Patterns of venous insufficiency after an acute deep vein thrombosis

INTRODUCTION

The initial treatment of deep vein thrombosis (DVT) is directed at the prevention of thrombus propagation, the development of pulmonary embolism, and of minimizing the recurrence. In the treatment of DVT, administration of heparin followed by oral anticoagulant therapy is well defined, and recent studies have shown that the period of 3 to 6 months is the optimal duration of oral anticoagulant therapy for proximal DVT. On the contrary, prevention of postthrombotic syndrome (PTS) has emerged because of increasing awareness of their impact on patient quality of life.

The factors that affect the development of PTS are not fully understood. Severe PTS might be caused by gravitational venous reflux, venous obstruction, or combination of these. The PTS, however, is difficult to predict in the acute phase of DVT. Individual anatomic venous segments in the lower extremity respond in a different way to the presence of thrombus. In the late phase, the presence of reflux in the femoral and popliteal vein is considered to contribute the development of PTS, but the role of an obstruction, reflux, or a combination of the two in the rest of venous segment remains unclear.

Recently, venous duplex scanning (VDS) has largely replaced contrast venography as the initial diagnostic test for DVT, with high sensitivity and specificity. VDS has been shown to be a reliable and accurate means of identifying lower extremity venous thromboembolism, using B–mode and color flow Doppler imaging. The VDS has also shown to be a reliable technique in the diagnosis of venous insufficiency of the lower extremities.

In this study, venous abnormalities after development of DVT, including compressibility as well as reflux of the lower extremity venous system, were evaluated with duplex scans. The purpose of this study was to investigate the rate of resolution within 12 months after the diagnosis of acute DVT in various vein segments and to estimate the development of venous valvular reflux by serial duplex scanning.

MATERIALS AND METHODS

Patients

From January 2002 to September 2003, a total of 83 limbs in 80 patients with duplex–confirmed acute DVT were prospectively evaluated with follow–up serial duplex scanning. After initial diagnosis, all patients were treated with intravenous unfractionated heparin for 5 to 14 days during the acute phase adjusted to maintain the activated partial thromboplastin time (APTT) at 1.5 to 2.5 times control, followed by oral warfarin for at least 8 weeks with INR level of 2–3. All patients were encouraged to ambulation, and graduated thigh–high compression stockings were applied immediately upon making diagnosis. The patients in this study were placed on follow–up at 1 month, 3 months, 6 months, and 1 year. The exclusion criteria included patients who did not fully ambulate due to medical reasons. Patients with arterial insufficiency, identified on the basis of an ankle–brachial pressure index of < 0.8, were also excluded from the study.

Venous duplex scans

The presence of DVT was diagnosed with duplex scans. A color duplex scanner (LOGIQ 500MD: GE Medical Systems, Milwaukee, WI, USA) with a 5–10 MHz transducer was used. Initially, each patient was placed supine in a reverse Trendelenburg position at 15°. Venous duplex scanning began at the common femoral vein (CFV), and moved to the femoral vein (FV) at the adductor canal. The anterior and posterior tibial veins (ATV and PTV) were also recorded. Afterwards, the patient was placed in a prone position with the knee flexed at 30°, and the residual popliteal (POPV), peroneal (PV), gastrocnemius (GV) and soleal veins (SV) were evaluated. The diagnosis of DVT was based on both noncompressibility of the vein on B–mode, and no spontaneous flow on color Doppler imaging. Thrombosis was considered as proximal if involving the deep veins.
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in CFV, FV and POPV with or without calf vein (CV) thrombosis. Thrombosis was considered as
distal if involving only the CV. Duplex scanning was repeated at 1 month, 3 months, 6 months, and 1
year. At each examination, venous segments were assessed to determine the distribution of
thrombosis and whether they were occluded, partially recanalized, and totally recanalized. Venous
segments were considered as occluded if they showed incompressibility and absence of flow with
distal augmentation. Partial recanalization was defined by partial compressibility of the vein and
diminished cephalad flow by distal augmentation. Complete recanalization was determined by
complete compressibility of the vein and spontaneous cephalad flow on color Doppler imaging.

The development of venous reflux was evaluated as described by van Bemmelen et al. 22

Valve closure times (VCTs) were measured in the deep venous systems, with the patient in the
upright position with his/her weight supported on the contralateral leg. For evaluation of the CFV,
FV, and great saphenous vein, a pneumatic thigh cuff (Hokanson, Bellevue, WA, USA) was attached
to the thigh, inflated to 80 mmHg and then rapidly deflated. For evaluation of the POPV and small
saphenous vein, a cuff was applied to the calf, inflated to 100 mmHg and then rapidly deflated. Calf
veins were examined with the patients in the sitting position, with the foot resting on a stool, distal
calf compression was undertaken. VCTs of > 0.5 s were considered to be incompetent.

Statistical analysis

All data were analyzed using StatView for Windows (Version 5.0, SAS Institute Inc.,
Cary, NC). Chi–squared contingency table analysis was used to evaluate differences between
proportions. Statistical significance was defined as \( p < 0.05 \).

RESULTS

At 1 year, 5 patients were lost to follow–up, 8 had inadequate follow–up study, and the
remaining 70 limbs in 67 patients involving 147 anatomic segments with DVT were presented in this
study. There were 107 above–knee, and 40 below–knee segments. The segments investigated were
the CFV (38 segments), FV (33 segments), POPV (36 segments), and isolated CV (40 segments).

Table I shows baseline characteristics of the 67 study patients. There were 33 inpatients
and 34 outpatients. Among cases, 23 patients were male and 44 were female. Patients age ranged
from 27 to 86 years (mean, 60 years). DVT was the most common after operation or trauma
(21 patients, 31%). Immobilization and active cancer were present in 8 (12%) patients. Stroke was
found in 6 (9%) patients. Previous DVT was less common in this study (3 patients, 5%). Protein C
deficiency was the most common thrombophilia (5 patients, 8%) followed by protein S deficiency (4
patients, 6%), AT III deficiency (2 patients, 3%), positive antiphospholipid syndrome (2 patients,
3%), and hyperhomocysteinemia (2 patients, 3%).

Table II shows the anatomic distribution of DVT at initial examination. There were 35
limbs with isolated venous segments, and the remaining 35 with multisegments. The most common
anatomic distribution with single venous segment was isolated CV. On the contrary, the common
distribution with multisegments extended from CFV to POPV or CFV to CV.

The proportions of occlusion, partial recanalization, and total recanalization in each
venous segment are shown in Table III. At 1 year, thrombi had fully resolved in 75% of the segments,
20% remained partially recanalized, 5% were occluded. Of these, the CV had the highest rate of
recanalization (100%), whereas FV showed the highest rate of occlusion (21%). No complete
occlusion was found in CFV, POPV, and CV.

The duration of time for complete recanalization is shown in Table IV. At 1 month, 27 of
40 segments (68%) in the CV were fully resolved. On the other hand, proximal veins had statistically
lower proportions of complete recanalization: 24% of CFV, 18% of FV, and 44% of POPV (CFV and
FV vs. CV; \( P<0.0001 \), POPV vs. CV; \( P<0.05 \)). At 1 year, total resolution was found in 68% of CFV,
49% of FV, 81% of POPV, and 100% of CV, and similar statistically significant differences were
found between proximal veins and CV (CFV vs. CV; \( P<0.0001 \), FV vs. CV; \( P<0.0001 \), and POPV vs.
CV; \( P<0.01 \)).

Table V shows the comparison of the time duration for complete thrombus resolution in
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the CV between single segment and multisegments DVT. There were 23 segments with isolated CV, and 17 with CV thrombosis with multisegments DVT. There was no statistically significant difference in the proportion of recanalization between the two groups at any follow–up point.

The venous valvular competency for each venous segment is shown in Table VI. Venous valvular reflux was detected as early as 1 month, and 28% of the segments with initial POPV thrombosis had reflux by 3 months. At 1 year, POPV had the highest frequency of venous reflux (56%), followed by FV (18%), whereas no venous incompetence was found in the CV.

Table VII shows the development of venous reflux between single segment and multisegments DVT. At 1 year follow-up, deep vein insufficiency (DVI) was found in 7 patients with isolated DVT, and 15 who had multisegment DVT. The proportion of DVI was found to be statistically significant in patients who had multisegment DVT (P<0.01). Superficial venous insufficiency (SVI) was detected in 2 patients and, 4 with multisegment DVT. There was no statistically significant difference between the two groups. Similarly, perforating vein insufficiency (PVI) was found in 2 patients with isolated and 4 with multisegment DVT, resulting in no significant difference.

DISCUSSION

Duplex scanning is a useful tool for both indentifying an acute DVT and following-up studies to detect subsequent venous abnormalities.7-13 The initial anatomic distribution of an acute DVT can vary between the patients. In previous studies, DVT has been considered to originate from the calf and thence to propagate proximally.23,24 In contrast, many of the thrombi diagnosed by venous duplex scan did not begin in the calf. Cogo et al. found that 88% of the studied patients were located in proximal veins, and remaining 12% presented isolated calf vein thrombosis.25 He also investigated the distribution of proximal vein thrombosis and found that 42% of patients had POPV and FV involvement, and 35% had entire proximal vein involvement.25 Hill et al. showed CV thrombi in 24 % of all DVT cases, with 49% of patients showing proximal vein thrombosis without calf involvement.26 Of these, 13% of the patients had DVT in CFV, 19% in FV, 18% in POPV, and 16% in entire deep venous segment involving CV. Our results show that the overall incidence DVT involving proximal vein was 67% and the residual 33% demonstrated isolated CV thrombi. Only 17% of the patients demonstrated DVT in entire deep vein segment.

The natural course of venous thrombosis is three–fold; initial loose thrombi become adherent to the vein wall. The local inflammatory response of the vein wall initiates the organization of the thrombus. And lysis of the area within the thrombus leads to recanalization.27 In the past, the different rates of thrombus resolution and recanalization have been reported after diagnosis. Killiewich et al. found that the lysis was observed as early as 1 week, and as many as 50% cases of DVT will undergo complete resolution within 6 months.28 van Ramshorst et al. reported that recanalization occurred in 87% of the segments within 6 weeks, and that recanalization progressed at a similar rate in proximal vein segments.29 Caprini and associates studied the rate of normalization within 6 months after the diagnosis of acute DVT in various venous segments, and found that the rates of resolution of DVT were 78% in CFV, 70% in FV, 75% in POPV, and 70% in CV, and concluded that the rate of resolution were similar for the different veins.30 On the contrary, O'Shaughnessy and associates described that individual anatomic venous segments in the lower extremity respond in a different way to the presence of thrombus. A 1–year follow–up study revealed that the rates of resolution were 78% in CFV, 42% in FV, and 54% in POPV.31 Our data show that the rates of resolution were 68% in CFV, 49% in FV, and 81% in POPV at 1-year, which results in the different rate of resolution between the venous segments confined to proximal DVT. In the CV thrombosis, however, Meissner found that thrombus load was reduced by 50% at 1 month and to zero at 1 year.32 McDonald investigated the natural history of isolated CV thrombosis, and found that 84% of the segments remained stable or resolved during the 3-month follow-up.33 These data show that recanalization proceeds rapidly confined to CV thrombosis.

Thrombosis damages the deep venous valves, and the destruction of venous valves
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results in venous reflux and venous hypertension in lower limbs. Previous studies have shown that the time to complete recanalization is considered to be important in the determination of the development of venous valvular reflux.\textsuperscript{28, 34, 35} van Bemmelen proposed that the development of venous incompetence is a two-stage process.\textsuperscript{36} Initially, the vein dilates in response to proximal venous obstruction. Valvular incompetence occurs because the cusps of the valves are not long enough to coapt with the increased venous diameter. Later, by approximately the sixth month, the valves are completely destroyed, and thus permanently incompetent. Killewich et al. studied 21 patients with acute DVT and found that 8% of patent segments contained incompetent valves at 30 days and that 25% percent of patent segments contained incompetent valves at 180 days.\textsuperscript{28} O’Shaughnessy and associates found that the venous segments that resolved within 6 months had a higher competence rate than that resolved from 6 months to 1 year.\textsuperscript{12} But the ideal time for resolution is not determined yet.\textsuperscript{37}

Severe PTS could be caused by either obstruction or reflux in the axial deep venous system, or combination of the two. Johnson and associates found that only 3% of limbs with PTS were normal, 18% had reflux alone, 15% had obstruction alone, and 65% had both reflux and obstruction, and concluded that a combination of residual reflux and residual partial or complete obstruction in the major deep veins is more likely to be associated with the development of the PTS.\textsuperscript{38} On the contrary, Killewich et al showed that reflux in the deep veins was the main contributor to the severity of PTS.\textsuperscript{39} Haenen and associates also demonstrated that the advanced CVI was found in FV and POPV with reflux, and reflux in the deep proximal veins significantly contributed to the development of PTS.\textsuperscript{40} Meissner found that the extent of venous reflux, the presence of persistent POPV obstruction, and the rate of recanalization were related to the ultimate CVI. These reports suggest that the residual abnormalities in the proximal deep veins play major role in the development of late PTS. In isolated CV thrombosis, few will develop significant clinical symptoms attributable to CVI,\textsuperscript{41} but reflux in the primary uninvolved POPV may be associated with more severe disease.\textsuperscript{42} Our results show that the proportion of DVI was found to be significant in patients who had multisegment DVT. Therefore, much longer follow–up may be required in patients with multisegment DVT on initial examination.

Other investigators revealed the primary importance of superficial venous reflux in the development of symptoms of PTS.\textsuperscript{43, 44} If the perforating veins become insufficient, the venous pressure in the superficial veins might increase and cause an overload of the superficial system, resulting in a gradually developing insufficiency of the superficial veins.\textsuperscript{28} Labropoulos et al. found that the incidence of ulceration increased with an increased extent of reflux in the presence of SVI and that absence of SVI was associated with low incidence of leg ulceration even in the presence of DVI.\textsuperscript{43} Haenen et al studied venous reflux and calf muscle dysfunction in relation to the severity of PTS in 70 legs and found that there was no correlation between deep reflux and severity of PTS and that SVI was an increasing risk factor for advanced CVI within 2 years. However, because 2–year follow–up is a short time, much longer investigation will be required on this matter.\textsuperscript{44}

Patients with acute DVT should be treated with anticoagulation, and treatment should be longer in patients with proximal DVT than in those with distal DVT.\textsuperscript{45} Previous studies revealed that oral anticoagulant is indicated for \( \geq 3 \) months in patients with proximal DVT,\textsuperscript{46} \( \geq 6 \) months in those with proximal DVT in whom reversible cause cannot be identified, and for 6–12 weeks in those with symptomatic CV DVT.\textsuperscript{47} In the current study, different rate of recanalization was found in each venous segment. Our data show that the rates of resolution were 98% in CV at 3 months, 81% in POPV at 6 months, 68% in CFV at 12 months, and 49% in SFV at 12 months. And this study provides recommended protocol for oral anticoagulant therapy: The oral anticoagulant therapy should be indicated for \( \leq 3 \) months in patients with isolated CV DVT, 3–6 months in those with isolated POPV DVT, and \( \geq 6 \) months in those with isolated CFV, SFV, and multisegment DVT. Patients with thrombophilia may require life–long oral anticoagulant therapy.

In conclusion, the lower extremity venous segments show different proportions of occlusion, partial recanalization, and total recanalization. The CV shows more rapid recanalization than proximal veins regardless of the involvement in proximal veins. Venous reflux was noted as early as 1 month. The limbs involving multisegment DVTs on initial examination had higher incidence of DVI and may require much longer follow–up studies.
References

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44. Haenen JH, Janssen MCH, Wollersheim H, van’t Hof MA, de Rooij MJM, van Langen H,
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Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
<thead>
<tr>
<th>Parameters</th>
<th>n = 67 patients</th>
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</thead>
<tbody>
<tr>
<td><strong>Characteristics</strong></td>
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<tr>
<td>Mean age (y)</td>
<td>59.6 ± 16.1</td>
</tr>
<tr>
<td>Gender (% male)</td>
<td>23 (34.3)</td>
</tr>
<tr>
<td>Outpatients (%)</td>
<td>34 (50.7)</td>
</tr>
<tr>
<td><strong>Risk factors (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Immobilization</td>
<td>8 (11.9)</td>
</tr>
<tr>
<td>Known malignancy</td>
<td>8 (11.9)</td>
</tr>
<tr>
<td>Stroke</td>
<td>6 (9.0)</td>
</tr>
<tr>
<td>Operation or trauma</td>
<td>21 (31.3)</td>
</tr>
<tr>
<td>Previous history of DVT</td>
<td>3 (4.5)</td>
</tr>
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<td>Inflammatory bowel disease</td>
<td>2 (3.0)</td>
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<tr>
<td>Hormon replacement therapy</td>
<td>3 (4.5)</td>
</tr>
<tr>
<td><strong>Thrombophilia (%)</strong></td>
<td></td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>4 (6.0)</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>5 (7.5)</td>
</tr>
<tr>
<td>AT III deficiency</td>
<td>2 (3.0)</td>
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<tr>
<td>Positive antiphospholipid syndrome</td>
<td>2 (3.0)</td>
</tr>
<tr>
<td>Hyperhomocysteinemia</td>
<td>2 (3.0)</td>
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<tr>
<td>Abnormal plasminogen</td>
<td>1 (1.5)</td>
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Table.I
Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
<thead>
<tr>
<th>Isolated segment</th>
<th>No. of segments (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CFV</td>
<td>4 (5.7)</td>
</tr>
<tr>
<td>SFV</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td>POPV</td>
<td>7 (10)</td>
</tr>
<tr>
<td>CV</td>
<td>23 (32.9)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>35 (50)</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Multisegment</th>
<th>No. of segments (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CFV+CV</td>
<td>2 (2.9)</td>
</tr>
<tr>
<td>CFV–SFV+CV</td>
<td>2 (2.9)</td>
</tr>
<tr>
<td>CFV–CV</td>
<td>12 (17.1)</td>
</tr>
<tr>
<td>CFV–POPV</td>
<td>16 (22.9)</td>
</tr>
<tr>
<td>CFV–SFV</td>
<td>2 (2.9)</td>
</tr>
<tr>
<td>POPV–CV</td>
<td>1 (1.4)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>35 (50)</strong></td>
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</table>

Table.II
Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
<thead>
<tr>
<th>Site</th>
<th>No. of segments</th>
<th>Resolved</th>
<th>Partial</th>
<th>Occluded</th>
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</thead>
<tbody>
<tr>
<td>CFV (%)</td>
<td>38</td>
<td>26 (68.4)</td>
<td>12 (31.6)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>FV (%)</td>
<td>33</td>
<td>16 (48.5)</td>
<td>10 (30.3)</td>
<td>7 (21.2)</td>
</tr>
<tr>
<td>POPV (%)</td>
<td>36</td>
<td>29 (80.6)</td>
<td>7 (19.4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>CV (%)</td>
<td>40</td>
<td>40 (100)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Total</td>
<td>147</td>
<td>111 (75.5)</td>
<td>29 (19.7)</td>
<td>7 (4.8)</td>
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Table III
Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
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<tr>
<th>Site</th>
<th>No. of segments</th>
<th>1M</th>
<th>3M</th>
<th>6M</th>
<th>12M</th>
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<tr>
<td>CFV (%)</td>
<td>38</td>
<td>9 (23.7)*</td>
<td>17 (44.7)*</td>
<td>23 (60.5)*</td>
<td>26 (68.4)†</td>
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<tr>
<td>FV (%)</td>
<td>33</td>
<td>6 (18.2)*</td>
<td>9 (27.3)*</td>
<td>12 (36.4)*</td>
<td>16 (48.5)*</td>
</tr>
<tr>
<td>POPV (%)</td>
<td>36</td>
<td>16 (44.4)§</td>
<td>24 (66.7)†</td>
<td>29 (80.6)‡</td>
<td>29 (80.6)‡</td>
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<tr>
<td>CV (%)</td>
<td>40</td>
<td>27 (67.5)</td>
<td>39 (97.5)</td>
<td>40 (100)</td>
<td>40 (100)</td>
</tr>
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</table>

* Proximal veins vs. calf veins: p<0.0001
† Proximal veins vs. calf veins: p<0.001
‡ Proximal veins vs. calf veins: p<0.01
§ Proximal veins vs. calf veins: p<0.05

Table IV
Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
<thead>
<tr>
<th>Site</th>
<th>No. of segments</th>
<th>1M</th>
<th>3M</th>
<th>6M</th>
<th>12M</th>
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<tbody>
<tr>
<td>Isolated CV (%)</td>
<td>23</td>
<td>14 (60.9)</td>
<td>22 (95.7)</td>
<td>23 (100)</td>
<td>23 (100)</td>
</tr>
<tr>
<td>Multisegment + CV (%)</td>
<td>17</td>
<td>13 (76.5)</td>
<td>17 (100)</td>
<td>17 (100)</td>
<td>17 (100)</td>
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Table.V
Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
<thead>
<tr>
<th>Site</th>
<th>No. of segments</th>
<th>1M</th>
<th>3M</th>
<th>6M</th>
<th>12M</th>
</tr>
</thead>
<tbody>
<tr>
<td>CFV (%)</td>
<td>38</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>3 (7.9) *</td>
<td>4 (10.5) *</td>
</tr>
<tr>
<td>FV (%)</td>
<td>33</td>
<td>0 (0)</td>
<td>1 (3) ‡</td>
<td>4 (12.1) †</td>
<td>6 (18.2) *</td>
</tr>
<tr>
<td>POPV (%)</td>
<td>36</td>
<td>2 (5.6)</td>
<td>10 (27.8)</td>
<td>19 (52.8)</td>
<td>20 (55.6)</td>
</tr>
<tr>
<td>CV (%)</td>
<td>40</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
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</tbody>
</table>

*CFV or SFV vs. POPV: p<0.0001
†CFV or SFV vs. POPV: p<0.001
‡CFV or SFV vs. POPV: p<0.01
§CFV or SFV vs. POPV: p<0.05

Table. VI
Patterns of venous insufficiency after an acute deep vein thrombosis

<table>
<thead>
<tr>
<th></th>
<th>No. of patients</th>
<th>DVI</th>
<th>SVI</th>
<th>PVI</th>
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<tbody>
<tr>
<td>Single segment</td>
<td>35</td>
<td>7*</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Multisegment</td>
<td>35</td>
<td>15</td>
<td>4</td>
<td>4</td>
</tr>
</tbody>
</table>

*Single segment vs. multisegment: p<0.01

Table. VII
Patterns of venous insufficiency after an acute deep vein thrombosis

Legends

Table I.
Baseline clinical characteristic of the 67 study patients

Table II.
The anatomic distribution of thrombi at initial examination

Table III.
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Table IV.
The duration of time for complete recanalization

Table V.
The comparison of the time duration for complete thrombus resolution in the CV between single segment and multisegments DVT

Table VI.
The venous valvular competency for each venous segment

Table VII.
The development of venous reflux between single segment and multisegments DVT