Palmaris Brevis Spasm Syndrome: Localizing Features

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Summary

Introduction: Palmaris brevis spasm syndrome (PBSS) is spontaneous, involuntary contractions of the palmaris brevis (PB) muscle. Here, we aim to examine origin of the contractions in four patients diagnosed as PBSS.

Methods: Clinical, neurological and electrophysiological findings, information regarding demographics, and treatment details of PBSS patients were assessed. Routine electrophysiological and polymyographic investigations, cervical magnetic resonance imaging, and roentgenograms of the wrist and elbow were studied. To clarify the origin of the involuntary activity, (1) we compressed ulnar nerve with ice bags over the wrist and over the hypothenar region for 10 minutes, (2) we compressed ulnar nerve for 10 minutes by bare hands at the level of wrist, (3) we induced 10-minutes ischemia of the affected arm produced by suprasystolic cuff compression at the upper arm, (4) we injected 10 ml lidocain in the midpoint of the line between medial epicondyle of humerus and olecranon process of ulna, and (5) we injected botulinum toxin type A into the PB muscle.

Results: Polymyographic electromyography revealed bursts of frequently recurring motor unit potentials over PB muscle. None of the applications except botulinum toxin changed the contractions. The only way to suppress the contractions was botulinum toxin injection.

Discussion: PBSS likely originates from the most distal part of the nerve and botulinum toxin is the choice of treatment.

Key words: Palmaris brevis muscle, palmaris brevis spasm syndrome, electromyography, superficial branch of the ulnar nerve, botulinum toxin

Palmaris Brevis Spazm Sendromu: Lokalizan Özellikler

Özet

Giriş: Palmaris brevis spazm sendromu (PBSS), palmaris brevis (PB) kasının istemsiz, spontan kasılmalarıdır. Burada, PBSS tanısı konan dört hastada kasılmaların kökeninin araştırılması amaçlanmıştır.

Yöntem: PBSS hastalarının klinik ve nörolojik bulguları, demografik verileri ve tedavi detayları değerlendirildi. Rutin elektrofizyolojik ve polimiyografik incelemeler, servikal manyetik rezonans görüntüleme ve el bileği ve dirsek röntgenleri yapıldı. İstemsiz hareketlerin orijini açıkça kavuşturmak için (1) el bileği ve hipotenar bölgeye buz torbaları ile 10 dakika soğuk uyguladık, (2)10 dakika boyunca el bileğine bası uyguladık, (3) kol üst kısmına tansiyon aleti manşonuya suprasistolik bası uygulayarak iskemi oluşturduk, (4) humerus medial epikondili ile ulna’nın olekranon çıkıntısı arasındaki hattın orta noktasına 10 ml lidokain enjekte ettik ve (5) PB kasına botulinum toksin enjekte ettik.

Tartışma: PBSS, olasılıkla sinirin en distal kısımdan kaynaklanmaktadır ve botulinum toksini tedavi seçeneğidir.

Anahtar Kelimeler: Palmaris brevis kası, palmaris brevis spazm sendromu, elektromiyografi, ulnar sinir yüzeyel dalı, botulinum toksin

INTRODUCTION
Palmaris brevis spasm syndrome (PBSS) is a spontaneous, irregular, dimpling contraction of the palmaris brevis (PB) muscle, of which the underlying mechanism is not clear⁵. Spasms are generally painless and do not result in disability⁶.

The aim of this report was to clarify the nosology, electrophysiology, and treatment of PBSS. In particular, we aimed to identify the i) clinical features, ii) required etiologic work-up, iii) pattern and origin of electromyographic (EMG) activity, and iv) treatment effectiveness.

MATERIAL AND METHODS
We assessed patients diagnosed with PBSS between 2008 and 2010 and collected clinical neurological and electrophysiological findings, information regarding demographics, and treatment details.

Electrophysiological investigations comprised routine sensory and motor conduction studies, late responses of the median and ulnar nerves, needle electromyography of muscles innervated by C5-T1 segments, and polymyographic needle EMG recordings of the PB and abductor digiti minimi (ADM) muscles on the symptomatic side and when needed, on the asymptomatic side. All patients underwent radiologic investigations, including cranial and cervical magnetic resonance imaging, and roentgenograms of the wrist and elbow. To clarify the origin of the involuntary activity, (1) we compressed ulnar nerve with ice bags over the wrist and over the hypothenar region for 10 minutes, (2) we compressed ulnar nerve for 10 minutes by bare hands at the level of wrist, (3) we induced 10-minutes ischemia of the affected arm produced by suprasystolic cuff compression at the upper arm, (4) we injected 10 ml lidocain in the midpoint of the line between medial epicondyle of humerus and olecranon process of ulna using peripheral stimulation needle and blockage was approved by the loss of pinprick sensation over little finger, and (5) we injected botulinum toxin type A into the PB (Botox®).

RESULTS
We identified four cases with PBSS. Age at examination was between 23 to 48 years, and all patients were women. Disease duration was between 2 months and 5 years. Clinically, the involuntary contractions were at the base of the fifth finger or in the hypothenar region, causing dimpling. Involuntary contractions occurred approximately 50 to 100 times a day in 3 patients and 1 (case 3) had fewer contractions (approximately 10 - 15 times a day). Two patients reported arm pain. In one patient, physiotherapy for tendinitis of the elbow preceded the onset of the symptoms. Involuntary movements were exacerbated in 1 patient by sewing and activities performed by hand and in another patient by carrying heavy bags. None of the patients expressed the use of a sensory trick. Neurological examination was normal other than the spontaneous, irregular dimpling contractions in the hypothenar region (Figure 1A). None of the patients had parkinsonism, psychiatric

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problems, family history of any movement disorder, or used neuroleptic drugs. All findings are summarized in Table 1.

Radiologic and routine electrophysiological investigations were normal. Polymyographic analysis revealed involuntary tonic bursts that were spontaneous, irregular, frequently recurring motor unit potentials (MUPs) with slow recruitment lasting 2 to 10 seconds. MUPs obtained between the episodes of involuntary activity were normal (Figure 1B).

Although presence of spasms confined to PB suggested PBSS, an ice bag test was performed to exclude myotonic or myokimic discharges which generally increase by cold application. Ice bag test did not precipitate clinical or electrophysiological discharges. Lidocaine blockage did not prevent spasms. Ischemic forearm test which may provoke tetanic spasms demonstrated no change. None of the other maneuvers except botulinum toxin changed the contractions. Diagnosis were compatible with PBSS both clinically and electrophysiologically.

Botulinum toxin type A injections into the PB terminated the involuntary activity within 1 week in treated patients. Case 1 was treated with 15 units of botulinum toxin. Her symptoms were relieved within 1 week and did not reappear at the 1st and 3rd month follow-up visits. Case 2 was administered 25 units of botulinum toxin. Her symptoms were also relieved within 1 week and did not reappear at the 1st and 4th month follow-up visits. Although she developed claw hand in the first month after the treatment, she improved within 1 month. At the last follow-up visit, the patient reported subjective sensation of mild contractions, but electrophysiological examination revealed no evidence of spasms. Case 3 declared that involuntary contractions were not disturbing, and she was therefore not treated. Case 4 improved within 1 week of receiving 25 units of botulinum toxin injection and she had no further complaints at the 1st and 3rd month follow-up visits.

**Figure 1 A:** Involuntary contractions at the base of the right fifth finger spreading to the hypothenar eminence and consequent relaxation phase (case 2). **B.** Needle EMG recordings using two channel needle electrodes over the palmaris brevis (PB) and abductor digiti minimi (ADM) shows spontaneous motor unit potential (MUP) bursts of PB (channel 1) and no contractions over ADM (channel 2), followed by MUPs of voluntary activity in two muscles (after the arrow) (case 1).
Table 1. Demographic, clinical, electrophysiological and radiological features.

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
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<th>Case 4</th>
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<td>39</td>
<td>48</td>
<td>37</td>
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<tr>
<td>Age at onset (year)</td>
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<td>38</td>
<td>5</td>
<td>37</td>
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<td>5 years</td>
<td>2 months</td>
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<tr>
<td>Gender</td>
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<tr>
<td>Medical history</td>
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<td>Migraine</td>
<td>DM for 15 years, HT, hypothyroidism, Raynaud p.</td>
<td>Nothing</td>
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<table>
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<tr>
<th>Involuntary contractions</th>
<th>Type</th>
<th>Number (/day)</th>
<th>Duration (sec)</th>
<th>Side</th>
<th>Tenderness</th>
<th>Pain</th>
<th>Aggravating factors</th>
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<tbody>
<tr>
<td></td>
<td>Irregular dimpling</td>
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<td>1-10</td>
<td>Right</td>
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<td>No</td>
<td>None</td>
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<tr>
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<td>Irregular dimpling</td>
<td>50-100</td>
<td>Momentary</td>
<td>Left</td>
<td>No</td>
<td>Yes, radiating to neck</td>
<td>Carrying heavy bags</td>
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<td>15-20</td>
<td>Momentary</td>
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<td>No</td>
<td>No</td>
<td>Sewing and activities performed by hand</td>
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<td>100</td>
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<td>Yes, left arm</td>
<td>None</td>
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<tr>
<td></td>
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<td>50-100</td>
<td>Momentary</td>
<td>Left</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Irregular dimpling</td>
<td>100</td>
<td>Momentary</td>
<td>Left</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
<tr>
<td></td>
<td>Irregular dimpling</td>
<td>50-100</td>
<td>Momentary</td>
<td>Left</td>
<td>No</td>
<td>No</td>
<td>None</td>
</tr>
</tbody>
</table>

| Preceding events | No | No | None | Tendinitis of left elbow |
| NE             | Normal | Normal | Normal | Normal |
| Routine CV    | Normal | Normal | Normal | Normal |
| Routine EMG   | Normal | Normal | Normal | Normal |
| EMG of PB     | Spontaneous, irregular, MUPs frequently recurring with slow recruitment | Spontaneous, irregular, MUPs frequently recurring with slow recruitment | Spontaneous, irregular, MUPs frequently recurring with slow recruitment | Spontaneous, irregular, MUPs frequently recurring with slow recruitment |
| EMG of ADM    | Normal | Normal | Normal | Normal |
| Radiological exam | Normal | Normal | Normal | Normal |
| Treatment     | 15 units | 25 units | None | 25 units |
| Botulinum toxin type A | Cure | Cure | None | Cure |
| Duration (months) | 3      | 4     | 3     | 3      |

*(DM, diabetes mellitus; HT, hypertension; MUPs, motor unit potentials; NE, neurological examination; CV, conduction values; EMG, electromyography; PB, palmaris brevis; ADM, abductor digiti minimi.)*

DISCUSSION

PBSS is easily recognized as irregular, high frequency motor unit discharges over the PB by surface or needle EMG. The mechanism is unclear and pathogenesis is not known. The PB is a thin, quadrangle-shaped muscle that localizes subcutaneously in the hypothenar region^{2,4}. It originates at the flexor retinaculum and the ulnar side of the palmar aponeurosis, extends beneath the ADM and flexor digitorum minimi muscles, and inserts into the skin of ulnar side of the palm. It is innervated by the superficial branch of the ulnar nerve. The PB helps to increase palmar dimpling and when it contracts, dimpling develops over the hypothenar eminence (dimple)^{4}.

Localized demyelination and consequent ephaptic transmission are suggested to be responsible for PBSS^{1}. In addition to possible focal damage, this syndrome is related to entrapment neuropathies of the ulnar nerve at different levels and C8 radiculopathies^{3,4}. As observed in our patients, PBSS is not a form of dystonia because typical features, such as use of a sensory trick, co-contraction, and overflow, were not present. Theoretically, in entrapment neuropathies of the ulnar nerve or C8 radiculopathies, other sensory and motor symptoms should also be present. Our patients had none of those symptoms. Furthermore, examinations of the ulnar nerve or cervical radices were normal. Therefore, in our patients none of
the aforementioned etiological factors were demonstrated. Moreover, spasms continued to occur even after chemical blockade of the ulnar nerve at the elbow level. Continuation of spasms after blockade of the peripheral nerve generally suggests that the spasm activity originates from an area distal to the blockade. Cold compression may induce myotonic or myokimic discharges which are included in differential diagnosis. Ice bag test did not precipitate clinical or electrophysiological discharges. Cold compression or sustained compression may also block spasms similar to effect of chemical blockade, however, they might be inadequate because they can only be administered within the patients' limits. It is impossible, however, for such an isolated spasm to originate between the wrist and elbow because this segment innervates various muscles other than the PB and provides sensory supply to the ulnar side of palm. Therefore, the origin of the spasms might be the most distal part of the ulnar nerve (superficial sensory branch), neuromuscular junction, or muscle. The presence of MUPs all over the muscle decreases the likelihood that the PBSS originates from the muscle or neuromuscular junction. Thus, we think that the spasm originates at the most distal part of the nerve.

PBSS might be related to the use of a computer or mouse\(^3\). Two of our patients stated that their spasms increased with activities performed by hand, specifically continuous sewing and carrying heavy bags, or that the spasms were preceded by physiotherapy for tendinitis, which may result in a trauma similar to that caused by the use of a mouse. All of our patients were women. Although this may be a coincidence and this disorder might not be specifically related to gender, the women in our country use their hands for various household tasks, which is likely partially responsible for the higher prevalence of carpal tunnel syndrome among women and might also predispose them to PBSS, which is considered as an occupational syndrome\(^3\). The nondominant hand tended to be involved more often, which is an interesting finding that contrasts with general findings of carpal tunnel syndrome. This might be secondary to leaning on the nondominant hand and compression of the superficial ulnar branch while using the dominant hand.

PBSS is painless and does not result in functional problems, therefore treatment is not mandatory. Case 3 was not treated, because her complaints were intermittent and PBSS did not impair her daily living activities. Drugs such as carbamazepine, phenytoin, and baclofen are ineffective\(^4\). Botulinum toxin, however, is an effective choice of treatment\(^5\). In our three patients who received treatment, botulinum toxin provided clinical improvement within the first week of administration and the improvement continued for at least 4 months.

In conclusion, the generator of PBSS seems to be the most distal part of the superficial branch of the ulnar nerve. Routine electrophysiologic investigations, however, are required to exclude underlying or accompanying neuropathy/radiculopathy. If PBSS is disturbing for patients, botulinum toxin is the treatment of choice.

**Abbreviations**

PBSS, Palmaris brevis spasm syndrome
PB, Palmaris brevis
EMG, electromyography
ADM, abductor digiti minimi
MUPS, motor unit potentials

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