Iliac Vein Compression Syndrome in an Asymptomatic Patient Population: A Prospective Study

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Background: Iliac vein compression syndrome (IVCS) is an important cause of deep vein thrombosis, but the incidence of IVCS is still unclear. The purpose of this prospective study was to determine the incidence of IVCS in an asymptomatic patient population and to evaluate the risk factors in patients with and without IVCS.

Methods: From October 2011 to November 2012, a total of 500 patients (228 women and 272 men; mean age of 55.4 ± 14.7 years) with no vascular-related symptoms were enrolled in this study. Computed tomography was performed to evaluate all patients. The degree of venous compression was calculated as the diameter of the common iliac vein at the site of maximal compression divided by the mean diameter of the uncompressed proximal and caudal left common iliac vein (LCIV). We compared the stenosis rate of the common iliac vein in women and men according to age and followed up patients to evaluate outcomes.

Results: The mean compression degree of the LCIV was 16% (4%, 36%); 37.8% of patients had a compression degree ≥25% and 9.8% had a compression degree ≥50%. There was a significant difference between men and women in the LCIV compression degree (9% [3%, 30%] vs. 24% [8%, 42%]; U = 4.66, P < 0.01). In addition, the LCIV compression degree among younger women (≤40 years) was significantly different compared with that in older women (>40 years) (42% [31%, 50%] vs. 19% [5%, 39%]; U = 5.14, P < 0.001). Follow-up was completed in 367 patients with a mean follow-up of 39.5 months (range, 6–56 months). The incidence of IVCS in the follow-up period was 1.6%. Stenosis rate and the diameter of the site of maximal compression correlated with the incidence of IVCS. Multivariable Cox regression analysis showed that the stenosis rate was an independent risk factor of IVCS (Wald $\chi^2 = 8.84$, hazard ratio = 1.13, P < 0.001).

Conclusions: The incidence of IVCS was low and correlated with the stenosis rate of iliac vein. Preventative therapy may be warranted for common iliac vein compression in patients at an increased risk of venous thromboembolism, especially patients with a higher iliac vein compression degree.

Key words: Deep Vein Thrombosis; Iliac Vein Compression Syndrome; May–Thurner Syndrome

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57 patients suffering from acute iliofemoral DVT caused by IVCS. They termed the disease Cockett syndrome. Meanwhile, Cockett and Thomas also pointed out that this disorder was responsible for a significant percentage of left iliofemoral DVT cases, causing venous reconstruction failure and leading to long-term occlusion of the iliac vein.

The incidence of IVCS is still controversial and its definition is also unclear. The majority of existing studies focus on the compression degree of the iliac vein, but few studies have evaluated the incidence of IVCS in patients with no vascular-related symptoms. A patient with no vascular-related symptoms is defined as one who had no venous diseases such as DVT and varicose veins, or no venous symptoms such as swelling, edema, hyperpigmentation, and ulcer. Asymptomatic patients should be evaluated to exclude the influence of primary vascular disease and vascular-related symptoms on the incidence of IVCS. This study aimed to determine the incidence of and risk factors for IVCS in asymptomatic patients.

**Methods**

**Ethical approval**

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Beijing Shijitan Hospital (Beijing Shijitan Hospital Research Ethics Approval No. 5, 2017). Informed written consent was obtained from all patients prior to their enrollment in this study.

**Patients**

A total of 628 patients who underwent helical abdominal-enhanced computed tomography (CT) but with no vascular-related symptoms at Beijing Shijitan Hospital (Beijing, China) from October 2011 to November 2012 were included in this study.

Inclusion criteria were as follows: (1) age ≥18 years; (2) no vascular-related symptoms (swelling, edema, hyperpigmentation, and venous ulcer) or diagnostically confirmed vascular diseases (varicose veins, DVT, thrombophlebitis); (3) performance of helical abdominal-enhanced CT.

Exclusion criteria were: (1) scanning parameters >5 mm, or unclear imaging; (2) expansion from the inferior vena cava to the LICV (we identified patients with vein dilation from the inferior vena cava to the LCIV on CT evaluation; we thought that this might affect the calculation of the stenosis rate); (3) congenital anomalies of the inferior vena cava, RIA, or LCIV; (4) history of trauma, abdominal surgery, or vascular bypass surgery; (5) LCIV compressed by tumor, foreign matter, or implant; (6) stent implantation in the inferior vena cava, RIA, or LCIV.

Finally, 500 patients were enrolled in our study. We divided patients into groups according to gender, and each group was subdivided into younger (≤40 years) and older (>40 years) subgroups.

**Computed tomography evaluation**

CT (Siemens, Munich, Germany) was used in all cases [Figure 1]. The spatial resolution of this CT is 0.75 mm. Scanning parameters included 2-mm to 5-mm axial images. We divided the LCIV into proximal, middle, and distal segments and recorded the diameter proximal and distal to the crossing site of the RIA over the LCIV and at the site of crossing. Measurements of the minor diameter of the LCIV were obtained from the segment of vessel that was foremost in the plane of the image. The degree of venous compression was calculated as the diameter of the common iliac vein at the site of maximal compression divided by the mean diameter of the uncompressed proximal and caudal LCIV. In general, obvious hemodynamic changes can occur when the degree of artery stenosis is >70%. Low velocity of flow and pressure in the venous system would cause evident hemodynamic changes without a high degree of venous stenosis. We used a compression degree ≥50% as the diagnostic criterion.

**Follow-up**

Follow-up was in the form of medical records' review and telephone interview. We evaluated the patients’ medical records after measuring the LCIV compression degree. If the patient was not an inpatient during the follow-up period, we used telephone interview to obtain information. The interval time of follow-up was 3 months, and patients who developed target events, defined below, were invited to our hospital for ultrasound examination.

The main components of follow-up were as follows:

1. Whether the following target events occurred during the follow-up period: lower limb swelling, varicose veins, hyperpigmentation, ulcers, DVT, pulmonary embolism (PE), and/or hemodynamic changes. Hemodynamic
changes refer to alterations in blood flow velocity in the iliac vein. In our hospital, the normal blood flow velocity in the iliac vein is defined as 30–50 cm/s. Blood flow velocity is faster proximal to the compression site and slower distal to the compression site; the ratio between these two values is >2.5. Hemodynamic changes and diagnosis of target events were based on ultrasound examination:

2. Coagulation function, including prothrombin time (PT), activated partial thromboplastin time (APTT), thrombin time (TT), fibrinogen, platelets, and D-dimer. Any abnormality in these parameters was considered coagulation dysfunction;

3. Risk factors, including coronary heart disease (CHD), hypertension, diabetes, cerebrovascular disease, hyperlipidemia, malignancy, coagulation function, anti-coagulation therapy, antiplatelet therapy, lipid-lowering therapy, and surgery.

Diagnostic criteria of iliac vein compression syndrome
A compression degree ≥50% and the occurrence of target events were used to diagnose IVCS.

Statistical analysis
All statistical analyses were performed using SPSS version 20.0 statistical package (IBM, Armonk, NY, USA). Continuous variables are reported as mean ± standard deviation (SD) or median (quartile) and categorical variables as frequency or percentage of events. Student’s t-test was used for normally distributed continuous data, and Mann-Whitney U-test was used for abnormally distributed continuous data. Pearson’s Chi-square test or Fisher’s exact test was used to compare differences in proportion between groups. Cox univariate and multivariate regression analyses were performed to identify independent risk factors predicting the incidence of IVCS. All statistical tests were two sided, with a statistical significance level set at P < 0.05.

RESULTS
Overall comparison of the iliac vein compression degree
This study evaluated 228 women and 272 men (mean age: 55.4 ± 14.7 years; range, 18–89 years). The mean compression degree of the LCIV in the entire population was 16% (4%, 36%) [Figure 2]. The mean compression degree of the LCIV was 24% (8%, 42%) in women and 9% (3%, 30%) in men. There was a statistically significant difference in mean compression of the LCIV in terms of gender [24% (8%, 42%) vs. 9% (3%, 30%), U = 4.66, P < 0.01, Table 1].

CT measurements showed that 37.8% of patients (n = 189) had ≥25% compression of the LCIV and 9.8% (n = 49) had ≥50% compression. There were statistically significant differences in these parameters between women and men [≥25% compression, χ² = 17.85, P < 0.01; ≥50% compression, χ² = 5.35, P = 0.021; Table 1].

We divided the women and men groups into younger (≤40 years) and older (>40 years) subgroups. There were statistically significant differences between women and men in both the younger and older groups [younger groups, U = 4.31, P < 0.001; older groups, U = 2.97, P < 0.001; Table 2]. This difference can also be seen in the scatter diagram, in which a compression degree ≥25% is more prominent among young women than men [Figure 3]. We found a statistically significant difference when comparing age among women [U = 5.14, P < 0.01, Table 3], while no strong correlation existed among men [U = 0.34, P = 0.736; Table 3].

Occurrence of target vascular events during the follow-up period
A total of 367 patients completed follow-up; the follow-up completion rate was 73.4% and the lost to follow-up rate was 26.6%. The average duration of the follow-up was 39.5 months (range, 6–56 months). Reasons for follow-up loss included patient death, incomplete medical records, and change of telephone number. Target events occurred in 17 patients (4.6%) during follow-up [Table 4].

Comparison of coagulation functions
Overall, 119 patients (32.4%) showed coagulation dysfunction among 367 patients completing follow-up. In patients with a tumor diagnosis, coagulation dysfunction was present in 60.4% (86 patients). When comparing patients with versus without a tumor diagnosis, the rates of abnormal PT, APTT, TT, and D-dimer were significantly different [Table 5]. We did not find a statistically significant correlation between tumor diagnosis or coagulation dysfunction and the incidence of IVCS [Table 6].

Incidence of iliac vein compression syndrome and risk factors
Among the 17 patients diagnosed with target events during follow-up, 6 patients showed a LCIV compression degree ≥50% [Table 4]. Thus, in our study, the incidence of IVCS was 1.6%. There was no statistical difference in patients with versus without IVCS in terms of age, gender, or other risk factors; there was a significant difference in the minimum diameter of LCIV and stenosis rate [minimum diameter of LCIV, t = 7.98, P < 0.001; stenosis rate, t = −4.43, P < 0.001; Table 6].

Figure 2: Individual results of compression percentage of the left common iliac vein as measured on axial computed tomography images with transverse linear measurements.
On ultrasound examination of patients with IVCS, the LCIV compression degree was >50% in all patients. The blood flow velocity on the pressure point was increased and the pressure distal to the pressure point was reduced; the ratio between the two sites was >2.5 [Figure 4].

**Discussion**

The precise incidence of IVCS is controversial. The purpose of this study was to confirm how often IVC occurs and to determine the precise incidence of IVCS in asymptomatic...
The results of early autopsy and modern studies showed the incidence of IVC to be 20% to 49%. In this study, 37.8% of patients had a LCIV compression degree ≥25%, while 9.8% had a compression degree ≥50%; our results also demonstrated that LCIV compression was very common.

| Table 4: Information of patients in whom target events occurred |
|-----------------|-----------------|-----------------|-----------------|
| n   | Gender | Age (years) | Target events | Compression degree (%) | Underlying disease | Occurrence time (months) |
|-----|--------|-------------|---------------|------------------------|-------------------|-------------------------|
| 1   | Female | 48          | Slow flow velocity in bilateral popliteal vein | 2.01                 | Colon cancer (Stage IV) | 6                       |
| 2   | Male   | 74          | Bilateral superficial femoral vein thrombosis | 3.15                 | Pancreatic cancer (Stage IV) | 12                      |
| 3   | Female | 77          | Bilateral great saphenous varicose veins | 3.68                 | Pneumonia | 27                     |
| 4   | Male   | 35          | Left-side DVT | 6.76                 | Gallstone | 35                     |
| 5   | Male   | 64          | Left-side DVT | 8.18                 | Insufficient cerebral circulation | 40                     |
| 6   | Female | 51          | Left great saphenous varicose veins | 8.52                 | Cervical cancer | 8                       |
| 7   | Female | 52          | Bilateral DVT | 9.15                 | Lung cancer (Stage IV) | 22                     |
| 8   | Female | 81          | Left great saphenous varicose veins | 11.59                | Gastric cancer (Stage II) | 41                     |
| 9   | Female | 80          | Left great saphenous varicose veins | 19.07                | Chronic gastritis | 13                     |
| 10  | Female | 73          | Right-side DVT | 19.12                | Cirrhosis | 30                     |
| 11  | Female | 49          | Left great saphenous varicose veins | 38.67                | Leiomyoma | 37                     |
| 12  | Male   | 68          | Bilateral DVT | 51.96                | Rectal cancer (Stage IIIb) | 6                       |
| 13  | Female | 61          | Left-side DVT | 57.96                | CHD | 14                     |
| 14  | Male   | 61          | Bilateral DVT | 62.24                | Rectal cancer (Stage IIIb) | 9                       |
| 15  | Female | 76          | Left-side DVT | 62.50                | Adrenal adenoma | 50                     |
| 16  | Male   | 46          | Bilateral great saphenous varicose veins | 63.72                | Hypertension | 28                     |
| 17  | Female | 66          | Left great saphenous varicose veins | 71.88                | Abnormal glucose metabolism | 18                     |

DVT: Deep vein thrombosis; CHD: Coronary heart disease.

| Table 5: Comparison of coagulation function between patients with and without tumor |
|-----------------|-----------------|-----------------|-----------------|
| Items          | Tumor group (n = 224) | Nontumor group (n = 143) | Statistics | P    |
|----------------|------------------------|--------------------------|------------|------|
| PT (s)         | 12.09 ± 3.58           | 10.61 ± 1.13             | 6.17*      | <0.01|
| APTT (s)       | 32.66 ± 6.54           | 30.63 ± 3.46             | 4.15*      | <0.01|
| TT (s)         | 15.01 ± 2.08           | 14.32 ± 1.01             | 4.56*      | <0.01|
| Fib (g/L)      | 3.15 ± 1.14            | 3.09 ± 0.57              | 0.68*      | 0.497|
| PLT (∼10^9/L)  | 191.63 ± 101.51        | 203.03 ± 47.84           | −1.55*     | 0.122|
| D-dimer (positive rate) | 19.5%                      | 2%                         | 24.85†      | <0.01|

* t value; † χ² value. PT: Prothrombin time; APTT: Activated partial thromboplastin time; TT: Thrombin time; Fib: Fibrinogen; PLT: Platelets.

Figure 4: (a) Ultrasound of a patient showing the left common iliac vein being compressed by the right iliac artery and the fifth lumbar vertebra. (b) Ultrasound showing the stenosis rate of the left common iliac vein to be >70%.

IVC occurs most commonly in young women, with 85% of cases occurring in women between 20 and 40 years old.[5] In modern theory, physiological curvature of the vertebral column in the lumbosacral portion displays more extrusion in women than in men, and this may be responsible for the increased incidence of IVCS in women. In our study, the LCIV compression degree of young women group was significantly different from that of other groups.

IVCS is a main cause of venous thromboembolic disease, and this anatomical variation was very common. Approximately 20% of the adult population has anatomical variations, but most are asymptomatic[11] symptoms include edema, swelling, pain, varicose veins, venous ulcer, DVT, and PE. IVC may be a contributing factor in 18–49% of patients with left-side DVT.[6,12] It may occur in 2–5% of patients who present with lower extremity venous diseases. Some studies
showed that IVC exists in approximately 22–24% of these patients.\textsuperscript{[13‑15]} In our prospective study with 39.5 months’ follow-up, the incidence of IVCS was 1.6% in asymptomatic patients, lower than previous findings. This difference may be due to the fact that previous studies were almost always retrospective research without follow-up. The patients included in previous studies were those with venous diseases, so the incidence may increase. In this study, we excluded patients with venous disease, and all included patients were asymptomatic; we then followed all patients, which may reflect a more accurate incidence of IVCS.

It was found that the number of patients developing target events increased with longer duration of follow-up, and the correlation between compression degree and the diameter of the site of maximal compression with the incidence of IVCS was statistically significant. In multivariable Cox regression analysis, we found the stenosis rate to be an independent risk factor for IVCS occurrence. These are novel findings compared to previously published articles. The long-term follow-up may have contributed to this.

Table 6: Analysis of hypothetical risk factors for the incidence of IVCS

| Factors                                | Sum (n = 367) | No IVCS (n = 361) | IVCS (n = 6) | Statistics | P    |
|----------------------------------------|--------------|-------------------|--------------|------------|------|
| Gender, n (%)                          |              |                   |              |            |      |
| Male                                   | 193 (52.6)   | 189 (52.4)        | 4 (66.7)     | 0.26*      | 0.688|
| Female                                 | 174 (47.4)   | 172 (47.7)        | 2 (33.3)     |            |      |
| Age (years), mean ± SD                 | 56.5 ± 14.7  | 56.5 ± 14.6       | 54.8 ± 16.5  | 0.28\textsuperscript{†} | 0.782|
| Mean diameter (cm), mean ± SD          | 0.91 ± 0.20  | 0.91 ± 0.20       | 1.00 ± 0.14  | −1.02\textsuperscript{†} | 0.309|
| Minimum diameter (cm), mean ± SD       | 0.72 ± 0.28  | 0.72 ± 0.28       | 0.41 ± 0.09  | 7.98\textsuperscript{†} | 0.000|
| Stenosis rate (%), mean ± SD           | 0.22 ± 0.20  | 0.22 ± 0.20       | 0.58 ± 0.11  | −4.43\textsuperscript{†} | 0.000|
| CHD, n (%)                             |              |                   |              |            |      |
| No                                     | 292 (79.6)   | 289 (80.1)        | 3 (50.0)     | 0.09*      | 0.103|
| Yes                                    | 75 (20.4)    | 72 (19.9)         | 3 (50.0)     |            |      |
| Hypertension, n (%)                    |              |                   |              |            |      |
| No                                     | 244 (66.5)   | 241 (66.8)        | 3 (50.0)     | 0.22*      | 0.407|
| Yes                                    | 123 (33.5)   | 120 (33.2)        | 3 (50.0)     |            |      |
| Diabetes, n (%)                        |              |                   |              |            |      |
| No                                     | 294 (80.1)   | 290 (80.3)        | 4 (66.7)     | 0.25*      | 0.342|
| Yes                                    | 73 (19.9)    | 71 (19.7)         | 2 (33.3)     |            |      |
| Cerebrovascular disease, n (%)         |              |                   |              |            |      |
| No                                     | 300 (81.7)   | 296 (82.0)        | 4 (66.7)     | 0.22*      | 0.302|
| Yes                                    | 67 (18.3)    | 65 (18.0)         | 2 (33.3)     |            |      |
| Hyperlipidemia, n (%)                  |              |                   |              |            |      |
| No                                     | 301 (82.0)   | 297 (82.3)        | 4 (66.7)     | 0.22*      | 0.295|
| Yes                                    | 66 (18.0)    | 64 (17.7)         | 2 (33.3)     |            |      |
| Anticoagulation, n (%)                 |              |                   |              |            |      |
| No                                     | 327 (89.1)   | 323 (89.5)        | 4 (66.7)     | 0.11*      | 0.131|
| Yes                                    | 40 (10.9)    | 38 (10.5)         | 2 (33.3)     |            |      |
| Antiplatelet, n (%)                    |              |                   |              |            |      |
| No                                     | 306 (83.4)   | 301 (83.4)        | 5 (83.3)     | 0.34*      | 1.000|
| Yes                                    | 51 (13.9)    | 50 (13.9)         | 1 (16.7)     |            |      |
| Dual                                   | 10 (2.7)     | 10 (2.8)          | 0            |            |      |
| Lipid lowering, n (%)                  |              |                   |              |            |      |
| No                                     | 333 (90.7)   | 328 (90.9)        | 5 (83.3)     | 0.35*      | 0.444|
| Yes                                    | 34 (9.3)     | 33 (9.1)          | 1 (16.7)     |            |      |
| Operation, n (%)                       |              |                   |              |            |      |
| No                                     | 136 (37.1)   | 132 (36.6)        | 4 (66.7)     | 0.06*      | 0.393|
| Once                                   | 121 (33.0)   | 120 (33.2)        | 1 (16.7)     |            |      |
| Multiple                                | 110 (30.0)   | 109 (30.2)        | 1 (16.7)     |            |      |
| Malignancy, n (%)                      |              |                   |              |            |      |
| No                                     | 224 (61.0)   | 220 (60.9)        | 4 (66.7)     | 0.23*      | 0.386|
| Yes                                    | 143 (39.9)   | 141 (39.1)        | 2 (33.3)     |            |      |
| Coagulation dysfunction, n (%)         |              |                   |              |            |      |
| No                                     | 248 (67.6)   | 245 (67.9)        | 3 (50.0)     | 0.22*      | 0.328|
| Yes                                    | 119 (32.4)   | 116 (32.1)        | 3 (50.0)     |            |      |

*Fisher value; †t value. IVCS: Iliac vein compression syndrome; SD: Standard deviation; CHD: Coronary heart disease.
risk factors. It was difficult to determine if the presence of a coagulation disorder or the compression degree was more important for the formation of DVT. The coagulation abnormalities found in the 17 patients were variable, and the number of patients with each abnormality was small, preventing us from identifying statistically significant differences between them.

It is important to note that malignancy is an independent risk factor for DVT. In the evaluation of coagulation parameters, we found coagulation abnormalities to be more common in patients with a tumor diagnosis versus those without a tumor diagnosis. We used both univariate and multivariate regression analyses to investigate the correlation between risk factors (CHD, hypertension, malignancy, etc.) and IVCS. In univariate analysis, we compared a single factor with IVCS, and in multivariate analysis, we evaluated all confounding factors to analyze the relationship between them. We found that malignancy was not an independent risk factor of the incidence of IVCS. We hypothesized that better recognition and treatment for the prevention of thrombosis in patients with a tumor diagnosis might explain why malignancy was not identified as a risk factor for IVCS in our study.

In the recent years, with the rapid development of endovascular technology, percutaneous transluminal balloon angioplasty and stent implantation have become primary treatments for IVCS. Compared with anticoagulation, catheter-directed thrombolysis is more effective for symptom improvement and clot removal. The patency of the iliac vein after treatment ranges from 78.3% to 100%, as shown in Table 1. In the recent years, we have accumulated experience in the treatment of IVCS. The combination of catheter-directed thrombolysis with balloon angioplasty and stent implantation has demonstrated favorable curative effect, and the patency rate and prognosis are similar to previous research.

The limitation of our study is the simplistic method of follow-up; this may be the cause of the high rate of follow-up loss. We expect to continue our study and extend the follow-up time to 5 years or greater and to improve the methodology as this study continues. In addition, we have found the stenosis rate to be an independent risk factor associated with the incidence of IVCS, but the necessary degree of preventative treatment is still unclear. This is an important topic of our further research.

In conclusion, iliac vein compression is common in asymptomatic population, but the incidence of IVCS is low. Stenosis rate is an independent risk factor for IVCS. More active prevention of DVT should be considered in patients with high-grade compression of the iliac vein and risk factors for DVT.

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Conflicts of interest
There are no conflicts of interest.

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