Shear wave elastography of median nerve at carpal tunnel in upper extremity spasticity

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Purpose

Spasticity is a velocity-dependent increase in the muscle tone as a part of the upper motor neuron (UMN) syndrome. Hypertonia and immobilization in spasticity can cause joint contractures. Joint contractures and limb deformities are chief complications of long-term spasticity, causing significant pain and functional impairment. Depending on the location of the spasticity, these contractures and deformities can impact mobility and activities of daily living such as toileting, dressing, and transferring[1]. Spasticity can cause a flexed wrist posture and, therefore, symptoms such as painful passive stretching of wrist flexors may occur. This flexed wrist posture may compress the median nerve at the carpal tunnel. Median nerve compression in these patients can aggravate the neurologic symptoms such as numbness, paresthesia, and motor impairment.

Phalen’s maneuver is a diagnostic test for carpal tunnel syndrome (CTS)[2], which requires the patients to hold their wrist in forced flexion by pushing the dorsal surfaces of both hands together for 30-60 s. This maneuver can increase the pressure at the carpal tunnel via mass effect.

Characteristic symptoms such as burning, tingling, or numb sensation over the thumb, index, middle, and ring fingers suggests CTS due to the compression of the median nerve. On the basis of this data, the prolonged wrist flexion posture in the hemiparetic patients with wrist spasticity can impact their median nerves at the carpal tunnel. Previously reported studies showed that elastography could be a useful diagnostic method for both detecting CTS and evaluating its severity[3-5]. Thus, this study aimed to compare the median nerve elasticity of the paretic side with that of the nonparetic side of patients with wrist spasticity through SWE at the carpal tunnel and to assess the correlations between shear wave velocities (Vs) and clinical outcomes.
Methods and materials

This study was approved by the Baskent University Institutional Review Board and Ethics Committee and supported by the Baskent University Research Fund. Written informed consent was obtained from all patients.

The inclusion criteria were as follows:

• Wrist spasticity, at least Modified Ashworth Scale (MAS) 1 on the hemiplegic side
• Age above 18 years
• Sufficient cognitive and communication ability to be able to give written informed consent

The exclusion criteria were as follows:

• Established contracture or other neurological impairments without stroke
• Received botulinum toxin or treatments involving neurolytic agent (such as phenol or alcohol) injections within the past 6 months
• Previously treated with oral antispastic treatment medications, intrathecal baclofen, or surgery for spasticity
• Recurrent stroke affecting extremities on both sides, which were healed with sequelae
• Prior surgery or major trauma to the upper extremity and any other muscular disorders that may have affected the wrist
• Prior brachial plexus lesions or median nerve neuropathies or their surgeries
• Suspicious physical examination findings for median neuropathy of the unaffected side

Power analysis during the biostatistical preliminary assessment indicated a study population of 24 patients with 95% confidence level and 80% power.

A total of 30 patients were enrolled in the study. Of these, two patients were excluded for having bilateral wrist spasticity and three for having taken botulinum toxin injection 3 months ago. Thus, 25 patients were included in the study.

Clinical Evaluation

A neurologic examination was performed as follows. First, the subjects were briefly interviewed concerning their present illness and previous history. The presence of
subjective symptoms in the nonparetic upper extremity, such as muscle weakness and numbness, was also questioned.

Second, patients underwent manual muscle tests of the abductor pollicis brevis and the flexor carpi radialis muscles and sensory tests of light touch and a pinprick of the arm, hand, and fingers on the nonparetic side. Third, Phalen's maneuver and Tinel's signs on the median nerve at the nonparetic wrist were performed for the clinical exclusion of CTS.

Paralysis of the spastic extremity was graded according to the Brunnstrom's motor staging (BMS). BMS is a six-stage evaluation tool for motor recovery in stroke patients. It has three sections that measure the upper extremity, lower extremity, and hand (6). It comprises six stages: stage I, flaccidity in muscles, in which the patient is incapable of voluntary movement on the most affected side; stage II, spasticity appears, in which the patient is capable of involuntary movement only in synergy patterns; stage III, spasticity increases, in which the patient gains voluntary control of movement in synergy patterns; stage IV, spasticity decreases, in which the patient is capable of voluntary movement without synergy patterns; stage V, spasticity continues to decline, in which the patient is capable of more complex natural movements; and stage VI, spasticity disappears, except for when fatigued, in which the patient is capable of moving individual joints, which is almost normal. A higher stage of BMS represents better recovery (7). This study evaluated the hand section of BMS.

Then the degree of spasticity of the wrist was measured by MAS. MAS is the most commonly accepted method to assess spasticity in individuals with UMN syndrome (8). MAS was described as the degree of resistance to quick passive movement and was rated as follows:

0 No increased resistance
1 (MAS 1) = Minimal resistance at the end of the range of motion (ROM)
2 (MAS 1+) = Minimal resistance throughout less than half of the ROM
3 (MAS 2) = Clear resistance throughout most of the ROM
4 (MAS 3) = Strong resistance, passive movement is difficult
5 (MAS 4) = Rigid wrist flexion

MAS and BMS were assessed by a single physiatrist with the patients sitting on an examination chair comfortably with their arms resting at their sides.

**US and SWE Examinations**
The US and SWE examinations were performed with a US system (Acuson S 2000; Siemens, Erlangen, Germany). After the clinical evaluation, patients underwent SWE of the median nerves on both paretic and nonparetic sides.

SWE was performed by using a probe with an L9-4 linear array. The US and SWE examinations were performed by a radiologist with 2 years of experience in the musculoskeletal imaging. This radiologist was blinded to physician's assessment and patients' clinical history. Moreover, the patients were requested not to tell anything about the physician's assessment to the radiologist.

A standard US examination was initially performed to view the median nerve in the axial plane. Images were obtained at the proximal carpal row level while the transducer was perpendicular to the long axis of the nerve (Fig. 1). If any movement occurred during SWE, the scanning was repeated.

The quality of the images was assessed by color-coded quality maps provided by the US system in which the green areas were considered reliable.

A rectangular electronic box-shaped region of interest (ROI) was used for $V_s$ measurements that were automatically provided by the system software. Care was taken to place the ROIs at the median nerve. Three separate SWE measurements were performed in each wrist. Both maximum and mean $V_s$ were recorded.

**Statistical analysis**

Statistical analysis was performed using the statistical package SPSS software (version 17.0, SPSS Inc., IL, USA). If continuous variables were normal, they were described as mean ± standard deviation ($P > 0.05$ in Kolmogorov-Smirnov test or Shapira-Wilk [n < 30]), and if they were not normal, they were described as the median. Comparisons between groups were applied using the Student $t$ test for normally distributed data and the Mann-Whitney $U$ test for data that were not normally distributed. Correlations between the clinical outcomes and $V_s$ were tested using the Pearson's correlation test. The correlation coefficients were interpreted as either excellent $r \geq 0.91$; good $0.90 \leq r \leq 0.71$; fair $0.70 \leq r \leq 0.51$; weak $0.50 \leq r \leq 0.31$; or little or none $r \leq 0.3$. Values of $P < 0.05$ were considered statistically significant.
Fig. 1: (A) B-mode ultrasound image showing the median nerve (white arrow) in the axial plane. (B) SWE of the median nerve.

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Results

Patient demographic and clinical characteristics are summarized in Figure 2.

The stroke types of the study population were as follows: ischemic stroke \((n = 21)\), hemorrhagic stroke \((n = 2)\) and hemorrhagic transformation following ischemic stroke \((n = 2)\).

BMS and MAS scores of the present study population are categorized in Figure 3.

Comparisons of SWE measurements of the median nerve between the paretic and nonparetic sides are summarized in Figure 4.

\[ V_{\text{max}} \] was 3.11 ± 0.54 and 3.16 ± 0.78 m/s on the paretic and nonparetic sides, respectively \((P = 0.797)\). \[ V_{\text{mean}} \] was 2.94 ± 0.51 and 3.01± 0.71 m/s on the paretic and nonparetic sides, respectively \((P = 0.637)\).

Also, no significant correlation existed between the \( V_s \) of the median nerve at the wrist with MAS, BMS scores, and time since stroke \( (r < 0.3, \ P > 0.05) \).
Table: Clinical characteristics of the study population

<table>
<thead>
<tr>
<th>Characteristics</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) *</td>
<td>56.4±13.6</td>
</tr>
<tr>
<td>Gender (Females/ Males),(n)</td>
<td>9/16</td>
</tr>
<tr>
<td>Duration of stroke, months*</td>
<td>20.6±22.5</td>
</tr>
<tr>
<td>Paretic side, Right/left,( n)</td>
<td>9/16</td>
</tr>
<tr>
<td>Stroke etiology, (n)</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>13</td>
</tr>
<tr>
<td>Multifactorial</td>
<td>5</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Mitral valve surgery</td>
<td>1</td>
</tr>
<tr>
<td>Trauma</td>
<td>1</td>
</tr>
<tr>
<td>Unknown etiology</td>
<td>4</td>
</tr>
</tbody>
</table>

*mean ±standard deviation

Fig. 2: Clinical characteristics of the study population were shown.
Table: Clinical outcomes of study population, (n=25)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Brunnstrom's Motor Staging of upper extremity</strong></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>15</td>
</tr>
<tr>
<td>Stage 3</td>
<td>3</td>
</tr>
<tr>
<td>Stage 4</td>
<td>4</td>
</tr>
<tr>
<td>Stage 5</td>
<td>3</td>
</tr>
<tr>
<td><strong>Spasticity degree measured by Modified Ashworth Scale</strong></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>7</td>
</tr>
<tr>
<td>Stage 2</td>
<td>11</td>
</tr>
<tr>
<td>Stage 3</td>
<td>6</td>
</tr>
<tr>
<td>Stage 4</td>
<td>1</td>
</tr>
</tbody>
</table>

**Fig. 3:** Clinical outcomes of study population were shown.

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Table 3: Comparisons of SWE measurements of median nerve (m/s), (mean ± standard deviation)

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Value</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>V_s maximum</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paretic side</td>
<td>3.11±0.54</td>
<td>0.797</td>
</tr>
<tr>
<td>Non-paretic side</td>
<td>3.16±0.78</td>
<td></td>
</tr>
<tr>
<td>V_s mean</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paretic side</td>
<td>2.94±0.51</td>
<td>0.637</td>
</tr>
<tr>
<td>Non-paretic side</td>
<td>3.01±0.74</td>
<td></td>
</tr>
</tbody>
</table>

V_s: Shear Wave Velocity

Fig. 4: SWE measurements of the median nerve (m/s) were summarized.

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Conclusion

In this prospective study, the elasticity of the median nerve on the paretic and nonparetic sides was compared at the carpal tunnel. Only those patients with unilateral spasticity were included. The $V_s$ of the median nerve did not show any significant difference between the normal and unaffected sides.

Previously, a few studies assessing elastography of the median nerve at the carpal tunnel was reported in the literature \(^{(4,5,10,11)}\). A recently published study showed the posture-induced changes detected by SWE at the median nerve \(^{12}\). Posture-induced changes are acute changes; however, the postural changes in spasticity are a long-standing period after the acute phase. The median nerve might have shown increased stiffness due to compression at the carpal tunnel. Moreover, the elasticity of the median nerve did not correlate with time since stroke in this study. The duration of time since stroke showed a wide variety in the present study population, but most of the patients were in the chronic phase. The mean duration of time since stroke was 20 months in the present study population.

Additionally, peripheral nerves were shown to have a slide and stretch mechanism to accommodate changes in the nerve bed length during joint movements \(^{13}\). Likewise, the median nerve might have adapted the changes at the nerve bed length by this slide and stretch mechanism in the study population.

In healthy individuals, motor cortical activity, descending via the corticospinal tract is the predominant pathway of voluntary behaviors. These voluntary behaviors are provided by co- or reciprocal activation of muscles during movement. These activation patterns of muscles are defined as muscle synergies. After stroke, the damage to the motor pathways may cause other descending pathways to be upregulated to compensate. The contribution of these pathways may emerge as new synergy patterns after stroke. Flexion synergy is a common pattern of synergy following stroke with arm impairment. Flexion synergy is defined as the obligatory coupling between shoulder abduction and elbow flexion \(^{(14, 15)}\). Previous studies have shown the median nerve to be easily unloaded when the shoulder is adducted or elbow is flexed \(^{(13)}\). Additionally, the median nerve had increased stress in the following positions: depression of the shoulder girdle, shoulder abduction associated with external rotation, and both elbow and wrist extension \(^{(16)}\). The extension position of the wrist caused increased tension on the median nerve when compared with the flexion position. In a cadaver study, the median nerve showed increased tension mainly during shoulder abduction combined with the extension of the elbow and wrist \(^{(17)}\). Thus, the flexion posture of the wrist may not affect the median nerve in the chronic phase.
The first limitation of this study was the limited sample size. The second limitation was that the absence of peripheral neuropathy was not confirmed by performing electrophysiological studies on the unaffected side (18-20). Although the nonparetic extremities in patients with stroke are generally considered to be normal, ambulatory assistive devices, peripheral nerve conduction abnormalities, or overuse of the nonparetic muscles may cause peripheral neuropathies of the median nerve on the unaffected side. Another limitation of this study was including patients with low levels of spasticity.
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References


