other exclusion criteria were excluded from the study. 64 patients with AS were included in analyses. The control group was originally planned to consist of 40 healthy subjects, but 10 patients were excluded from the study, because they did not agree to undergo electrophysiological examination or presented with one or more of the same exclusion criteria. As a result, 64 AS patients and 30 controls were evaluated (Fig. 1). The control group consisted of hospital employees and their relatives. The local ethics committee approval was obtained for the study and informed consent forms were signed by all patients at the beginning of the study.

For both groups, the demographic characteristics, neurological, and musculoskeletal system examinations findings recorded. The visual analog scale (VAS) was used to determine the presence and intensity of neck pain. The patients were asked to score their neck pain within the last week from 1 to 10 (0: no pain, 10: unbearable pain). The upper limb disability of the patients was evaluated using the Turkish version of the Disabilities of the Arm, Shoulder, and Hand (Quick-DASH) questionnaire, a self-reported assessment tool for the measurement of physical function and symptoms. The scores obtained from this questionnaire indicate the level of disability and its severity, ranging from 0 (no disability) to 100 (most severe disability) [15]. Furthermore, in the AS patient groups, the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), Bath Ankylosing Spondylitis Disease Metrology Index (BASMI) and Bath Ankylosing Spondylitis Disease Functional Index (BASFI) were calculated [16–18].

Electrophysiological assessments were undertaken using Nihon-Kohden MI (Tokyo, Japan) electroneuromyography equipment at room temperature (25 °C). Bilateral median,

ulnar, radial nerve motor and sensory nerve conduction and F responses, sensory responses and F responses of bilateral medial antebrachial cutaneous nerve (MAC) were studied in all groups in accordance with Oh's protocol as Ref. [19]. Orthodromic method was used for motor nerve conduction studies and antidromic method was used for sensory nerve conduction studies. All electrophysiological evaluations were performed by the same researcher, who was blinded to the group and clinical parameters of the patients.

Statistical analysis

Statistical analysis was performed using SPSS version 20. Descriptive statistics were expressed as mean \pm standard deviation for continuous variables and as numbers (*n*) for categorical variables. The normality of distribution was checked using the Shapiro–Wilk test. One-way ANOVA was used to compare three or more groups with normal distribution. The comparison of three or more groups without normal distribution was undertaken using the Kruskal–Wallis test. The group that caused a significant difference was determined by the Tukey HSD test and Tamhane test. Pearson's Chi-square test was employed to compare the qualitative data. Significance was assessed at $p < 0.01$ and $p < 0.05$.

Results

The mean age was calculated as 42.6 ± 8.4 years for the control group (Group 1), 43.3 ± 7.5 years for the AS group without FHP (Group 2), 42.9 ± 9.2 years for the AS group with mild FHP (Group 3), and 44.1 ± 6.4 years for the AS group with severe FHP (Group 4). There was no statistically significant difference between the groups in terms of demographic data like gender, body mass index, work related activities ($p > 0.05$) (Table 1). There was no significant difference in disease duration among AS groups ($p > 0.05$) (Table 1).

CVA was measured as 53.20 ± 1.08 , 53.45 ± 1.11 , 47.69 ± 1.79 , and 41.91 ± 1.47 in Groups 1–4, respectively. While there was no statistically significant difference in CVA values between Groups 1 and 2, these two groups significantly differed from the remaining groups ($p < 0.001$).

The VAS scores that provided an assessment of neck pain were significantly higher in Groups 3 and 4 compared to Groups 1 and 2 ($p = 0.035$).

The Quick-DASH scores did not statistically significantly differ between Groups 1 and 2, but a statistically significant difference was observed between these two groups and the two FHP groups ($p < 0.001$). The BASDAI, BASMI and BASFI values differed between each pair of the AS groups (Groups 2–4). This difference was statistically significant for the comparison between Groups 2 and 4, and Groups 3 and 4

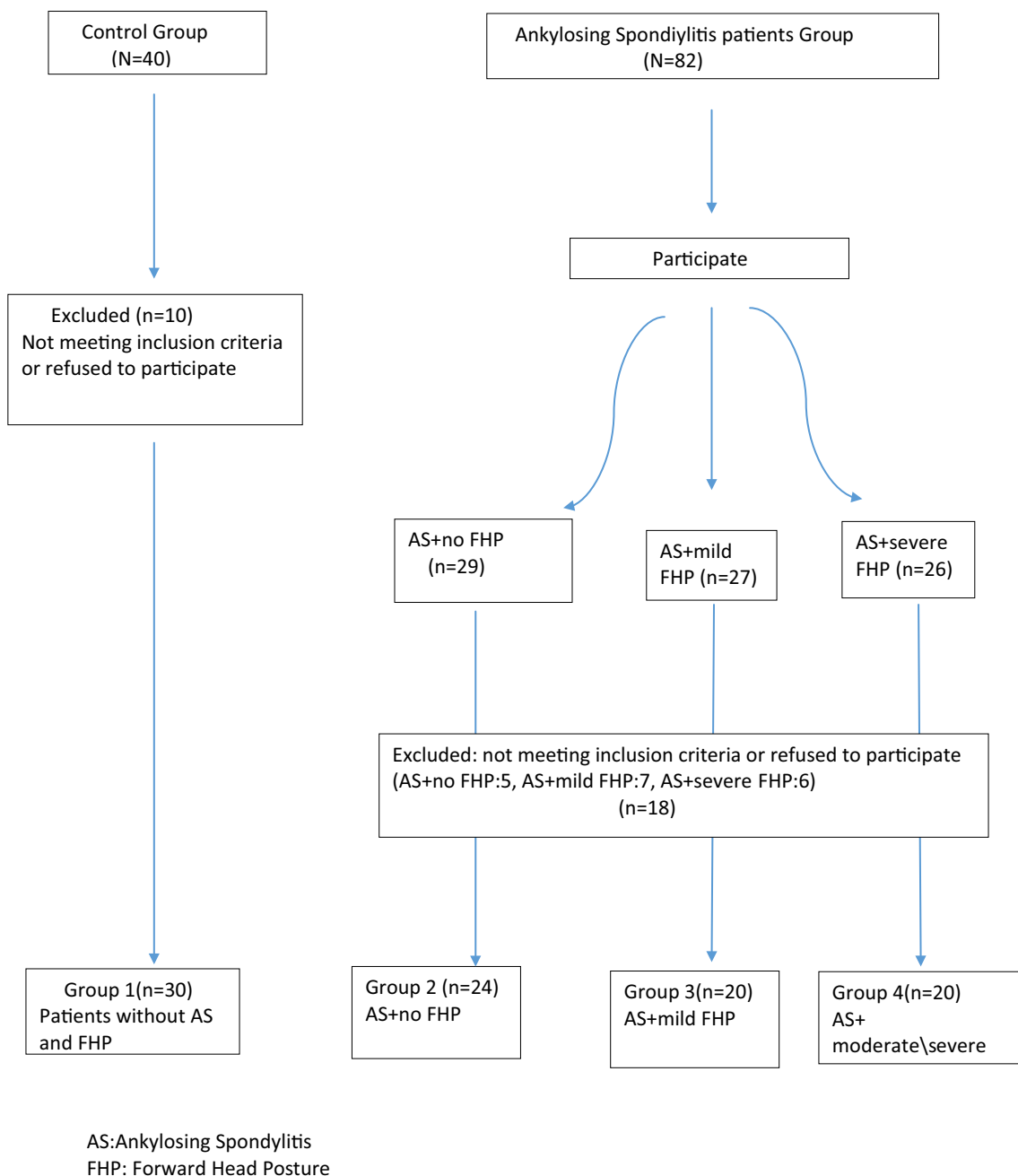


Fig. 1 Flow diagram

($p < 0.001$, respectively); however, no significant difference was found between Groups 2 and 3 ($p > 0.05$) (Table 1).

In electrophysiological studies, a total of 60 extremities belonging to 30 individuals in the control group and 128 extremities of 64 AS patients were evaluated for a bilateral assessment.

The evaluation of sensory conduction of the median nerve showed that the SNAP (sensory nerve action potential) amplitude (wrist–second finger segment) was lower in Group 3 than Group 1 ($p < 0.001$) and also lower in group 4

than Groups 1, 2, and 3 (respectively, $p < 0.001$, $p = 0.033$, $p = 0.039$). Median nerve sensory conduction velocity in Group 4 was lower than all groups (respectively, $p < 0.001$, $p < 0.001$, $p = 0.02$).

The evaluation of motor conduction of median nerve showed that distal latency of compound muscle action potential (CMAP) was longer in Group 4 than in Groups 1, 2 and 3 (respectively, $p < 0.001$, $p < 0.001$, $p = 0.09$). The amplitude of median nerve CMAP decreased in Group 4 compared to Groups 1, 2, and 3. (respectively, $p < 0.001$,

Table 1 The demographical and clinical characteristics of the patients

	Control group (Group 1) <i>n</i> = 30	AS patients without FHP (Group 2) <i>n</i> = 24	AS patients with mild FHP (Group 3) <i>n</i> = 20	AS patients with moderate\severe FHP (Group 4) <i>n</i> = 20	<i>p</i>
Age (years)	42.6 ± 8.4	43.3 ± 7.5	42.9 ± 9.2	44.1 ± 6.4	> 0.05
Gender male/female <i>n</i> (%)	20 (66.6)\10 (33.4)	15 (62.5)\9 (37.5)	13 (65)\7 (35)	15 (75)\5 (25)	> 0.05
Body Mass index (kg/m ²)	29.3 ± 4.1	28.6 ± 3.7	30.2 ± 5.1	27.4 ± 6.1	> 0.05
Work -related activities					
Mild work <i>n</i> (%)	3 (10)	2 (8.33)	2 (10)	4 (20)	
Moderate work <i>n</i> (%)	20 (66.6)	16 (66.6)	14 (70)	13 (65)	
Heavy work <i>n</i> (%)	7 (23.4)	6 (25.1)	4 (20)	3 (15)	
Disease Duration (month), mean (min–max)	–	47.7 (3–220)	56.5 (5–220)	60.5 (3–186)	> 0.05
Cervicogenic complaint <i>n</i> (%)	–	–	9 (45)	13 (65)	0.039 ^f
Cervicogenic headache <i>n</i> (%)	–	–	7 (35)	9 (45)	> 0.05
Visual analogue scale (mm) ± SD	2.6 ± 21.65	31.45 ± 21.67	33.12 ± 31.12	43.82 ± 27.12	0.035 ^{cef}
Craniovertebral angle ± SD	53.20 ± 1.08	53.45 ± 1.11	47.69 ± 1.79	41.91 ± 1.47	< 0.001 ^{bcd^{ef}}
Quick-DASH ± SD (min– max)	6.43 ± 8.76	5.91 ± 7.48	23.07 ± 17.71	31.19 ± 16.11	< 0.001 ^{bcd^{ef}}
BASDAI ± SD (min– max)	–	1.39 ± 0.91 (0–2.60)	2.39 ± 1.92 (0–6.4)	4.1 ± 1.61 (1.6–7)	< 0.001 ^{ef}
BASMI ± SD (min–max)	–	4.75 ± 0.62 (5–7)	6.61 ± 1.09 (5–9)	8.2 ± 1.5 (5–11)	< 0.001 ^{ef}
BASFI ± SD (min–max)	–	1.1 ± 0.54 (0.4–2.2)	1.9 ± 1.61 (0.4–6.1)	3.36 ± 1.47 (1.1–6.3)	< 0.001 ^{ef}

^aGroup 1–Group 2, ^bGroup 1–Group 3, ^cGroup 1–Group 4, ^dGroup 2–Group 3, ^eGroup 2–Group 4, ^fGroup 3–Group 4

$p = 0.03$, $p = 0.01$). The F response latency of median nerve was prolonged in Group 4 than Groups 1, 2, and 3 (respectively, $p < 0.001$, $p < 0.001$, $p = 0.032$) and also prolonged in Group 3 than Group 1 ($p = 0.037$).

The evaluation of sensory conduction of ulnar nerve (wrist–fifth finger segment) showed that the amplitude of SNAP was lower in Group 3 than Group 1 ($p < 0.001$) and also Group 4 than Groups 1, 2, and 3 (for all, $p < 0.001$). The F response latency of ulnar nerve was prolonged in Group 3 compared to Group 1 ($p = 0.10$) and also Group 4's F response latency was prolonged compared to Groups 1, 2, and 3 (for all, $p < 0.001$).

The evaluation of sensory and motor nerve conduction study of radial nerve revealed no statistically significant difference between groups.

The evaluation of the MAC nerve conduction showed that amplitude of SNAP was lower in Group 4 than Groups 1 and 3 (respectively, $p < 0.001$, $p = 0.018$), and also was lower in Group 3 than Group 1 ($p = 0.016$).

All results of electrophysiological evaluations of the AS groups and the control group presents in Table 2.

Discussion

In this study, we evaluated whether FHP, which is a common postural disorder in AS patients, electrophysiologically affects the upper extremity peripheral nerves, as well as its effects on the functionality and daily life activities of these patients. In light of the data obtained from the study, we conclude that FHP is an important factor affecting the functions of upper extremity nerves and as the severity of FHP increases, its effect on the functionality of AS patients is greater. To the best of our knowledge, this was the first study to evaluate the upper extremity nerve conduction in AS patients according to the degree of cervical postural disorder and evaluate them electrophysiologically.

We found no significant difference between the AS patients without FHP and the control group in terms of any of the parameters included in peripheral nerve conduction studies. This suggests that having AS alone does not create an additional risk of damage to the peripheral nerves of the upper extremities unless it is accompanied by an associated postural disorder. Similarly, there was no difference in the

Table 2 Electrophysiological evaluation of the groups

	Control group (Group 1) (n=60)	AS patients without FHP (Group 2) (n=48)	AS patients with mild FHP (Group 3) (n=40)	AS patients with mod- erate\severe FHP (Group 4) (n=40)	p	Normal limits*
Median nerve (SCS)						
Digit 2-wrist						
SNAPamp (μV)	25.63 ± 3.83	26.2 ± 9.99	22.35 ± 3.62	19.82 ± 4.23	< 0.001 < 0.001 ^{bc} 0.033 ^c 0.039 ^f	10.0
Vsens (m\sn)	52.76 ± 3.23	53.41 ± 2.16	51.56 ± 4.78	47.24 ± 5.06	< 0.001 ^{ce} 0.02 ^f	49.4
Median nerve (MCS)						
DL (ms)	3.38 ± 0.38	3.32 ± 0.42	3.48 ± 0.30	3.83 ± 0.58	0.01 ^{ce} 0.09 ^f	3.8
CMAPamp (μV)	18.33 ± 3.88	18.85 ± 4.81	17.32 ± 2.19	11.33 ± 4.25	< 0.001 ^c 0.03 ^e 0.01 ^f	4.3
Vmotor (m\sn)	56.80 ± 4.29	55.68 ± 2.65	55.33 ± 3.02	53.28 ± 5.02	< 0.001 ^c 0.01 ^e 0.09 ^f	49.7
F _{min} median nerve	25.2 ± 25.42	25.10 ± 1.77	25.86 ± 2.24	25.60 ± 2.70	> 0.05	32
Carpal tunnel syndrome (%)	5 (8.3)	7 (14.5)	9 (22.5)	13 (32.5)	< 0.001 ^{ce} 0.037 ^b 0.032 ^f	
Ulnar nerve (SCS)						
Digit 5-Wrist						
SNAPamp (μV)	28.32 ± 3.60	25.88 ± 4.54	23.14 ± 4.02	19.10 ± 4.39	< 0.001 ^{bcef}	7.0
Wrist–below elbow						
Vsens (m\sn)	51.82 ± 3.16	49.48 ± 3.76	51.03 ± 3.68	52.14 ± 4.84	> 0.05	47.3
Ulnar nerve (MCS)						
DL (ms)	2.52 ± 0.33	2.65 ± 0.51	2.59 ± 0.45	2.70 ± 0.27	> 0.05	3.3
CMAPamp (μV)	14.92 ± 2.84	13.72 ± 2.54	15.28 ± 2.11	14.71 ± 3.15	> 0.05	7.0
Wrist–below elbow						
Vmotor (m\sn)	58.60 ± 5.34	56.79 ± 5.70	54.41 ± 2.48	51.38 ± 4.50	< 0.001 ^{bce} 0.02 ^f	49.9
Below elbow–above elbow						
Vmotor (m\sn)	60.86 ± 6.31	60.84 ± 7.81	55.45 ± 3.40	54.23 ± 3.64	< 0.001 ^{bc} 0.01 ^d	
F _{min} Ulnar nerve	24.33 ± 1.80	25.06 ± 2.27	25.74 ± 1.64	27.12 ± 2.81	0.10 ^b < 0.001 ^{cef}	
Radial nerve (SCS)						
First web-wrist						
SNAPamp (μV)	19.95 ± 3.45	18.25 ± 2.86	18.48 ± 3.91	19.19 ± 5.16	> 0.05	
Vsens (m\sn)	54.08 ± 3.16	53.41 ± 4.23	52.16 ± 4.48	53.13 ± 4.56	> 0.05	
Wrist–elbow						
Vsens (m\sn)	57.34 ± 4.51	56.78 ± 3.65	58.12 ± 4.67	57.12 ± 3.40	> 0.05	44.31
Radial nerve (MCS)						
DL (ms)	2.47 ± 0.30	2.27 ± 0.29	2.82 ± 0.66	2.69 ± 0.54	> 0.05	
CMAPamp (μV)	10.79 ± 2.79	9.47 ± 2.66	10.12 ± 3.23	10.97 ± 3.76	> 0.05	
Wrist–elbow						
Vmotor (m\sn)	54.47 ± 4.41	55.92 ± 7.93	55.92 ± 7.93	55.87 ± 3.38	> 0.05	
F _{min} radial nerve	24.76 ± 1.96	24.34 ± 2.25	24.11 ± 1.51	24.77 ± 1.80	> 0.05	

Table 2 (continued)

	Control group (Group 1) (n=60)	AS patients without FHP (Group 2) (n=48)	AS patients with mild FHP (Group 3) (n=40)	AS patients with mod- erate/severe FHP (Group 4) (n=40)	<i>p</i>	Normal limits*
Medial antebrachial cutaneous nerve (SCS)						
Latency (ms)	2.04±0.20	2.01±0.19	2.19±0.53	2.12±0.50	> 0.05	
NAPamp (μV)	19.64±3.70	18.54±4.10	17.86±1.94	16.62±1.50	0.016 ^b < 0.001 ^c 0.018 ^f	10
Vsens (m/s)	53.93±4.83	54.83±3.85	54.66±4.53	54.90±5.21	> 0.05	41.7

Vsens sensory nerve conduction velocity, *Vmotor* Motor nerve conduction velocity, *SNAPamp* Sensory nerve action potential amplitude, *CMA-Pamp* Compound muscle action potential amplitude, *DL* Distal latency, *MCS* Motor nerve conduction study, *SCS* Sensory nerve conduction study

*The normal lower and upper limits for our electroneuromyography laboratory. The lower limits for *Vsens*, *Vmotor*, *SNAPamp*, *CMA-Pamp* and the upper limits for *DL*

^aGroup 1–^aGroup 2, ^bGroup 1–^aGroup 3, ^cGroup 1–^aGroup 4, ^dGroup 2–^aGroup 3, ^eGroup 2–^aGroup 4, ^fGroup 3–^aGroup 4

presence of cervicogenic headache between the two groups, but a significant difference was observed in the VAS scores. These results indicate that in patients with AS, cervical compartment-specific neck pain may be due to the presence of FHP. This was an expected result considering that AS patients have a rheumatic disease of an inflammatory character, and even if they did not develop FHP, their VAS scores were higher than the healthy controls. Previous studies similarly reported higher pain scores for the AS patients compared to the control groups, which was attributed to the fact that AS was an inflammatory disease. It was also noted that central sensitization mechanisms were more common in AS patients than in controls, and this is known to increase pain perception and alter the processing of pain [20].

There are not sufficient publications on the effect of postural changes related to the cervical spine on upper extremity functions of patients and indices used in clinical evaluation in AS patients. However, in this group of patients, the effect of postural disorder on general balance has been addressed [21, 22]. In the current study, we did not evaluate the effect of posture on balance but observed that functionality was affected by cervical posture. Although there were no significant differences between the AS patient groups in terms of demographic data, we found an increase in all three BASDAI, BASMI and BASFI scores of the patients in parallel to the increase in CVA. This is a predictable outcome for BASMI, which is a metrological index, considering that one of the metrological data scored in BASMI is the tragus wall distance that is naturally greater in AS cases that develop FHP [18]. We think that the increase in BASDAI and BASFI scores can be attributed to the negative effects of cervical posture disorder and upper extremity peripheral nerve involvement in patients with AS. In a study comparing AS

cases with kyphoscoliosis and other postural disorders, the BASFI scores were found to be higher in the presence of kyphoscoliosis, whereas HAQ-DI, another index for quality of life, was lower [23]. Another study evaluating 1538 patients with AS reported a correlation between BASFI and reduced cervical rotation, but the authors did not group patients in terms of CVA or severity of cervical posture disorder [24].

According to the results of our study, concerning the Quick-DASH scores evaluating upper extremity functionality, the upper limb disability was higher in AS patients with moderate to severe FHP, whereas they were lower and similar in the control group and AS patients without FHP. In the light of these results, we consider that postural disorder in the cervical region may have a negative effect on the arm and hand disability by affecting the peripheral nerves of the upper extremity. Similarly, in the literature, a negative correlation between CVA and cervical disability was previously noted [25]. In another study, pain and disability were investigated and found correlated in young women with FHP and no additional disease [26]. Furthermore, in a study that evaluated the relationship of FHP presence and severity with disability, the Quick-DASH scores were determined to be higher in the group with FHP [27].

In rheumatologic patients, nerve compression, amyloidosis, autoimmunity, and side effects of drugs are often implicated in the etiology of peripheral neuropathy [7, 10]. The use of tumor necrosis factor (TNF)-α antagonists, a drug group frequently used in the treatment of AS patients, have also been considered to cause the development of peripheral neuropathy. Therefore, when forming our study groups, we selected patients that did not use TNF-α antagonist to eliminate the possibility of drug-associated nerve involvement [28].

Electrophysiological studies are the most sensitive and specific method for evaluating peripheral nerve pathologies. Sensory and motor nerve conduction studies can reveal both not only symptomatic but also asymptomatic nerve pathologies [29]. In our study, the nerve conduction values of all nerves examined were within normal limits, but in AS patients with moderate to severe FHP, reduced SNAP and CMAP amplitudes of the median nerve and SNAP amplitude of the ulnar nerve, as well as delayed F responses of the ulnar nerve were observed, and the SNAP amplitude of the MAC nerve was significantly lower compared to the remaining groups. These electrophysiological findings are similar to those detected in thoracic outlet syndrome. Similar electrophysiological results were reported in a study conducted with individuals followed up for FHP with no additional rheumatologic disease [27]. In patients with FHP, postural disorder, abduction in the scapula, weakness in the middle and lower trapezoidal and serratus anterior muscles lead to the development of secondary scapular rotation and hypertrophy in the upper rhomboid, sternocleidomastoid and scalar muscles. This imbalance in the muscles and the postural disorder in the cervical spine may cause increased compression on the brachial plexus nerves [29, 30]. We believe that in AS patients, a clinical picture that resembles TOS emerges due to the increased pressure on the brachial nerves caused by FHP that develops due to the cervical postural disorder.

In patients with TOS, nerve structures belonging to the brachial plexus may be compressed alone or together with the arterial structures in their proximity. Pain, paresthesia and weakness in muscles are frequent clinical findings accompanying TOS. In these patients, electrophysiological studies may reveal a decrease in the CMAP amplitude of the MAC nerve, delayed latency, and reduced velocity, as well as reduced SNAP amplitude of the ulnar nerve, reduced CMAP amplitude of the median nerve, and longer F response latency of the ulnar nerve [31].

In a study that performed biomechanical analysis of cervical posture disorder in patients with AS, the authors suggested that this postural condition caused the center of gravity to shift forward, which was compensated by the body through the development of retraction of the shoulders, extension of the hips, and plantar flexion of the ankles [32]. Shoulder retraction, one of the postural compensation mechanisms in AS patients, can strain the proximal of the peripheral nerves in the upper extremity and lead to electrophysiological changes in the peripheral nerves through chronic straining of the muscles and compression caused by the trigger points on the affected muscles.

In 2010, Gündüz et al. reported that the rate of peripheral nerve involvement in AS patients was 40.6% [11]. However, the authors did not divide the patients according to cervical involvement or the degree of FHP. They

evaluated the median, ulnar, common peroneal, tibial and sural nerves, but excluded the assessment of radial and MAC nerve responses. Based on their results, they attributed the effect on peripheral nerves in patients with AS to the general systemic problems of the nervous system, such as nerve compression, drug toxicity, vasculitis, amyloidosis, and autoimmunity. In line with the electrophysiological data in our study, in addition to these factors, we consider that the FHP disorder that develops in AS patients may be a further risk factor for peripheral nerve involvement. Therefore, we believe that preventing or delaying postural disorders in AS patients can also reduce the incidence of associated neuropathies.

This was the first study to evaluate the electrophysiological effects of FHP disorder on the peripheral nerves of the upper extremity in AS patients. However, there are some limitations to our study. For example, the number of patients included in the study was relatively low. In addition, the SEP responses of the patients were not evaluated for technical reasons. SEP abnormalities are commonly seen in neurogenic TOS and provide sensitive results in differential diagnosis [33].

In conclusion, the FHP disorder that develops in AS patients may cause electrophysiological effects, similar to those of TOS, on the peripheral nerves of the upper extremity. In addition, the functional status of these patients is worsened as the severity of FHP increases. However, there is a need for further detailed studies in this area involving a greater number of cases.

Compliance with ethical standards

Conflict of interest All the authors declare that they have no conflict of interest.

Ethical approval All procedures performed in this study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments.

Informed consent Informed consent was obtained from all individual participants included in the study.

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