Clinical and radiologic asymmetric arthritic differences between paralyzed and nonparalyzed limbs of stroke patients have been reported. Arthritic pathology aggravates motor dysfunction and compromises rehabilitation. Musculoskeletal ultrasonography plays an important role in showing soft tissue and the articular cartilage of the knee. Fifty-nine patients with either ischemic or hemorrhagic stroke-induced right or left hemiplegia were recruited to evaluate soft-tissue and intra-articular cartilage changes in hemiplegic knees of stroke patients using ultrasonography. An additional 15 subjects (30 knees) without knee disease or a history of knee trauma or surgery were used as controls. There were significant differences in suprapatellar effusion and patellar tendinitis between hemiplegic and nonhemiplegic knees. Suprapatellar effusion and pes anserinus tendinitis were correlated with Brunnstrom stage. The length of time since stroke onset was not significantly correlated with positive ultrasonographic findings in hemiplegic knees. In conclusion, ultrasonography is useful for detecting periarticular soft-tissue changes and intra-articular lesions in hemiplegic knees of stroke patients.


Cerebrovascular accidents frequently cause upper motor neuron syndrome, including weakness, spasticity, and abnormal gait pattern or synergistic movement. The impairment of lower limb function results in an asymmetric and interrupted hemiplegic gait because patients prefer to bear weight on the unaffected limb. Biomechanical changes in both limbs lead to soft-tissue injuries and arthritic cartilage changes may be suspected [1,2]. Segal et al reported that paralyzed hands show significantly fewer osteoarthritic changes than nonparalyzed hands, both clinically and radiologically, and that the severity of paralysis and loss of muscle strength correlate with the degree of osteoarthritis (OA) asymmetry [3]. In rheumatoid arthritis, where symmetry is a diagnostic criterion, it has been reported that impairment of the nervous system alters arthritis symmetry [4]. In arthritis of the lower limbs, involvement of the knee is the most common cause of disability in older people or hemiplegic patients. In patients with hemiplegia, knee arthritis induces joint pain, limited range of motion (ROM), loss of muscular strength, and eventual motor dysfunction. Therefore, early detection of arthritis in stroke patients is important, and ultrasonography plays an important role in screening and follow-up [5].

Roentgenography and magnetic resonance imaging (MRI) are commonly used to evaluate arthritic knees. While radiographs of the knee are usually obtained first, these provide little information on soft-tissue structures. Though an MRI can provide more information about soft-tissue changes in knees, including cartilage, tendons, ligaments, menisci, and bone marrow, it is expensive and there is a lack of dynamic real-time assessment [6].

Ultrasonography is increasingly advocated as a valuable diagnostic tool for evaluating the knee joint. It has many
advantages, including its noninvasive nature, low cost, portability, dynamic real-time assessment, and easy side-to-side comparison. It provides quality images of the knee joints, including the synovial sac, tendons, ligaments, meniscus, and articular cartilage. In addition, the use of extended field imaging has helped in the imaging of larger anatomic structures; and split-screen imaging is beneficial in comparing the changes in both knees [5,7,8].

Currently, no paper has discussed soft-tissue and cartilage changes in stroke patients. This study was designed to use ultrasound to investigate structural changes in bilateral knees of hemiplegic patients, to analyze the correlation between sonographic findings and both post-stroke duration and motor status, respectively. The probable risk factors for knee joint lesions were also assessed.

**Methods**

**Subjects**

Fifty-nine ischemic or hemorrhagic stroke patients with right or left hemiplegia were recruited between January and April 2004. The bilateral knees were evaluated using high-resolution ultrasound. Patients were excluded if they had knee injury in the past 6 months, a history of knee OA or other form of arthritis, or a history of knee surgery. An additional 15 subjects (30 knees) without knee disease or a history of knee surgery were used as controls. In terms of age and sex ratio, the control group was not significantly different from the experimental group, as determined by independent-samples $t$ test and Fisher’s exact test.

Sex, age, height, weight, body mass index (BMI), diabetes mellitus status, hypertension, motor status (Brunnstrom stage), and the interval from the onset of hemiplegia were recorded. The severity of knee pain was measured using a visual-analog pain scale, and signs of local inflammation and ROM of the knee joint were also assessed.

**Ultrasound assessment**

Ultrasoundography was performed on bilateral knees using a real-time 5–12 MHz high-resolution linear scanner (HDI 1500, Advanced Technology Laboratories, Bothell, WA, USA). The techniques used in this study have previously been described [5,7–11].

To examine the suprapatellar pouch and patellar tendon, the patient was placed in a supine position with knee flexion of 30°. The transducer was swept from the suprapatellar pouch to the tibial tuberosity, to give both longitudinal and transverse images. The pes anserinus tendon insertion, 2.5–3 cm distal to the medial joint line, was located with the transducer placed longitudinally.

The deep infrapatellar bursa was examined with the transducer positioned longitudinally between the patellar tendon and tibia. The femoral condylar cartilage and knee joint space were viewed with subjects supine and with knees held at maximum flexion (about 120°); the superior margin of the medial and lateral femoral condyle and the center of the intercondylar groove were used as markers.

**Ultrasonographic diagnostic criteria**

Suprapatellar effusion was defined as transducer-compressible localized anechoic or hypoechoic fluid in the suprapatellar pouch (Figure 1).

Ultrasoundographic images of acute patellar tendinitis show the tendon to be thickened and hypoechoic. In chronic tendinitis, the tendon is thickened, heterogeneous, and hypoechoic. The maximal thickness of the normal patellar tendon is about 4–5 mm and the width is about 20–25 mm; thickening is defined as patellar tendon thickness greater than 5 mm (Figure 2) [12].

Pes anserinus tendinitis was characterized by thickening and loss of the normal fibrillar echotexture. Bursitis was defined as a circumscribed anechoic fluid collection of 2 mm or more (Figure 3) [13,14].

Baker’s cyst was defined as a hypoechoic mass in the popliteal space, measuring at least 1 cm in two dimensions. The characteristics are visualization of the stem of the cyst, originating in the medial aspect of the popliteal fossa, between the semimembranous tendon and the medial gastrocnemius head.

The characteristic sonographic features of osteoarthritic cartilage are loss of the normal sharpness of the synovial space cartilage interface, loss of clarity of the cartilaginous layer, narrowing of the joint cartilage (thickness < 2 mm), and increased intensity of the posterior bone–cartilage interface (Figure 4).

**Statistical analysis**

Chi-squared analysis was used to determine the differences in ultrasonographic abnormalities between the hemiplegic and nonhemiplegic knees, in the durations from the onset of hemiplegia, and in the motor status of the hemiplegia. We defined $p$ less than 0.05 as a significant difference. The independent samples $t$ test was used to determine the difference in mean age between stroke patients and control subjects. Fisher’s exact test was used to determine the
Positive ultrasonographic changes were found in 54 (91.5%) affected knees, 51 (86.4%) sound knees, and 27 (90%) control knees. There was no significant difference between affected and sound knees ($p = 0.378$). Table 1 summarizes the sonographic findings.

**Extra-articular sonographic findings**

The incidences of suprapatellar effusion ($p = 0.04$) and patellar tendinitis ($p = 0.021$) were significantly higher on the affected side than the sound side, but there were no

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**RESULTS**

The 59 patients had a mean age of $60.2 \pm 12.7$ years (range, 38–87 years) and a male to female ratio of 41:18. The time from stroke onset ranged from 2 weeks to 9 years (mean, 2 years $\pm$ 2 weeks). The 15 control subjects had a mean age of $60.5 \pm 7.4$ years (range, 39–87 years) and a male to female ratio of 10:5. There were no significant differences among stroke patients and control subjects in terms of mean age ($p = 0.88$) or sex ratio ($p = 1.00$).

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**Figure 1.** Suprapatellar effusion (longitudinal view). (A) Normal suprapatellar bursa without fluid accumulation. (B) Hypoechoic fluid effusion (E, arrow) between the quadriceps tendon (Q), upper pole of the patella (P), and the femur (F).

**Figure 2.** Patellar tendinitis (longitudinal view). (A) Normal, homogeneous, 4.5-mm-thick (between calipers) patellar tendon (P). (B) Thickening of and hypoechoic changes in the 6-mm-thick (between calipers) patellar tendon (arrow) inserting into the tibial tuberosity (T).
significant differences between the sound side and controls ($p = 0.832$ and $0.829$, respectively) (Table 1). Additionally, there were no significant differences in the incidences of deep infrapatellar tendon bursitis ($p = 0.729$) or pes anserinus tendinitis ($p = 0.387$) between affected and sound knees or between sound and control knees ($p = 0.361$ and $0.677$, respectively).

**Intra-articular sonographic findings**

There were no significant differences in the incidences of joint space narrowing ($p = 0.822$), hyaline cartilage heterochogenity ($p = 0.285$), blurred cartilage–tissue margin ($p = 0.847$), or irregular cartilage–bone margin ($p = 0.837$) between affected and sound knees or between sound and control knees ($p = 0.729, 0.871, 0.571, 0.462$, respectively) (Table 1).

**Comparison of intra- and extra-articular sonographic findings**

There were no significant differences in the incidences of intra-articular ($p = 0.577$) or extra-articular ($p = 0.223$), or a combination of the two ($p = 0.305$) sonographic findings between affected and sound knees or between sound and control knees ($p = 0.589, 0.711, 0.199$, respectively) (Table 2).

Extra-articular sonographic findings were noted in more
Table 1. Comparison of sonographic findings in affected and sound knees of stroke patients and control knees

<table>
<thead>
<tr>
<th></th>
<th>Affected, n (%)</th>
<th>Sound, n (%)</th>
<th>Control, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suprapatellar effusion</td>
<td>30 (50.8)</td>
<td>19 (32.0)*</td>
<td>9 (30.0)</td>
</tr>
<tr>
<td>Patellar tendinitis</td>
<td>27 (45.7)</td>
<td>15 (25.0)*</td>
<td>8 (26.7)</td>
</tr>
<tr>
<td>Deep infrapatellar tendon bursitis</td>
<td>4 (6.0)</td>
<td>5 (8.4)</td>
<td>2 (6.7)</td>
</tr>
<tr>
<td>Pes anserinus tendinitis</td>
<td>16 (27.1)</td>
<td>12 (20.0)</td>
<td>5 (16.0)</td>
</tr>
<tr>
<td>Baker’s cyst</td>
<td>9 (15.2)</td>
<td>10 (16.9)</td>
<td>4 (14.3)</td>
</tr>
<tr>
<td>Joint space narrowing</td>
<td>12 (20.3)</td>
<td>13 (22.0)</td>
<td>7 (25.0)</td>
</tr>
<tr>
<td>Hyaline cartilage heteroechogenicity</td>
<td>17 (28.8)</td>
<td>12 (20.3)</td>
<td>6 (20.0)</td>
</tr>
<tr>
<td>Blurred cartilage–tissue margin</td>
<td>21 (35.6)</td>
<td>20 (33.8)</td>
<td>10 (33.3)</td>
</tr>
<tr>
<td>Irregular cartilage–bone margin</td>
<td>17 (28.8)</td>
<td>16 (27.1)</td>
<td>6 (20.0)</td>
</tr>
</tbody>
</table>

*p < 0.05 between affected and sound knees.

Table 2. Comparison of intra- and extra-articular sonographic findings in affected and sound knees in stroke patients and control knees

<table>
<thead>
<tr>
<th></th>
<th>Affected n (%)</th>
<th>Sound n (%)</th>
<th>Control n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nil</td>
<td>6 (10.2)</td>
<td>9 (15.3)</td>
<td>3 (10.0)</td>
</tr>
<tr>
<td>Intra</td>
<td>27 (45.8)</td>
<td>25 (42.3)</td>
<td>15 (50.0)</td>
</tr>
<tr>
<td>Extra</td>
<td>45 (76.3)</td>
<td>39 (66.1)</td>
<td>20 (66.7)</td>
</tr>
<tr>
<td>Intra and extra</td>
<td>19 (32.2)</td>
<td>14 (23.7)</td>
<td>8 (26.7)</td>
</tr>
</tbody>
</table>

Table 3. Correlation of intra- and extra-articular sonographic findings in affected knees with risk factors for stroke

<table>
<thead>
<tr>
<th></th>
<th>Diabetes mellitus, %</th>
<th>Hypertension, %</th>
<th>Sex, %</th>
<th>BMI, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+</td>
<td>-</td>
<td>p</td>
<td>+</td>
</tr>
<tr>
<td>Intra</td>
<td>11.9</td>
<td>32.2</td>
<td>0.595</td>
<td>27.1</td>
</tr>
<tr>
<td>Extra</td>
<td>94.4</td>
<td>68.3</td>
<td>0.03*</td>
<td>50.8</td>
</tr>
</tbody>
</table>

*p < 0.05. BMI = body mass index.

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The lack of difference in the incidences of various sonographic findings between nonparalyzed knees and control knees suggested that nonparalyzed knees did not suffer more soft-tissue injury due to compensatory gait. The greater incidence of suprapatellar effusion in hemiplegic knees than nonhemiplegic knees (50.8% vs 32%; *p = 0.04*) may have been due to a biomechanical mechanism (such as underuse of the paralyzed knee), compensatory behavior (such as overuse of the nonparalyzed knee), or a combination of both [15,16].

The transverse section of the patellar tendon is crescent shaped with a convex anterior and a rectilinear posterior margin. In sports medicine, tendinitis results from over-stress and repeated minor trauma; in acute tendinitis, tendon volume is increased, echogenicity is decreased, and contours may be blurred. Osgood-Schlatter disease, caused by repetitive injury and overuse, occurs in adolescents when the patellar tendon inserts into the tibial tubercle [12]. In this study, the incidence of patellar tendinitis group were 23.2%, 19.6%, 16.1%, and 41.1% respectively (*p = 0.597*).

**DISCUSSION**

The incidences of sonographic findings according to Brunnstrom stage are shown in Table 4. There were higher incidences of suprapatellar effusion in stages II, III, and IV than in stages I, V, and VI (*p = 0.034*). There were also higher incidences of pes anserinus tendinitis in stages III, IV, and V than in stages I, II, and VI (*p = 0.042*).

We divided the duration from the onset of stroke into four groups: 0–1 months, 2–3 months, 3–6 months, and longer than 12 months. The number of subjects in each group was 13, 12, 9, and 25, respectively. The incidences of positive ultrasonographic findings in affected knees in each
was significantly higher in hemiplegic knees than non-hemiplegic knees (45.7% vs 25%; \( p = 0.021 \)). This may have been due to incorrect knee joint posture or movement following repeated minor trauma.

Usually resulting from meniscal tears and degenerative or inflammatory arthropathy, Baker’s cysts represent fluid accumulation in the gastrocnemius–semimembranosus bursa. Popliteal cysts often complicate rheumatoid arthritis in adults, where the cysts may enlarge and rupture into the soft tissue of the calf, causing severe pain and disability [17,18]. Cyst formation is strongly correlated with the presence of intra-articular pathology, and cyst formation is believed to be due to either trauma or an underlying inflammatory process [5,18]. Popliteal cysts are readily documented in children with knee effusions using ultrasonography, and their presence and evolution correlate with the size of the suprapatellar effusion [5,7,17,18]. In this study, there was no significant difference in the incidence of popliteal cysts in hemiplegic knees (15.2%) and non-hemiplegic knees (16.9%).

Most patients clinically diagnosed with pes anserinus tendinitis or bursitis syndrome (PATB syndrome) do not have morphologic ultrasonographic changes of the pes anserinus tendons. The etiology of medial knee pain is probably a result of a complex relationship between structural changes along with the peripheral and central pain processing mechanism [13,14]. In this study, the incidence of pes anserinus tendinitis in hemiplegic knees was 27.1%, compared with 20% in non-hemiplegic knees (\( p = 0.387 \)).

Type 2 diabetic patients may have muscle weakness at the ankle and knee related to the presence and severity of peripheral neuropathy [19]. In the present study, extra-articular sonographic findings were noted in 94.4% of patients with diabetes and 68.3% of patients without (\( p = 0.03 \)) (Table 3), indicating that patients with diabetes are at a greater risk of extra-articular soft-tissue damage, perhaps due to poor limb control or impairment of sensory protection.

BMI, a relative measure of weight, has been reported to have a strong association with OA or periarticular soft-tissue lesions of the knee in a number of studies [21]. In the present study, there were no significant correlations between BMI and ultrasonographic findings (Table 3), either intra-articular or extra-articular, which is contrary to previous studies. It is possible that the relationship was not apparent due to the small sample size and a larger study would be needed to corroborate or refute these findings.

The time from the onset of stroke was not significantly correlated with the presence of any ultrasonographic findings in hemiplegic knees (\( p = 0.597 \)), or with findings of suprapatellar effusion (\( p = 0.548 \)), patellar tendinitis (\( p = 0.447 \)), or intra-articular cartilage changes (\( p = 0.301 \)). This showed that the length of time from stroke onset was not a major risk factor for ultrasonographic changes. The average time from the onset of stroke in this study was 2 years. The effect of time on cartilage may not be obvious over this

**Table 4. Incidence of various sonographic findings in patients with different motor status, as defined by Brunnstrom stage**

<table>
<thead>
<tr>
<th></th>
<th>II (n = 11)</th>
<th>III (n = 18)</th>
<th>IV (n = 19)</th>
<th>V (n = 9)</th>
<th>VI (n = 2)</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suprapatellar effusion, %</td>
<td>33.3</td>
<td>26.7</td>
<td>30.0</td>
<td>6.7</td>
<td>3.3</td>
<td>0.034*</td>
</tr>
<tr>
<td>Patellar tendinitis, %</td>
<td>7.4</td>
<td>40.7</td>
<td>33.3</td>
<td>14.8</td>
<td>3.7</td>
<td>0.275</td>
</tr>
<tr>
<td>Deep infrapatellar tendon bursitis, %</td>
<td>25.0</td>
<td>0</td>
<td>50.0</td>
<td>25.0</td>
<td>0</td>
<td>0.692</td>
</tr>
<tr>
<td>Pes anserinus tendinitis, %</td>
<td>6.3</td>
<td>25.0</td>
<td>31.3</td>
<td>37.5</td>
<td>0</td>
<td>0.042*</td>
</tr>
<tr>
<td>Baker’s cyst, %</td>
<td>0</td>
<td>22.2</td>
<td>66.7</td>
<td>11.1</td>
<td>0</td>
<td>0.158</td>
</tr>
<tr>
<td>Joint space narrowing, %</td>
<td>25.0</td>
<td>25.0</td>
<td>41.0</td>
<td>8.3</td>
<td>0</td>
<td>0.758</td>
</tr>
<tr>
<td>Hyaline cartilage heteroechogenity, %</td>
<td>11.8</td>
<td>35.3</td>
<td>35.3</td>
<td>17.6</td>
<td>0</td>
<td>0.781</td>
</tr>
<tr>
<td>Blurred cartilage-tissue margin, %</td>
<td>19.0</td>
<td>42.9</td>
<td>28.6</td>
<td>9.5</td>
<td>0</td>
<td>0.467</td>
</tr>
<tr>
<td>Irregular cartilage-bone margin, %</td>
<td>17.6</td>
<td>41.2</td>
<td>35.3</td>
<td>5.9</td>
<td>0</td>
<td>0.532</td>
</tr>
</tbody>
</table>

\*\( p < 0.05 \). No patients had Brunnstrom stage I motor status.
short period. Patients with a longer time interval from the onset of stroke should be recruited to observe long-term effects on cartilage changes.

Motor status was correlated with two sonographic findings, suprapatellar effusion and pes anserinus tendinitis. Both were more common in Brunnstrom stages III and IV, where patients begin to regain motor function. This showed that injuries to hemiplegic knees occurred more easily when stroke patients initially regained motor control of their knees. Incorrect posture, motor pattern, knee instability, and over training of the hemiplegic lower limb may have led to this phenomenon.

In conclusion, suprapatellar effusion and patellar tendinitis were the major sonographic observations in the hemiplegic knees of stroke patients. In this study, diabetics had a higher risk for extra-articular sonographic findings and women had a higher risk for intra-articular sonographic findings. The amount of time from stroke onset was not correlated with sonographic findings, and long-term follow-up of cartilage changes is warranted.

REFERENCES

半身癱瘓患者患側膝關節之超音波影像特徵

楊照彬  李佳玲  陳天文  李素  翁銘正  黃茂雄
高雄醫學大學附設中和紀念醫院  復健科

中風病人雙側肢體呈現不對稱關節病變的現象已經在許多研究中被報告，而關節病變會加重運動功能喪失並且阻礙復健治療的進行。骨骼肌肉超音波對於軟組織和膝關節軟骨具有良好的診斷力。本篇研究主要是使用超音波來評估中風合併半身癱瘓病患的雙側膝關節軟組織及關節內軟骨的變化。59 位中風病患及 15 位健康對照組，分別以骨骼肌肉超音波 5-12 MHz 寬頻高解析度的探頭 (HDI 1500，Advanced Technology Laboratories，Bothell，WA，USA) 評估雙膝之影像變化。結果顯示關節上方液體積積 (suprapatellar effusion) 以及關節肌腱炎 (patellar tendinitis) 是偏癱側膝關節的主要超音波變化，且與非偏癱側比較呈現有統計學上的差異。在危險因子之分析結果顯示，糖尿病是發生關節外病變的危險因子，而女性則是發生關節內病變的主要危險因子。關節上方液體積積 (suprapatellar effusion) 和鵝足狀肌腱發炎 (pes anserinus tendinitis) 之變化與運動功能狀態 (Brunnstrom stages) 呈現有統計上的相關。然而，罹病時間長短和癱瘓側膝關節的超音波異常表現的發生率並沒有顯著相關。結論，超音波是可以當作評估中風患者膝關節軟組織病變的良好工具。

關鍵詞：超音波，半身偏癱，膝關節，中風

(高雄醫誌 2005;21:70–7)