

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

Long Cheng, Hui Zhao, Fu-Xian Zhang

Department of Vascular Surgery, Beijing Shijitan Hospital, Beijing 100038, China

## Abstract

**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  $55.4 \pm 14.7$  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  $\geq 25\%$  and 9.8% had a compression degree  $\geq 50\%$ . 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 ( $\leq 40$  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

## INTRODUCTION

Iliac vein compression syndrome (IVCS), also known as May–Thurner syndrome or Cockett syndrome, is characterized by left common iliac vein (LCIV) compression by the right iliac artery (RIA) and the fifth lumbar vertebra. This chronic venous compression may cause local intimal injury, inflammation, and scarring, ultimately leading to a spectrum of venous occlusive lesions. Due to the establishment of collateral circulation over time, patients with iliac vein compression (IVC) may have no clinical symptoms; however, IVC is associated with an increased incidence of left lower limb deep vein thrombosis (DVT).

In 1851, Virchow<sup>[1]</sup> first noted that iliofemoral vein thrombosis was 5 times more likely to occur in the left leg than the right.

In 1957, May and Thurner<sup>[2]</sup> brought more attention to this anatomic variation. They described the development of “intraluminal spurs” in the LCIV. They postulated that this was due to the combination of chronic arterial pulsations by the RIA and mechanical compression leading to the development of intimal hypertrophy of the LCIV. In 1965, Cockett and Thomas<sup>[3]</sup> associated clinical symptoms with the pathological characteristics of this disorder and treated

**Address for correspondence:** Dr. Fu-Xian Zhang,

Department of Vascular Surgery, Beijing Shijitan Hospital, Capital Medical University, Beijing 100038, China  
E-Mail: fuxian@263.net

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

**For reprints contact:** reprints@medknow.com

© 2017 Chinese Medical Journal | Produced by Wolters Kluwer - Medknow

**Received:** 27-01-2017 **Edited by:** Yuan-Yuan Ji

**How to cite this article:** Cheng L, Zhao H, Zhang FX. Iliac Vein Compression Syndrome in an Asymptomatic Patient Population: A Prospective Study. Chin Med J 2017;130:1269-75.

### Access this article online

#### Quick Response Code:



**Website:**  
www.cmj.org

**DOI:**  
10.4103/0366-6999.206341

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  $\geq 18$  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 ( $\leq 40$  years) and older ( $>40$  years) subgroups.

## Computed tomography evaluation

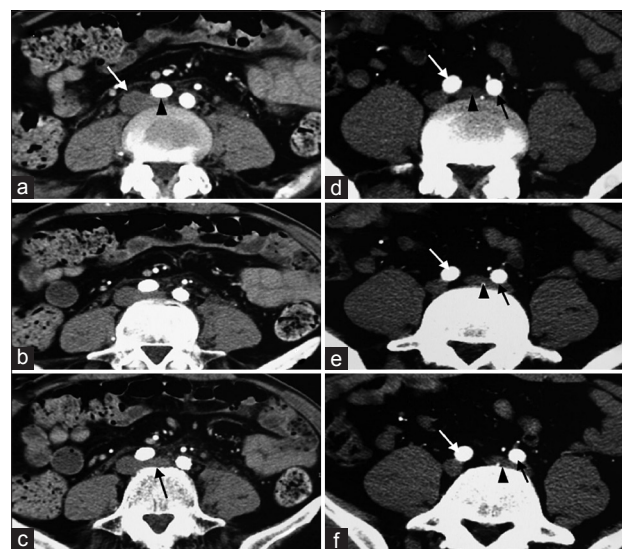
CT (Siemens, Munich, Germany) was used in all cases [Figure 1]. The spatial resolution of this CT is 0.75 mm. Scanning parameters included 2-mm to 5-mm axial images. We divided the LCIV into proximal, middle, and distal segments and recorded the diameter proximal and distal to the crossing site of the 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  $\geq 50\%$  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



**Figure 1:** Computed tomography scans of compression of the left iliac vein by the right iliac artery in one patient (a–c). White arrow = inferior vena cava; black arrowhead = right iliac artery; black arrow = left iliac vein. Computed tomography scans of compression of the left iliac vein by the left iliac artery in one patient (d–f). White arrow = right iliac artery; black arrowhead = left iliac vein; black arrow = left iliac artery.

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  $\geq 50\%$  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  $\pm$  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:  $55.4 \pm 14.7$  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  $\geq 25\%$  compression of the LCIV and 9.8% ( $n = 49$ ) had  $\geq 50\%$  compression. There were statistically significant differences in these parameters between women and men [ $\geq 25\%$  compression,  $\chi^2 = 17.85$ ,  $P < 0.01$ ;  $\geq 50\%$  compression,  $\chi^2 = 5.35$ ,  $P = 0.021$ ; Table 1].

We divided the women and men groups into younger ( $\leq 40$  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  $\geq 25\%$  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

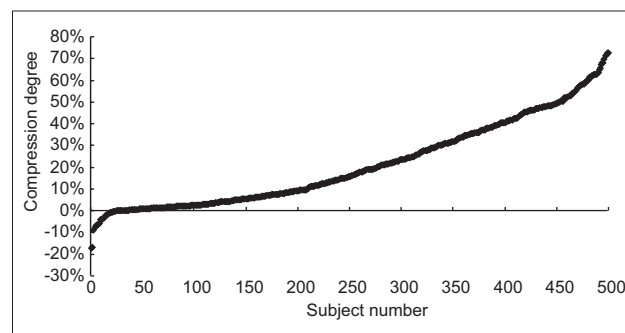
A total of 367 patients completed follow-up; the follow-up completion rate was 73.4% and the lost to follow-up rate was 26.6%. The average duration of the follow-up was 39.5 months (range, 6–56 months). Reasons for follow-up loss included patient death, incomplete medical records, and change of telephone 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.

**Table 1: Comparison of compression degree of LCIV between females and males**

Items	Female (n = 228, 45.6%)	Male, (n = 272, 54.4%)	Statistics	P
Age (years)	55.1 ± 14.0	55.6 ± 15.3	0.40*	0.687
Compression degree				
Median (%)	24 (8, 42)	9 (3, 30)	4.66 <sup>†</sup>	<0.01
≥25%	109 (47.8)	80 (29.4)	17.85 <sup>‡</sup>	<0.01
≥50%	30 (13.2)	19 (7.0)	5.35 <sup>‡</sup>	0.021

Values are shown as mean ± SD, median (quartile), or n (%). \**t* value; <sup>†</sup>*U* value; <sup>‡</sup> $\chi^2$  value. LCIV: Left common iliac vein; SD: Standard deviation.

**Table 2: Comparison of compression degree of LCIV between females and males in both younger and older subgroups**

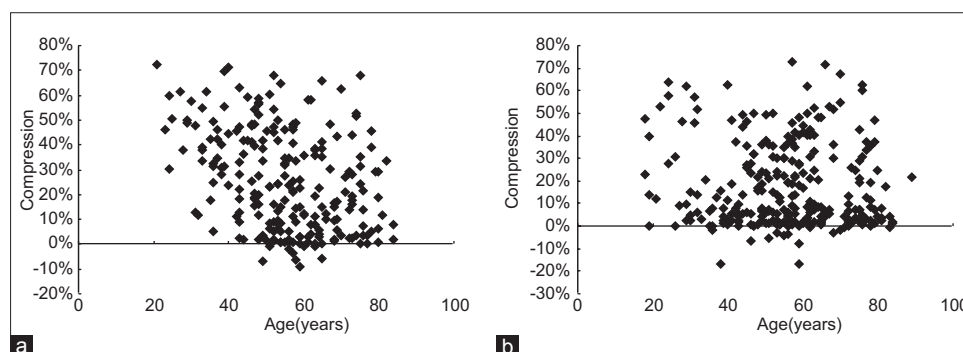
Compression degree	Younger group (≤40 years)		Statistics	P	Older group (>40 years)		Statistics	P
	Female (n = 37)	Male (n = 43)			Female (n = 191)	Male (n = 229)		
≥25%	32	13			77	67		
≥50%	11	7			19	12		
Median (%)	42 (31, 50)	11 (3, 40)	4.31*	<0.001	19 (5, 39)	9 (3, 30)	2.97*	<0.001

Values are shown as n or median (quartile). \**U* value. LCIV: Left common iliac vein.

**Table 3: Comparison of compression degree of LCIV between younger and older subgroups in both female and male groups**

Compression degree	Female		Statistics	P	Male		Statistics	P
	Younger (n = 37)	Older (n = 191)			Younger (n = 43)	Older (n = 229)		
≥25%	32	77			13	67		
≥50%	12	19			7	12		
Median (%)	42 (31, 50)	19 (5, 39)	5.14*	<0.001	11 (3, 40)	9 (3, 30)	0.34*	0.736

Values are shown as n or median (quartile). \**U* value. LCIV: Left common iliac vein.



**Figure 3:** Scatter diagram of the left common iliac vein compression degree in all patients. (a) Left common iliac vein compression degree in the female group (n = 228, 24% [8%, 42%]). (b) Left common iliac vein compression degree in the male group (n = 272, 9% [3%, 30%]). We observe that a compression degree of ≥25% is more common among young women than in men ( $\chi^2 = 17.85$ ,  $P < 0.01$ ).

Univariate Cox regression analysis showed that stenosis rate (Wald  $\chi^2 = 8.84$ , hazard ratio [HR] = 1.13, 95% confidence interval (CI) 1.04–1.23,  $P < 0.001$ ) and the diameter of the site of maximal compression (minimum diameter of LCIV, Wald  $\chi^2 = 6.29$ , HR = 0.00, 95% CI 0.00–0.27,  $P = 0.01$ ) correlated with the incidence of IVCS. Multivariable Cox regression analysis showed that the stenosis rate was an independent risk factor of IVCS (Wald  $\chi^2 = 8.84$ , HR = 1.13, 95% CI 1.04–1.23,  $P < 0.001$ ) (the onset of IVCS as the response variable, and stenosis rate and diameter of the site of maximal compression of LCIV as the independent variables).

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



**Table 4: Information of patients in whom target events occurred**

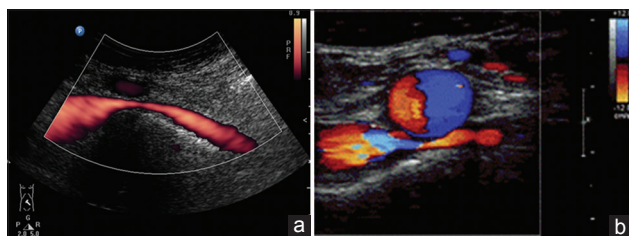
<i>n</i>	Gender	Age (years)	Target events	Compression degree (%)	Underlying disease	Occurrence time (months)
1	Female	48	Slow flow velocity in bilateral popliteal vein	2.01	Colon cancer (Stage IV)	6
2	Male	74	Bilateral superficial femoral vein thrombosis	3.15	Pancreatic cancer (Stage IV)	12
3	Female	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 IIb)	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 ( <i>n</i> = 224)	Nontumor group ( <i>n</i> = 143)	Statistics	<i>P</i>
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 <sup>9</sup> /L)	191.63 ± 101.51	203.03 ± 47.84	-1.55*	0.122
D-dimer (positive rate)	19.5%	2%	24.85 <sup>†</sup>	<0.01

\**t* value; <sup>†</sup> $\chi^2$  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%.

patients. The results of early autopsy and modern studies showed the incidence of IVC to be 20% to 49%.<sup>[4-10]</sup> In this study, 37.8% of patients had a LCIV compression degree  $\geq 25\%$ , while 9.8% had a compression degree  $\geq 50\%$ ; our results also demonstrated that LCIV compression was very common.

IVC occurs most commonly in young women, with 85% of cases occurring in women between 20 and 40 years old.<sup>[5]</sup> 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;<sup>[11]</sup> 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.<sup>[6,12]</sup> It may occur in 2–5% of patients who present with lower extremity venous diseases. Some studies

**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†	0.782
Mean diameter (cm), mean ± SD	0.91 ± 0.20	0.91 ± 0.20	1.00 ± 0.14	-1.02†	0.309
Minimum diameter (cm), mean ± SD	0.72 ± 0.28	0.72 ± 0.28	0.41 ± 0.09	7.98†	0.000
Stenosis rate (%), mean ± SD	0.22 ± 0.20	0.22 ± 0.20	0.58 ± 0.11	-4.43†	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.

showed that IVC exists in approximately 22–24% of these patients.<sup>[13-15]</sup> 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.

No statistical differences among patients with versus without IVCS were found in terms of age, gender, or other

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.<sup>[16]</sup> 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.<sup>[17]</sup> The patency of the iliac vein after treatment ranges from 78.3% to 100%.<sup>[18-22]</sup> 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.

### Financial support and sponsorship

Nil.

### Conflicts of interest

There are no conflicts of interest.

## REFERENCES

1. Virchow R. Uber die erweiterung kleiner gefasse [in German]. Arch Pathol Anat 1851;3:427.
2. May R, Thurner J. The cause of the predominantly sinistral occurrence of thrombosis of the pelvic veins. Angiology 1957;8:419-27. doi: 10.1177/000331975700800505.
3. Cockett FB, Thomas ML. The iliac compression syndrome. Br J Surg 1965;52:816-25. doi: 10.1002/bjs.1800521028.
4. Vaidyanathan S, Hughes P, Soni BM, Oo T, Singh G, Parsons KF, et al. Occlusion of left common iliac vein by a distended urinary bladder in a male with paraplegia due to spinal cord injury. Spinal Cord 2001;39:394-8. doi: 10.1038/sj.sc.3101165.
5. Mickley V, Schwagierek R, Rilinger N, Görlich J, Sunder-Plassmann L. Left iliac venous thrombosis caused by venous spur: Treatment with thrombectomy and stent implantation. J Vasc Surg 1998;28:492-7. doi: 10.1016/S0741-5214(98)70135-1.
6. Wolpert LM, Rahmani O, Stein B, Gallagher JJ, Drezner AD. Magnetic resonance venography in the diagnosis and management of May-Thurner syndrome. Vasc Endovascular Surg 2002;36:51-7. doi: 10.1177/153857440203600109.
7. Kibbe MR, Ujiki M, Goodwin AL, Eskandari M, Yao J, Matsumura J. Iliac vein compression in an asymptomatic patient population. J Vasc Surg 2004;39:937-43. doi: 10.1016/j.jvs.2003.12.032.
8. Raju S, Neglen P. High prevalence of nonthrombotic iliac vein lesions in chronic venous disease: A permissive role in pathogenicity. J Vasc Surg 2006;44:136-44. doi: 10.1016/j.jvs.2006.02.065.
9. Nazzal M, El-Fedaly M, Kazan V, Qu W, Renno AW, Al-Natour M, et al. Incidence and clinical significance of iliac vein compression. Vascular 2015;23:337-43. doi: 10.1177/1708538114551194.
10. Rigas A, Vomvoyannis A, Tzardakas E. Iliac compression syndrome. Report of ten cases. J Cardiovasc Surg (Torino) 1970;11:389-92.
11. McMurrich JP. The occurrence of congenital adhesion in the common iliac veins and their relation to thrombosis of the femoral and iliac vein. Am J Med Sci 1908;135:342-6. doi: 10.1097/00000441-190803000-00004.
12. Stein PD, Hull RD, Patel KC, Olson RE, Ghali WA, Alshab AK, et al. Venous thromboembolic disease. Arch Intern Med 2003;163:1843-8. doi: 10.1001/archinte.163.15.1843.
13. Steinberg JB, Jacocks MA. May-Thurner syndrome: A previously unreported variant. Ann Vasc Surg 1993;7:577-81. doi: 10.1007/BF02000154.
14. Oteros Fernandez R, Bravo Rodriguez F, Delgado Acosta F, González Barrios I. May-Thurner syndrome and surgery for scoliosis [in Spanish]. Radiologia 2008;50:245-7.
15. Khorana AA. Venous thromboembolism and prognosis in cancer. Thromb Res 2010;125:490-3. doi: 10.1016/j.thromres.2009.12.023.
16. Lalka SG, Malone JM. Hemodynamics of revascularization for iliofemoral venous occlusion: A short-term canine model. J Vasc Surg 1988;8:592-9. doi: 10.1016/0741-5214(88)90310-2.
17. Binkert CA, Schoch E, Stuckmann G, Largiader J, Wigger P, Schoepke W, et al. Treatment of pelvic venous spur (May-Thurner syndrome) with self-expanding metallic endoprostheses. Cardiovasc Intervent Radiol 1998;21:22-6. doi: 10.1007/s002709900205.
18. Titus JM, Moise MA, Bena J, Lyden SP, Clair DG. Iliofemoral stenting for venous occlusive disease. J Vasc Surg 2011;53:706-12. doi: 10.1016/j.jvs.2010.09.011.
19. Hurst DR, Forauer AR, Bloom JR, Greenfield LJ, Wakefield TW, Williams DM. Diagnosis and endovascular treatment of ilio caval compression syndrome. J Vasc Surg 2001;34:106-13. doi: 10.1067/mva.2001.114213.
20. Meng QY, Li XQ, Qian AM, Sang HF, Rong JJ, Zhu LW. Endovascular treatment of iliac vein compression syndrome. Chin Med J 2011;124:2281-4.
21. Wang YP, Zhang XQ, Yu WN, Hao B, Ren KW, Pan JJ, et al. Endovascular treatment of acute proximal deep venous thrombosis secondary to iliac vein compression syndrome: A novel technique for thrombus removal. Chin Med J 2013;126:3184-6.
22. Shebel ND, Whalen CC. Diagnosis and management of iliac vein compression syndrome. J Vasc Nurs 2005;23:10-7. doi: 10.1016/j.jvn.2004.12.001.