İnme Sonrası Omuz Subluksasyonu ve Omuz El Sendromu; Beyin Lezyon Tarihi ve Lokalizasyonunun Etkisi

Levent Ediz1, Ozcan Hız1, Mehmet Fethi Ceylan1, Murat Toprak1

1Department of Physical Medicine and Rehabilitation, 2Department of Orthopaedics, Yuzuncu Yil University, Medical Faculty, Van, Turkey.

Amaç
Hemiplejik hastalarda, beyin lezyon lokalizasyonunun ve tutulan beyin tarafının omuz subluksasyonu (OS) ve omuz el sendromu (OES) gelişimindeki etkisi halen açık değildir. Bu çalışmamızın amacı enfarktulu hastalarda OS ve/veya OES’nin gelişiminde beyin lezyon lokalizasyonunun ve tutulan beyin tarafının ilişkisini değerlendirmektir. Gereç ve Yöntemler
Universiteler hastanesinde rehabilitasyon için yatırılan hemiplejik hastaların dosyaları retrospektif olarak tarandı. Beyin lezyon lokalizasyonu ve tutulan hemisfer tarafı inme anında beyin BT ile tespit edildi. Beyin lezyon lokalizasyonunu 35 hastaya 4 gruba ayrıldı. Grup I: Küçük yüzeyel enfarkt. Bu tip enfarktlar bir serebral lobda çap 20 mm den büyük olmayan küçük bölgelik enfarktları içermektedir. Grup II: Küçük yüzeyel enfarkt, bir serebral lobda 20 mm çapta büyük veya bir serebral hemisferin 2 veya daha fazla lobunda olan enfarktlar. Grup III: Büyük yüzeyel ve/veya derin enfarkt. Grup IV: Derin ve büyük yüzeyel enfarkt. Internal kapsül, basal ganglia veya thalamusun enfarktları. Grup IV: Combination of deep and large superficial infarcts (combined group II+III). Internal capsule or basal ganglia or thalamic infarct combined with fronto-temporal or fronto-parietal or temporo-parieto-occipital infarct. Toplam 140 hemiplejik hasta daha sonra OS ve/veya OES gelişimine göre 2 gruba ayrıldı. Bulgular
Beyin lezyon lokalizasyonu ile OS ve/veya OES gelişimi arasında anlamlı bir korelasyon bulundu. Kombine derin ve büyük yüzeyel enfarktlar, ve büyük yüzeyel ve derin enfarktlar gruplar daha siyah OS ve/veya OES gelişimi gösterdiler. OS ve/veya OES gelişimi ortalamada daha düşük Brunstroma ve Ashworth evreleri ile daha düşük fonksiyonel bağımsızlık düzeyi (FBI) skorlarına sahiptiler. Tutulan hemisfer tarafı açısından, OS ve/veya OES gelişimini açıdan anlamlı bir fark bulunmadı (p>0.05).

Sonuç
Bu çalışmamızda beyin lezyon lokalizasyonu, özellikle kombinert derin ve yüzeyel büyük enfarktlar, OS ve/veya OES gelişimi açısından bağımsız belirleyici bir faktör olarak gözlemlenmektedir. Ancak tutulan hemisfer tarafı belirleyici bir faktör değildir. Prospektif çalışmamızda, lezyon ve hareket etme sorununun etkisi daha iyice değerlendirilecektir.

Anadatia Kilimeler
Omuz El Sendromu, Omuz Subluksasyonu, Beyin Lezyon Lokalizasyonu, Beyin BT.

Özet
Amaç
Hemiplejik hastalarda, beyin lezyon lokalizasyonunun ve tutulan beyin tarafının omuz subluksasyonu (OS) ve omuz el sendromu (OES) gelişimindeki etkisi halen açık değildir. Bu çalışmamızın amacı enfarktulu hastalarda OS ve/veya OES’nin gelişiminde beyin lezyon lokalizasyonunun ve tutulan beyin tarafının ilişkisini değerlendirmektir.

Gereç ve Yöntemler
Universiteler hastanesinde rehabilitasyon için yatırılan hemiplejik hastaların dosyaları retrospektif olarak tarandı. Beyin lezyon lokalizasyonu ve tutulan hemisfer tarafı inme anında beyin BT ile tespit edildi. Beyin lezyon lokalizasyonunu 35 hastaya 4 gruba ayrıldı. Grup I: Küçük yüzeyel enfarkt. Bu tip enfarktlar bir serebral lobda çap 20 mm den büyük olmayan küçük bölgelik enfarktları içermektedir. Grup II: Küçük yüzeyel enfarkt, bir serebral lobda 20 mm çapta büyük veya bir serebral hemisferin 2 veya daha fazla lobunda olan enfarktlar. Grup III: Büyük yüzeyel ve/veya derin enfarkt. Grup IV: Derin ve büyük yüzeyel enfarkt. Internal kapsül, basal ganglia veya thalamusun enfarktları. Grup IV: Combination of deep and large superficial infarcts (combined group II+III). Internal capsule or basal ganglia or thalamic infarct combined with fronto-temporal or fronto-parietal or temporo-parieto-occipital infarct on one cerebral hemisphere. A total of 140 hemiplejik patients then divided into 2 groups according to the development of SS and/or SHS.

Bulgular
Beyin lezyon lokalizasyonu ile OS ve/veya OES gelişimi arasında anlamlı bir korelasyon bulundu. Kombine derin ve büyük yüzeyel enfarktlar, ve büyük yüzeyel ve derin enfarktlar gruplar daha siyah OS ve/veya OES gelişimi gösterdiler. OS ve/veya OES gelişimi ortalamada daha düşük Brunstroma ve Ashworth evreleri ile daha düşük fonksiyonel bağımsızlık düzeyi (FBI) skorlarına sahiptiler. Tutulan hemisfer tarafı açısından, OS ve/veya OES gelişimini açıdan anlamlı bir fark bulunmadı (p>0.05).

Sonuç
Bu çalışmamızda beyin lezyon lokalizasyonu, özellikle kombinert derin ve yüzeyel büyük enfarktlar, OS ve/veya OES gelişimi açısından bağımsız belirleyici bir faktör olarak gözlemlenmektedir. Ancak tutulan hemisfer tarafı belirleyici bir faktör değildir. Prospektif çalışmamızda, lezyon ve hareket etme sorununun etkisi daha iyice değerlendirilecektir.

Anadatia Kilimeler
Omuz El Sendromu, Omuz Subluksasyonu, Beyin Lezyon Lokalizasyonu, Beyin BT.

Abstract
Aim
The effect of brain lesion location and involved brain side in the development of shoulder subluxation (SS) and shoulder hand syndrome (SHS) is still unclear. The aim of the current study was to evaluate the relationships of SS and SHS with brain lesion location and stroke side.

Material and Methods
The inpatient files of the hemiplegic patients, who were hospitalized for rehabilitation, were evaluated retrospectively. Brain lesion location and involved hemisphere side were assessed by brain CT at the insult time. Brain lesion location groups were comprised as following 4 groups which included 35 patients in each group. Group I: Small superficial infarct. This type infarcts involve small area infarct (no more than 20 mm in diameter) on a cerebral lobe, such as a small right or left frontal lobe infarct. Group II: Large superficial infarct. Infarcts more than 20 mm in diameter on a cerebral lobe or two or more lobes of one cerebral hemisphere, such as a frontoparietal infarct in the left cerebral hemisphere or frontotemporo-parietal infarct in right or left cerebral hemisphere. Group III: Deep infarct. Infarcts of the internal capsule, basal ganglia or thalamus. Group IV: Combination of deep and large superficial infarcts (combined group II+III). Internal capsule or basal ganglia or thalamic infarct combined with fronto-temporal or fronto-parietal or temporo-parieto-occipital infarct on one cerebral hemisphere. A total of 140 hemiplegic patients then divided into 2 groups according to the development of SS and/or SHS.

Results
A significant correlation was found between brain lesion locations and SS and/or SHS development. The groups with combined deep and large superficial, and large superficial infarcts showed more frequent SS and/or SHS development. Patients with SS and/or SHS had got low stages at baseline in the upper extremity according to upper extremity Brunstroma level, upper extremity Ashworth stage and low stages of functional independence measure (FIM) scores. There wasn’t significant difference in shoulder problem development in terms of involved hemisphere side.

Conclusions
In our study, brain lesion location especially combined deep and large superficial, and large superficial infarcts seem to be a predictor factor for development of SS and/or SHS. But involved hemisphere side is not a predictor factor. Prospective trials are needed to make definite conclusions.

Keywords
Shoulder Hand Syndrome, Shoulder Subluxation, Brain Lesion Location, Brain CT.
Introduction
Shoulder subluxation (SS) and shoulder-hand syndrome (SHS) are frequent complications in patients with post-stroke hemiplegia. Paralysis of muscles around the shoulder joint may lead to SS and SHS [1]. Considering that SS and SHS creates negative effects on functional outcomes, it becomes very important to identify risk factors, to take measures relevant to SS and SHS, to take into account all clinical findings and functional anatomy, to assure implementation of conventional rehabilitation and other therapeutic methods and to establish an appropriate therapy plan and targets [1,2]. SS and SHS may be preventable if risk factors can be identified and appropriate prophylaxis applied [2].

SS most commonly occurs during the flaccid stage after stroke [3]. The reported incidence of SS in hemiplegic patients after stroke varies between 17% and 84% [4,5]. Some researchers found an increased incidence of SHS of the upper limb associated with SS [2].

SHS is considered to be one of the most disabling complications after stroke [6]. It is characterized by severe shoulder pain, swelling of the hand, vasomotor and sudomotor instability, joint contractures, and subsequent functional limitation of the affected extremity. Diagnostic criteria requires only subjective and historical signs and symptoms [7,8]. More than 12.5% of hemiplegic patients’ rehabilitation programs are often seriously hampered by the development of SHS [9]. It is believed to involve longstanding autonomic nervous system disturbance caused by brain damage [10].

The effect of brain lesion location and involved brain side in the development of SS and/or SHS is still unclear. The aim of the current study was to evaluate the relationships of SS and/or SHS with brain lesion location and stroke side. And to assess the effect of SS and/or SHS on the upper extremity recovery.

Methodology
Patient selection
We retrospectively evaluated inpatient chart files of 656 hemiplegic patients hospitalized in rehabilitation clinic of the university hospital for hemiplegia rehabilitation. Inclusion criteria of the study were as follows:
(1) hemiplegia or hemiparesis due to a stroke experienced for the first time, (2) age between 45 and 80 years, (3) Sufficient data in the inpatient chart files for study parameters before and after the rehabilitation program.

Exclusion criteria were as follows (1) patients with history of other disorders of central nervous system, including vascular malformations, tumors, multiple sclerosis, infectious diseases of central nervous system and history of previous head injury, ataxia, disthonia, dyskinesia and accompanying lower motor neuron or peripheral neural lesions in inpatient chart files. (2) Insufficient data in the inpatient chart files for study parameters. (3) Absence of brain CT scan at the insult time. (4) Hemorrhagic strokes.

Brain lesions, hemisphere side and locations, according to CT scan findings, were classified into the following four groups:

Group I: Small superficial infarct. This type infarcts involve small area infarct (no more than 20 mm in diameter) on a cerebral lobe, such as a small right or left frontal lobe infarct.

Group II: Large superficial infarct. Infarcts more than 20 mm in diameter on a cerebral lobe or two or more lobes of one cerebral hemisphere, such as a frontotemporal infarct in the left cerebral hemisphere or frontotemporoparietal infarct in right or left cerebral hemisphere.

Group III: Deep infarct. Infarcts of the internal capsule, basal ganglia or thalamus.

Group IV: Combination of deep and large superficial infarcts (combined group II+III). Internal capsule or basal ganglia or thalamic infarct combined with fronto-temporal or fronto-parietal or temporo-parieto-occipital infarct on one cerebral hemisphere.

Clinical evaluations
In addition to the demographic characteristics (sex, age) of the patients involved hemisphere side and side of hemiplegia, disease duration, duration of hospital stay were recorded.

The parameters of the study evaluations were as follows:
1. Spasticity in hemiplegic upper extremity measured and recorded using the Ashworth scale A5, on a scale of 0–4.
2. Motor status in the upper extremity graded on a scale of 1–6 according to the Brunnstrom Stage (BS).
3. Functional level for daily life activities were evaluated by the Functional Independence Measurement (FIM). Self-care, spincter control, transfers and locomotion scores added to calculate the FIM motor subscore.
4. Brain lesion side and location according the CT scan at the insult time.
5. Presence of shoulder-hand syndrome, shoulder subluxation or co-existence according to the inpatient rehabilitation file chart.

Upper limb motor function was assessed by Brunnstrom recovery stages at admission. The lowest stage (flaccid stage and no voluntary movement) was stage I and highest stage (isolated joint movement) was stage VI. Spasticity at the upper limb was evaluated on a 5-point Ashworth scale (range, 0–4). The scale is rated as follows: 0_ no increase in muscle tone; 1_ slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of ROM when the affected part is moved in flexion or extension; 2_ more marked increase in muscle tone through most of the ROM, but the affected part is easily moved; 3_ considerable increase in muscle tone and passive movement is difficult; and 4_ affected part is rigid in flexion or extension.

According to these radiologic classification criteria and clinical evaluations mentioned above, we retrospectively evaluated 656 hemiplegic patients’ inpatient file charts and CT at the insult time. And when we reached 35 patients in each radiologic location group (a total of 140 patients), we evaluated patients in terms of shoulder hand-syndrome, shoulder subluxation, shoulder-hand syndrome+shoulder subluxation co-existence at admission to the rehabilitation. Then we gathered all patients with shoulder problems of these four groups in one group in order to examine the relationships between these shoulder problems with age, FIM scores, hospital stay, involved hemisphere side, upper extremity Brunnstroma and Ashworth stages at admission to the rehabilitation.

Statistical Analysis
Student t test was used to compare constant variables between the two groups with shoulder problems and without shoulder problems. Pearson correlation coefficient was computed to determine linear relationships among these variables. The four groups of brain lesion location were compared in terms of proportions using proportion comparing with Z test or Chi-square
Table 1. Demographic data of the patients with shoulder problems (shoulder subluxation and/or shoulder-hand syndrome) (PatwSpat) and without shoulder problems (PatwoSpat).

<table>
<thead>
<tr>
<th></th>
<th>PatwSpat (n=65)</th>
<th>PatwoSpat (n=75)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Male/Female=39/26)</td>
<td>(Male/Female=48/27)</td>
</tr>
<tr>
<td>Age</td>
<td>64.88(79.1)</td>
<td>62.22(74.2)</td>
</tr>
<tr>
<td>BMI</td>
<td>26.61(3.02)</td>
<td>26.55(3.02)</td>
</tr>
<tr>
<td>Dis.durat</td>
<td>43.72(16.19)</td>
<td>46.65(15.76)</td>
</tr>
<tr>
<td>Dur.Hos. stay</td>
<td>37.53(13.42)</td>
<td>37.45(12.35)</td>
</tr>
<tr>
<td>TotalFIM</td>
<td>41.86(3.71)</td>
<td>42.05(3.72)</td>
</tr>
<tr>
<td>CogFIM</td>
<td>21.08(1.59)</td>
<td>22.58(1.98)</td>
</tr>
<tr>
<td>MotorFIM</td>
<td>21.06(1.89)</td>
<td>23.72(2.13)</td>
</tr>
</tbody>
</table>

*: p< 0.05. **: p<: 0.01

Table 2. Paerson correlation coefficients of constant variables in PatwSpat and PatwoSpat groups.

<table>
<thead>
<tr>
<th></th>
<th>PatwSpat (n=65)</th>
<th>PatwoSpat (n=75)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Age</td>
<td>BMI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1</td>
<td>-0.024</td>
</tr>
<tr>
<td>BMI</td>
<td>0.134</td>
<td>0.232</td>
</tr>
<tr>
<td>Dis.durat</td>
<td>0.023</td>
<td>0.144</td>
</tr>
<tr>
<td>Dur.Hos. stay</td>
<td>-0.053</td>
<td>-0.232</td>
</tr>
<tr>
<td>TotalFIM</td>
<td>-0.271*</td>
<td>-0.151</td>
</tr>
<tr>
<td>CogFIM</td>
<td>-0.209</td>
<td>-0.011</td>
</tr>
<tr>
<td>MotorFIM</td>
<td>-0.125</td>
<td>-0.026</td>
</tr>
</tbody>
</table>

*: p< 0.05. **: p<: 0.01

Table 3. Comparing of the proportions of the hemiplegic patients who developed shoulder subluxation (SS) and shoulder-hand syndrome (SHS) in different groups.

<table>
<thead>
<tr>
<th></th>
<th>PatwSpat (n=65)</th>
<th>PatwoSpat (n=75)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Male/Female=39/26)</td>
<td>(Male/Female=48/27)</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>BMI</td>
</tr>
<tr>
<td>Age</td>
<td>0.270*</td>
<td>0.212</td>
</tr>
<tr>
<td>BMI</td>
<td>-0.024</td>
<td>-0.156</td>
</tr>
<tr>
<td>Dis.durat</td>
<td>0.023</td>
<td>0.144</td>
</tr>
<tr>
<td>Dur.Hos. stay</td>
<td>-0.053</td>
<td>-0.232</td>
</tr>
<tr>
<td>TotalFIM</td>
<td>-0.271*</td>
<td>-0.151</td>
</tr>
<tr>
<td>CogFIM</td>
<td>-0.209</td>
<td>-0.011</td>
</tr>
<tr>
<td>MotorFIM</td>
<td>-0.125</td>
<td>-0.026</td>
</tr>
</tbody>
</table>

*: p< 0.05. **: p<: 0.01

Results

A total of 140 acute stroke patients (53 female and 87 male; age range, 45–80 years) included in this study. Demographic data of the patients with shoulder problems (PatwSpat) (SS, SHS or coexistence) and the patients without shoulder problems (PatwoSpat) are shown in Table 1. Statistically significant differences were found in between the 2 groups in terms of age (p<0.05) and FIM scores (p<0.01). FIM scores were significantly lower while age higher in patients with shoulder problems than in patients without shoulder problems respectively (Table 1). There was a statistically significant negative correlation (according to Pearson correlation coefficients) between total FIM scores and age in PatwSpat group but not in PatwoSpat group (Table 2). Table 2 shows the Pearson correlation coefficients of constant variables of patients in PatwSpat and PatwoSpat groups.

Numbers of the hemiplegic patients who developed shoulder subluxation and shoulder-hand syndrome of the groups were as follows; in the first group (Small superficial infarct), 11 (31.4%) patients out of 35 patients (3 shoulder-hand syndrome (SHS), 6 shoulder subluxation (SS), 2 shoulder hand syndrome and shoulder subluxation coexistence (SHS+SS coext.); in the second group (Large superficial infarct), 18 (51.4%) patients out of 35 patients (6 SHS, 9 SS, 3 SHS+SS coext.); in the third group (Deep infarct), 7 (20%) patients out of 35 patients (3 SHS, 3 SS, 1 SHS+SS coext.); in the fourth group (Combination of deep and large superficial infarcts (combined group II+III), 29 (82.8%) patients out of 35 patients (9 SHS, 12 SS, 8 SHS+SS coext.). Statistically significant differences were found between infarct location and development of shoulder problems. Table 3 shows number of the hemiplegic patients who developed SS and SHS and proportion comparisons of the groups.
The ratios of right hemisphere (RH) to left hemisphere (LH) in the patients with shoulder problems were 34/31 and in patients without shoulder problems were 37/38, respectively (Table 4). Right hemisphere involvement was slightly more in PatwSpat group but no statistically significant difference was found in terms of side of involved hemisphere between the 2 groups in Fischer’s exact Chi-Square test (p=0.738).

More flaccid stages of Ashworth were found in patients with shoulder problems while more spastic stages were found in patients without shoulder problems respectively. This difference was statistically significant (Table 5).

Statistically significant lower stages of upper extremity Brunnstrom motor recovery stages were found in patients with shoulder problems (Table 6).

Discussion
In this study, the effects of brain lesion location and involved brain side on development of SS and SHS were evaluated in stroke patients. To the best of our knowledge the current study is the first trial that reports SS and SHS incidence according to the brain lesion location and side in the CT. We found that brain lesion location especially combined deep and large superficial, and large superficial infarcts (but not involved hemisphere side) seem to be a predictor factor for development of SS and/or SHS.

According to the results of this study, the group of patients with small superficial infarct and deep infarct (group I and group III) showed a trend less likely to have SS with or without SHS (31.4%, 20.0% respectively) whereas other groups of patients with large superficial infarct and combined large superficial infarct and deep infarct (group II and group IV) showed a trend more likely to have SS with or without SHS (51.4%, 82.8% respectively). The occurrence of hemiplegic shoulder problems after stroke is reported to be as high as 84 % [12]. As in our results it is linked to increased length of hospital stay, poor functional recovery of the upper limb [13]. Given both the high incidence and limited success of current treatment in addressing hemiplegic shoulder problems, it is important that predisposing factors of shoulder problems development for its prevention are examined [14].

We found a rate of 31.4% SS in hemiplegic patients. Thus the current study also supports the previous studies reported SS is a frequent complication in patients with post-stroke hemiplegia. Paci et al found that SS was present in 48.6% post-stroke patients and correlated significantly to shoulder pain. They concluded that SS is a factor associated with shoulder pain development and with arm motor recovery and should be treated in the acute stage of hemiplegia [15]. Our results also support investigations suggest a higher incidence of SS in patients with complete or severe arm hemiplegia [16]. Especially we found up to 57% SS of the patients in the group 4 (combined large superficial and deep infarcts) while 22.8% in the group 1 (small superficial infarct). Our results also support previous studies reported that SS was correlated with low upper extremity Brunnstrom’s motor stage [17].

In this study we also investigated the effect of brain lesion location in the development of SHS in hemiplegic patients. SHS is a painful condition commonly observed after stroke that can hamper the functional recovery required to perform motor tasks, such as activities of daily living, and that consequently leads to disability [8]. According to epidemiological data, SHS occurs in 12–70% of patients with hemiplegia following stroke [8,18]. We found a rate of 25% in hemiplegic patients. This rate was 48.5% in patients of combined large and deep infarcts. Eto et al. studied seven autopsy cases of SHS following hemiplegia with regard to cerebral localization and found a lesion in the premotor area appears chiefly responsible for the primary mechanism of the SHS in post-stroke hemiplegia [19].

In this current study we found that patients with SS and SHS hospitalized longer duration, and had poor recovery of the arm. Two study showed that shoulder problems affected stroke outcome in a negative way and the presence of shoulder problems is strongly associated with prolonged hospital stay and poor recovery of the arm function in the first 12 weeks after stroke [13–15]. Another study, however, showed no relationship between shoulder problems and the Barthel Index scores [20]. SS may be a causative factor for SHS. Therefore, prevention and appropriate treatment of glenohumeral joint subluxation should be included in rehabilitation of hemiplegic patients. In this current study we found 25% SHS in 140 hemiplegic patients, and 40% of these patients together with SS. We also found up to 22.8% SS and SHS coexistence in the group of combination of deep and large superficial infarcts (group IV) while this proportion was 5.7% in the group of small superficial infarcts (group I). Our study supports the findings of previous studies on the effect of SS favoring SHS, namely the extent of the motor deficit, sensory deficits, and shoulder subluxation [2]. Dursun et al. reported that shoulder subluxation was found in 74.3% of the SHS and 40% of the non-SHS group [2].

In this current study we found that in patients with right hemisphere involvement was slightly more prone to have SHS and/or SS with rates of 54.2%, 45.8% respectively. But this didn’t reach to a statistically significant level. The reason of increased rate of SS and/or SS in right hemisphere involvement in this study may be increased sympathetic activity in right hemispheric infarcts. Strittmatter et al. [21] studied sympathetic function prospectively assessed by determining plasma norepinephrine and epinephrine in ischaemic stroke patients and found that plasma catecholamines especially norepinephrine was significantly higher in right hemispheric infarcts and they stated that hemispheric lateralization in autonomic control should be taken into account in the management of stroke because of an increased susceptibility to cardio-autonomic dysfunction in patients with...
right hemisphere stroke. Ay et al also found that infarctions in specific brain regions including the right hemisphere are associated with elevated serum cardiac troponin T level indicative of myocardial injury [22]. Although thalamic brain lesions are associated with allodynia (central post-stroke pain, CPSP), Misra et al. concluded that the symptoms and severity of clinical spectrum of CPSP in thalamic and extrathalamic stroke do not differ significantly [23]. The reported frequency of CPSP up to 35% in thalamic lesions [24,25]. Our results also showed that patients with deep lesions (infarcts of the internal capsule, basal ganglia or thalamus) are less likely to develop SHS. One reason of why CPSP is high incidence in deep lesions in the other studies while SHS is low incidence in this current study might be that CPSP (allodynia) alone seen in thalamic infarcts is not sufficient for SHS diagnosis. According to the results of the current study we recommend that physicians and therapists should aware of increased risk of shoulder problems and carefully plan and supervise shoulder rehabilitation in stroke patients especially with poor motor function and large superficial infarcts and large superficial infarcts combined with deep infarcts.

In conclusion; Brain lesion location seem to be a predictor factor for development of SS and SHS. The results of the current study indicate that large superficial infarcts and combined deep and large superficial infarcts in the CT indicate the more likely hemiplegic patient to develop SS or SHS. SS and/or SHS are related with low stages in the upper extremity according to the upper extremity Brunnstrom levels and Ashworth stages. Our results suggest that involvement of right or left hemisphere does not seem to be a predictor of the development of shoulder subluxation and shoulder hand syndrome. Still, prospective trials are needed to make definite conclusions.

References