Observational Study

Medicine®

OPEN

Application of computed tomography venography in the diagnosis and severity assessment of iliac vein compression syndrome

A retrospective study

Peng Liu, MD[a], Junlu Peng, MM[a], Lihua Zheng, MD[a], Haili Lu, MD[b], Weifang Yu, MD[a], Xia Jiang, MD[a], Lei Zhang, MD[a], Haixia Song, MD[b], Zengren Zhao, PhD[a,*]

Abstract

The objectives are to evaluate the application of computed tomography venography (CTV) in the diagnosis of iliac vein compression syndrome (IVCS), and to assess the factors related to the incidence and development of IVCS and the recurrence of varicose veins.

Imaging data of 120 patients with chronic venous disease (CVD) of the lower extremity and 68 subjects without CVD (control) were retrospectively reviewed by radiologists blinded to the groups. CTV, conventional venography, and Doppler ultrasound were compared in the diagnosis and contributing factors for IVCS were also analyzed.

CTV required less procedure time than venography or color ultrasonography ($P<.001$). The rate of iliac venous compression diagnosed by CTV was higher in the CVD group (53.3%) than in the control group (22.1%) ($\chi^2=17.425$, $P<.001$). Risk factors for IVCS included gender, hyperlipidemia, and course of disease ($P<.05$). Development of femoral vein collateral was more common in patients with IVCS ($P<.05$). The duration of disease was positively associated with the severity of iliac vein compression ($r=0.321$, $P<.001$). IVCS was an important contributing factor for varicose vein recurrence (51.2%). In patients with IVCS and venous ulcer (C5-C6), the healing time of the ulcer treated with stent was significantly shorter compared with those without stent treatment ($P<.001$).

CTV is accurate for the diagnosis and severity evaluation of IVCS. IVCS might be a contributing factor for varicose vein recurrence. Iliac vein stent implantation as a safe and effective interventional therapy promotes the healing of venous ulcer caused by IVCS.

Abbreviations: CTV = computed tomography venography, CVD = chronic venous disease, IVCS = iliac vein compression syndrome.

Keywords: computed tomography venography, iliac vein compression syndrome, recurrence, stent, venous ulcer

1. Introduction

Iliac vein compression syndrome (IVCS) is proverbially caused by compression of the left common venous outflow tract of the lower extremity. Clinically, IVCS is symptomized by leg swelling, pain, varicose veins, and deep vein thrombosis.[1] The conventional understanding of IVCS is the occlusion or stenosis of the left common iliac vein caused by chronic compression imposed by the right iliac artery or internal lesions.[2] However, compression of the right common iliac vein and iliac vein compression differs from May-Thurner syndrome, and contributes to general IVCS.[3]

It has been reported that 20% to 30% adults have symptomatic or asymptomatic iliac vein compression, with a higher prevalence among women than men.[4] Doppler ultrasound, computed tomography venography (CTV), venography, and magnetic resonance venography have been applied to diagnose IVCS. With the development of interventional technology, it is now believed that the prevalence of IVCS has been far underestimated,[5] especially upon consideration that iliac vein compression is often asymptomatic.[6] In addition, IVCS often afflicts young patients, for whom diagnostic accuracy and evaluation of severity are necessary for guiding endovascular management.[7]

Noninvasive ultrasound examination has been used for detecting IVCS, but is obviously limited due to technical difficulties in viewing the pelvic veins. Duplex ultrasonography is used to evaluate the anatomy of the venous system, but is also obviously limited by operator-dependent subjectivity, the time-consuming procedure, and the difficulty of assessing the iliac veins and inferior vena cava from within the abdomen.[4] Conventional venography is another standard for the diagnosis of IVCS, but currently is limited to cases that present with diagnostic challenges or in which intervention is planned. As a more effective alternative, CTV, which accurately evaluates IVCS and detects the external pressure source of the iliac vein, has been increasingly applied.[4] Evaluation of the venous system in the entire lower extremity is essential for safe and effective treatment.

Editor: Bernhard Schaller.

The study is funded by Medical Science Research Project of Health and Family Planning Commission of Hebei Province, China (Nos. 20160690, 20160691).

The authors have no conflicts of interest to disclose.

Supplemental Digital Content is available for this article.

[a] *Department of General Surgery, The First Hospital of Hebei Medical University,* [b] *Department of Neurology, The First Hospital of Shijiazhuang, Hebei, China.*

[*] *Correspondence: Professor Zengren Zhao, M.D., Ph.D., Department of General Surgery, The First Hospital of Hebei Medical University, Donggang Road 89, Shijiazhuang 050031, Hebei, China. (e-mail: l: zzr-doctor@163.com).*

Medicine (2018) 97:34(e12002)

Received: 21 March 2018 / Accepted: 23 July 2018

http://dx.doi.org/10.1097/MD.0000000000012002

Herein, in this study we evaluated the use of CTV for the diagnosis of IVCS in local Chinese patients, and assessed the factors that are potentially related to the development and severity of IVCS and the recurrence of varicose veins.

2. Materials and methods

2.1. Patients

In total, 120 patients with chronic venous disease (CVD; 80 men and 40 women; aged 22 to 84 years, median age 59.0 years) were retrospectively reviewed from April 2015 to May 2016 in the Department of Vascular Surgery in the First Hospital of Hebei Medical University, and 68 patients with non-CVD enrolled in the outpatient clinic (including 38 men and 30 women, aged 34–83 years, median age 53.5 years) were set as control. Among the 120 patients in CVD group, 78 had undergone CTV, conventional venography, and color ultrasonography, and 42 had CTV and color ultrasonography. Sixty-eight controls had also undergone CTV. Informed consent was obtained from each patient regarding the publication of demographic, clinical, and imaging data. This study was approved by the Ethics Review Board of the First Hospital of Hebei Medical University. The study also followed the Declaration of Helsinki.

CVD was diagnosed based on the structural or functional abnormalities of the veins, including telangiectasia, reticular and varicose veins, edma; and skin changes, including pigmentation, lipodermatosclerosis, dermatitis and ulceration.[4] Patients with any of the following criteria were excluded from this study: younger than 18 years; history of allergy to iodinated contrast media; peripheral arterial occlusive disease; chronic renal failure; pregnancy; pelvic or abdominal aneurysms; CVD caused by Klippel–Trenaunay–Weber syndrome, Budd–Chiari syndrome, post-thrombotic syndrome, or deep vein thrombosis.

2.2. CTV

Contrast-media allergy test was performed to preclude patients. Patients were examined with a 64-detector CT scanner (GE Healthcare, WI, Boston, MA) using a tube voltage of 110 kV and current of 200 mA. The thickness of prespecified slice was set at 1.00 mm and spiral pitch at 1.375 mm. A power injector (E-Z-EM, Westbury, NY) was connected to an indwelling needle in the dorsal pedal vein with ultrasound guidance. A bolus of 2.5 mL/kg of nonionic contrast material (350 mg/mL, Iohecol Injection, Yangtze River Pharmaceutical Group, TaiZhou, China) was administered with saline at a speed of 2 mL/s. Scanning was delayed 30 seconds after injection of the iodinated contrast media and covered both legs, from the ankle to diaphragm. The raw data were uploaded to a workstation (GE Healthcare, CT Advantage Workstation 4.4). Analysis of obstructions in regions of interest was conducted in axial, sagittal, and coronal slices. Reconstruction included multiplanar reconstructions and volume rendering. Two cardiovascular radiologists with 8- and 20-year of clinical experience and blinded to the patient's symptoms reviewed the films and analyzed the images.

2.3. Conventional venography

Conventional venography (Philips Medical Systems, The Netherlands) was applied to capture venograms of the patients in supine position. Similar to the CTV scanning protocol, an ankle tourniquet was applied to the ankle and a binocular high-pressure syringe was connected to the indwelling needle in the dorsalis pedis vein with ultrasound guidance to administrate nonionic contrast material (350 mg/mL, Iohecol Injection, Yangtze River Pharmaceutical Group, TaiZhou, China) solution (1:1 mixed with 40 mL saline) within 1 min. Reflux in the deep vein was monitored.

After injection of 20 mL contrast media, the calf muscle was rapidly compressed manually with both hands to speed the reflux of contrast agent to fill the IVC. The iliac vein and iliac vein compression was captured by imaging. The superficial vein flow was observed after release of the tourniquet. To determine potential circulation abnormalities, the patients were required to perform a forceful attempted exhalation against a closed airway (Valsalva maneuver) to monitor venous return and compression. Two cardiovascular radiologists with 8- and 20-year of clinical experience and blinded to the patients' symptoms reviewed the films and analyzed the images.

2.4. Classification of iliac vein compression

The severity of iliac venous compression was determined by the diameter of the common iliac vein at the site of maximal compression divided by the diameter of the uncompressed caudal common iliac vein. A luminal compression of the iliac vein > 50% tested in the computed tomography (CT) image cross-section was considered clinically significant. In CT images, iliac vein compression was classified as 3 grades (I, II, III) based on severity of compression: grade I, < 50% compression to the iliac vein (Fig. 1A, Figure S1A, http://links.lww.com/MD/C422); grade II, iliac vein compression > 50% without development of collateral circulation (Fig. 1B, Figure S1B, http://links.lww.com/MD/C422); or grade III, iliac vein compression > 50% accompanied by formation of collateral veins (Fig. 1C, Figure S1C, http://links.lww.com/MD/C422). Iliac vein compression < 50% with development of collateral circulation was not observable in the present study.

IVCS was defined as iliac vein compression > 50% in CVD patients. According to the above-mentioned classification, IVCS included patients with grade II or grade III of iliac vein compression in the CVD group. Images were randomly collected by 2 independent examiners who were blinded to the study allocation. If there was a discrepancy in the initial categorization, a third examiner reviewed the image.

In CTV images, iliac vein compression was divided into 3 types (A, B, C), according to the cause of compression: type A, compression caused by the right iliac artery (Fig. 1D); type B, vascular compression from vessels other than the right iliac artery (Fig. 1E); type C, compression by other occupying structures (lymph node, tumor, or intervertebral disc; Fig. 1F).

2.5. Parameters observed and follow-up

Based on our clinical experience, factors that may be involved in the development of iliac vein compression include continuous cold-water stimulus and venous bifurcation angle (Figure S2, http://links.lww.com/MD/C422), and femoral vein collateral formation (Figure S3, http://links.lww.com/MD/C422). Continuous cold-water stimulus was defined as intermittent or continuous exposure to cold water (< 4°C) ≥ 20 min/d for > 30 days in winter. The venous bifurcation angle was defined as the angle between the line vertical to the line connecting the near and far end of the opening of the left iliac vein to the IVC and the line parallel to the long axis line of IVC.

Smoking was defined when 1 person smoked at least 1 cigarette/d, with continuous or cumulative smoking for ≥ 6 months as

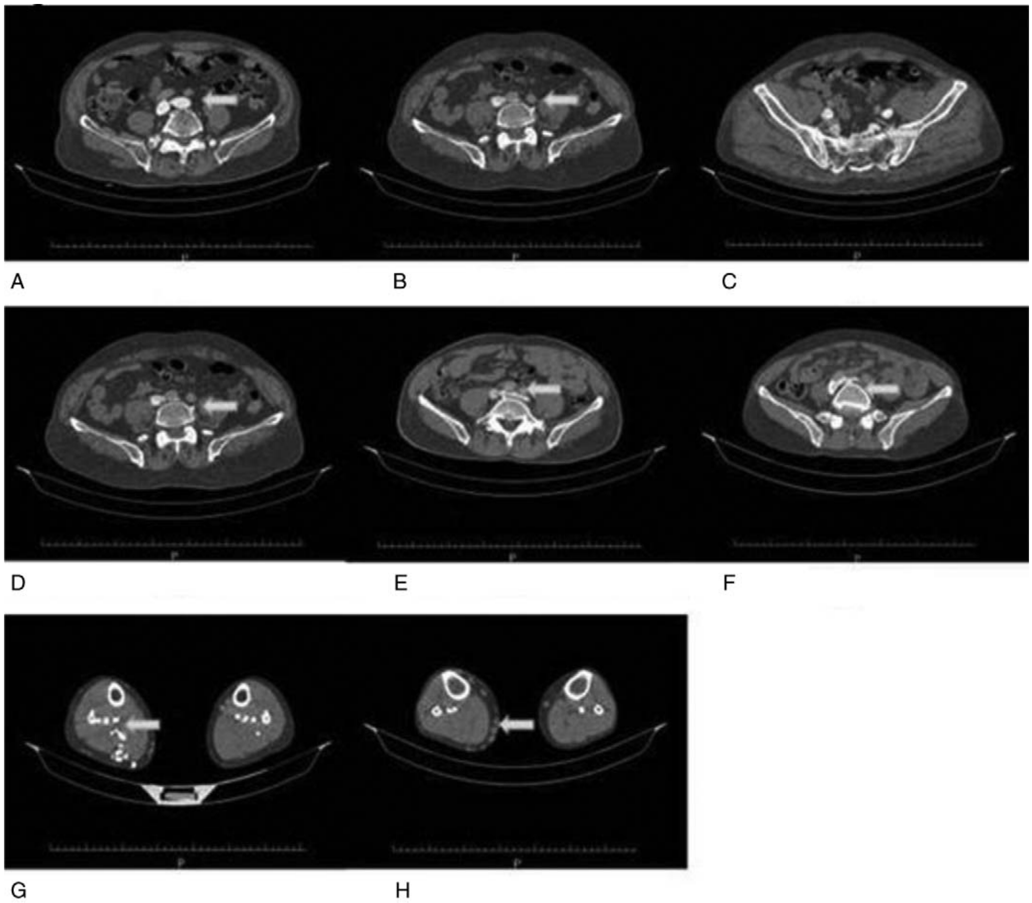

Figure 1. Axial CT scans of compression involving the iliac vein. A, Grade I, no compression or <50% compression to iliac vein. B, Grade II, iliac vein compression>50%, without development of collateral circulation. C, Grade III, iliac vein compression > 50% with collaterals. D–F, Types of IVCS categorized by compression source. D, Type A, left iliac vein compressed by right iliac artery. E, Type B, left iliac vein compressed by vascular (s) other than the right iliac artery. F, Type C, iliac vein compressed by other occupying structures. G and H, Enhanced reflux of communicating veins and postoperative residual trunk of the varicose veins, respectively. CT = computed tomography.

previously described.[8] Drinking was considered when 1 person drunk ≥ 2 drinks per day (10 g pure alcohol per drink). The course of disease was defined as the duration of disease from admission of the CVD patient to discharge.

To more accurately evaluate the severity and affected CVD region, the Clinical-Etiology-Anatomy-Pathophysiology (CEAP) classification with refined C classes was applied (C5, healed venous ulcer; C6, active venous ulcer). Using an ultrasonic Doppler instrument (M5, Mindray Medical International, ShenZhen, China), reflux was defined as retrograde flow lasting ≥ 0.5 seconds with the Valsalva maneuver, in the proximal part of the vein, or evaluated by manual compression and decompression of the calf to assess the distal part of the vein. The deep and superficial systems were scanned from the groin down to the ankle.

Healing of a vascular ulcer was defined as complete epithelialization, with the healing time recorded from the date of operation to the last follow-up or healing of the venous leg ulcer.

Patients were advised to attend follow-up every 1 month, and annually thereafter for clinical assessment and venous duplex ultrasonography performed. Ulcers were assessed clinically with regard to whether they had healed (completely epithelialized or incompletely epithelialized). The healing rate was calculated by dividing the number of healed patients by the total number of patients during the follow-up.

2.6. Statistical analysis

Quantitative measures were represented as mean ± standard deviation. Equality of variances was confirmed by Levene test and difference between groups was analyzed by *t* test (for parametric variables). Parametric variables were described using frequency statistics and analyzed by the chi-squared test (χ^2) or corrected χ^2 test. Correlation analysis was performed by Spearman coefficient analysis. Logistic regression was conducted to detect potential risk factors of iliac vein compression. The healing rate of leg venous ulcer and patency rate of iliac vein were analyzed using the Kaplan–Meier limit product method. Statistical significance was considered $P < .05$. Data were analyzed using SPSS 22.0 software (SPSS Inc, Chicago, IL).

3. Results

3.1. Comparison of CTV, conventional venography, and vein color Doppler for detecting iliac vein compression

CTV required less procedure time than conventional venography or color ultrasonography ($P < .001$) (Table 1). Comparing the 3 modalities, communicating venous reflux was shown in 24/78, 21/78, and 12/78 patients via CTV, venography, and color Doppler, respectively. In addition, CTV provided information regarding the possible factors for IVCS in 48/78 patients,

Table 1

Detection of iliac vein compression using CTV, venography, and color Doppler in 78 CVD patients*.

	CTV	Venography	Color Doppler	χ^2	P
Detection of iliac vein compression	48	33	—	1.926	.267
Cause of iliac vein compression	48	—	—	—	—
Procedure time, min	15.4±1.9	28.2±1.9	24.2±2.0	64.411	.000
Superficial venous reflux	—	33	45	1.231	.406
Deep venous reflux	—	33	39	0.310	.781
Communicating venous reflux	24	21	12	1.844	.505

The mark "—" indicates that the imaging method is not sensitive enough to detect the corresponding item.
CD = color Doppler, CTV = computed tomography venography.
* Reported as n, unless indicated otherwise.

compared with 33/78 with venography, and 0/78 with color Doppler. The rates of positive findings for superficial and deep venous reflux, respectively, using the 3 modalities, were as follows: color Doppler, 45/78 and 39/78; CT venography, 0/78 and 0/78; and venography, 33/78 and 33/78 (Table 1).

3.2. Prevalence of iliac vein compression assessed by CTV

The prevalence of iliac vein compression was significantly higher in the CVD group (64/120, 53.3%) compared with the non-CVD control group (15/68, 22.1%; $\chi^2=17.425$, $P<.001$).

3.3. Contributing factors for iliac vein compression and IVCS in CVD patients

Analysis of the relevance of categorical variables indicated that gender, and cold-water stimulation were significant factors for iliac vein compression in CVD patients ($P<.05$) (Table 2). Further analysis of the relevance of measurement data showed that, among the parameters observed (i.e., age, years of education, course of disease, body mass index, and bifurcation angle), only the course of disease was significantly associated with iliac vein compression in CVD patients (Table S1, http://links.lww.com/MD/C422). The logistic regression analysis showed that gender and hyperlipidemia were significant factors for IVCS (Table S2, http://links.lww.com/MD/C422). The risk of iliac vein compression was higher among women and patients with hyperlipidemia, and increased with the duration of disease ($P<.05$).

3.4. Classification of iliac vein compression and clinical characteristics

The clinical characteristics were analyzed according to the grade of iliac vein compression among the 120 patients in the CVD group (Table S3, http://links.lww.com/MD/C422). The rate of femoral vein collateral circulation was higher in the patients with IVCS (i.e., grades II and III) compared with those with grade I ($P=.039$). This indicated that the formation of femoral vein collateral circulation was associated with more severe compression. Patients with grades I and III had a higher risk of morbidity from varicosity compared with grade II ($P=.002$) (Table S3, http://links.lww.com/MD/C422). Cold-water stimulation contributed to higher rates of iliac vein compression. Potential factors related to the degree of iliac vein compression were analyzed (Table 3). The duration of disease ($r=0.321$, $P<.001$) was positively associated with the severity of iliac vein compression.

3.5. Risk factors for recurrent varicose veins using CTV

Of the 120 patients in the CVD group, 41 limbs in 31 patients developed recurrence of varicose veins of the lower extremity during the follow-up of 0.5 to 19 years (8.6±6.2 years). CTV revealed the source of the recurrence as follows: IVCS (21/41); venous reflux of the communicating branch (17/41; Fig. 1G); and

Table 2

Potential relevant factors in iliac vein compression: categorical variables.

		Without compression	With compression	χ^2	P
Gender	Male	45	35	8.856	.003
	Female	11	29		
Bifurcation site	L4 and above	50	6	0.060	.807
	Below L4	58	6		
Cold water stimulation		2	14	8.659	.003
Smoking		18	19	0.084	.771
Alcohol consumption		15	11	1.621	.203
Hypertension		16	24	1.071	.301
Diabetes		5	11	1.763	.184
Hyperlipidemia		11	24	3.673	.055
Homocysteinemia		19	15	1.619	.203
Coronary heart disease		13	7	3.241	.072
Lasting upright position		35	37	0.273	.601
Varicosity of SSV		3	5	0.029	.864

SSV = small saphenous vein.

Table 3

Association between severity of iliac vein compression and clinical characteristics in 120 patients.

	R	*P*
Age	0.154	.093
Duration of disease	0.342	.000
BMI	–0.041	.653
Bifurcation angle	–0.056	.541
CEAP classification	0.253	.105

BMI = body mass index, CEAP = clinical-etiology-anatomy-pathophysiology.

residual saphenous trunk (8/41; Fig. 1H). There were 7 patients who suffered from communicating branch and residual saphenous trunk simultaneously.

3.6. Analysis of treatment on IVCS with CEAP C5 and C6

All the patients in the CVD group who were affected by CEAP C5 or C6 venous ulcer of the lower limb (n = 24) were treated surgically, while 6 of the total with IVCS were in the stenting group (Fig. 2). These patients were categorized into 3 groups (A, B, and C) based on the presence of IVCS and implantation of iliac vein stenting (Table 4). Incomplete ulcer healing remained in 4 patients in Group B and 1 patient in Group C. The ulcer-free survival rates of groups A and C were both significantly higher compared with group B, in which patients were not treated with implantation of iliac vein stents ($P = .002$, Fig. 3). One patient (16.7%) suffered from minor bleeding at puncture point and was successfully treated with compression. The patency rates of iliac vein underwent stent implantation at 3-, 6- months, 1 year were 100.0%, 100.0%, 83.3%, respectively (Figure S4, http://links.lww.com/MD/C422).

Table 4

Outcome of 3 groups of patients treated differently for iliac venous ulcers*.

	Group A	Group B	Group C
IVCS	–	+	+
Iliac vein stent	–	+	–
Age, y	59.9 ± 16.9	51.5 ± 10.1	57.7 ± 17.3
BMI	27.2 ± 1.4	26.2 ± 1.7	26.7 ± 4.3
Varicose course, y	13.4 ± 11.4	13.9 ± 11.3	10.8 ± 11.0
Ulcer area, cm^2	4.0 ± 2.1	3.9 ± 2.2	4.7 ± 2.4
Ulcer healing rate	9/10 (90.0%)	6/6 (100.0%)	4/8 (50.0%)
Healing time, d	21.2 ± 2.1	19.2 ± 1.9	36.3 ± 8.0
95% CI	(17.1, 25.3)	(15.5, 22.8)	(20.6., 51.9)

IVCS = iliac vein compression syndrome.
* n = 24

4. Discussion

In the current study, we evaluated CTV relative to venography or color Doppler for detecting IVCS among patients with CVD. We found that among these imaging techniques, CT venography was effective for detecting IVCS. As iliac vein stenosis approaches closure of up to half the diameter of the normal vein, the incidence of venous thrombosis and clinical symptoms greatly increase.[4] In the present study, we differentiated simple iliac vein compression from IVCS. Iliac vein compression with a venous cross-sectional narrowing of > 50% was considered clinically significant. IVCS was diagnosed with iliac vein compression complicated with CVD. Iliac venous compression was further classified at 3 levels according to the degree of compression. Formation of collateral veins was usually accompanied with more severe compression (grade III compression).

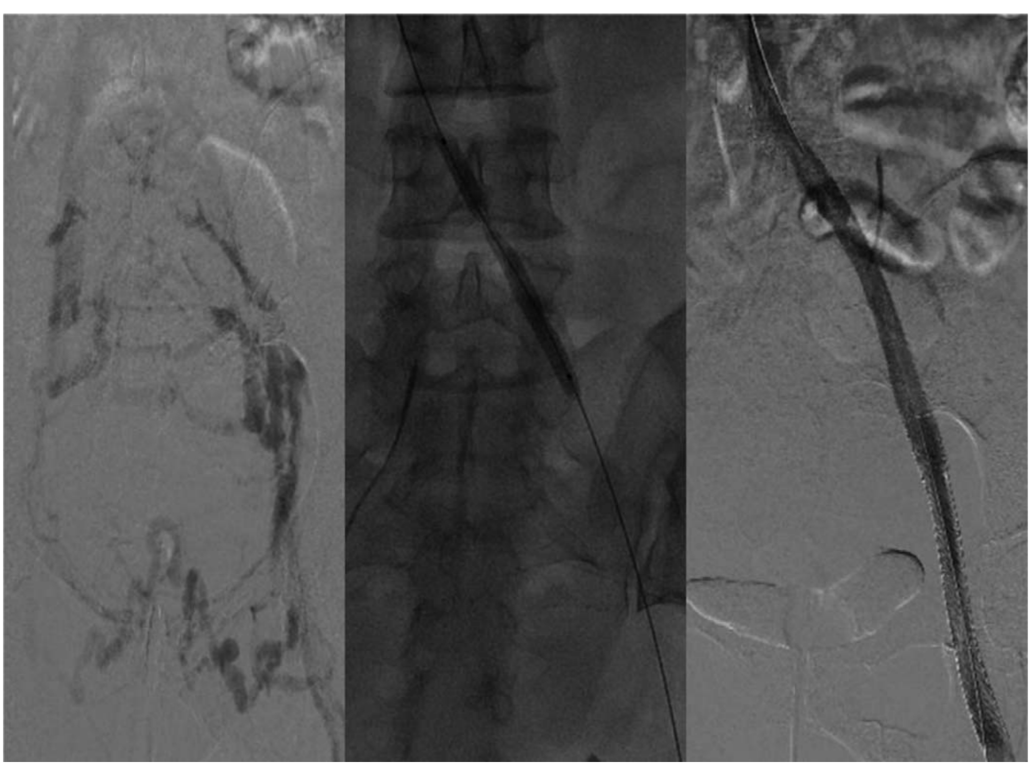

Figure 2. IVCS with CEAP C5 and C6 before and after stenting. CEAP = clinical-etiology-anatomy-pathophysiology, IVCS = iliac vein compression syndrome.

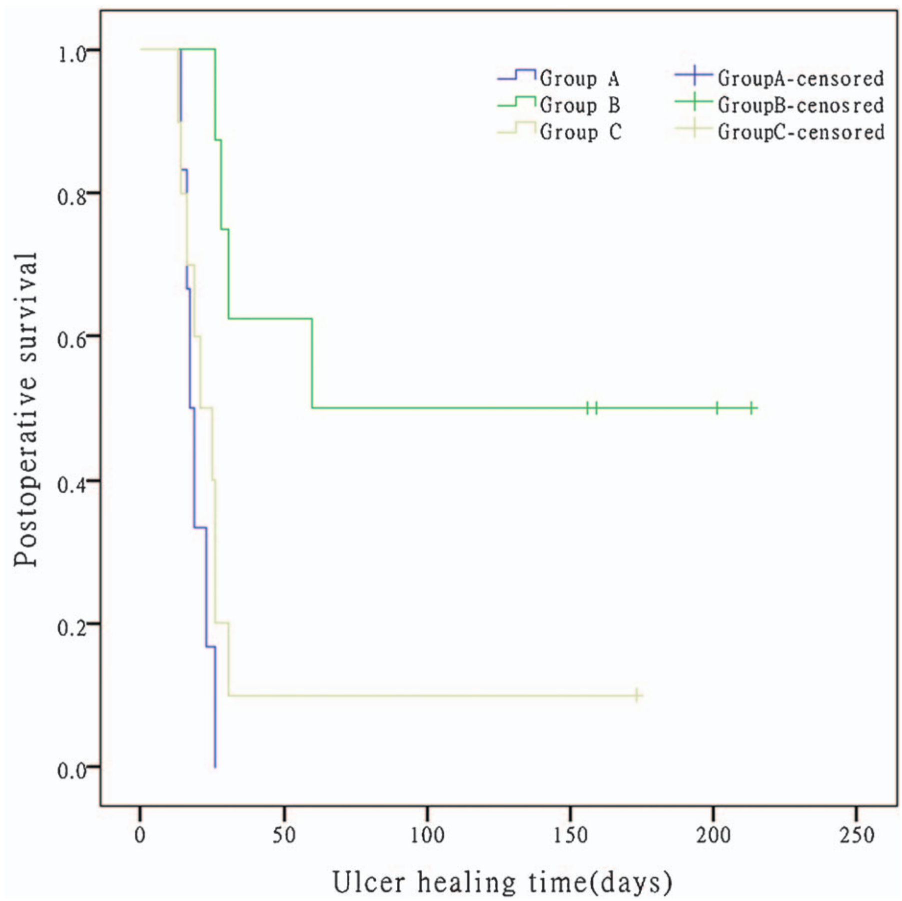

Figure 3. Ulcer healing duration was comparable between groups A and C ($\chi^2 = 1.287$, $P = .257$), and both were significantly shorter than that of group B (without stent; $\chi^2 = 12.635$, $P = .002$).

Initially, we confirmed that the severity of iliac vein compression was positively associated with the duration of CVD.[9] This may also be related to the presence of iliac vein spurs that result from extrinsic compression.[2] The formation rate of collateral femoral veins was significantly higher in patients with IVCS than in the non-IVCS control cohort, and the collateral formation rate significantly increased with compression. This may be due to the increasing venous pressure caused by femoral vein reflux, which leads to venous hypertension of the lower extremity, vascular dilatation of normal anastomotic branch, and the formation of collateral circulation.

To determine the potential risk factors for the recurrence of varicose veins using CTV in the present study, 41 limbs with recurrence were analyzed. Of the 41 patients, 21 (51.2%) had IVCS. Clinical evidence shows that insufficiency of chronic venous is linked to chronic venous hypertension and venous reflux, which is the result of restructuration of superficial veins and venules caused by the growth and remodeling of the venus wall.[4] The major reason that causes venous hypertension in the lower extremities with IVCS is due to a pressure gradient of > 2 mm Hg across the stenosis.[4] The embolization of dysfunctional pelvic veins effectively reduced the risk of postoperative recurrence.[4] This suggests that venous hypertension and venous reflux caused by iliac vein compression narrowing may be risk factors of varicose vein recurrence. The recurrence of varicose veins may be related to insufficient ligation of the saphenous vein trunk or erroneous stripping of the medial and lateral femoral veins during the initial surgery,[4] which is supported by our results. In our study, 8 of the 41 (19.5%) had residual saphenous trunk. Moreover, the presence of a communicating vein around the ulcer was also conductive to recurrence of venous ulcer,[10] which was detected by CTV in 17 of 41 (41.5%) patients.

We first reported cold-water stimulation as a stress factor in IVCS, based on the observation that the incidence of IVCS increased as the frequency of cold-water stimulation increased. Long-term cold water stimulation led to a series of pathological changes, including hypoxia, chronic inflammation, and blood flow disturbance.[4] Blood pooling in the lower extremity and chronic inflammation in response to vascular wall hypoxia may further aggravate the progressive fibrotic narrowing of the iliac vein wall.

Asymptomatic iliac vein compression does not warrant treatment. Conservative methods such as compression stockings and systemic anticoagulation are appropriate initial treatments for early signs of IVCS.[11] There are multiple procedures to manage chronic venous pressure, venous reflux, or flow obstruction to treat venous ulcer, with an optimal selection that is yet to be determined.[12] Endovascular interventions to the deep veins appear to be an effective adjunct in achieving the healing of recalcitrant ulcers.[4] The aim of stent implantation of IVCS is to remove iliac venous outflow obstruction, reducing venous pressure of the lower limb and relieving symptoms of chronic venous insufficiency and to prevent deep vein thrombosis of the lower extremities. In the present study, only CEAP 5 or 6 with iliac venous compression was treated by placing a stent to open the blocked segment of the vein. We thereby improved access to the lower extremity vein to keep blood from remaining in the leg, and improved healing by facilitating the local metabolic environment. Our experience indicates that the rate of ulcer healing via stent (6/6) is significantly higher than that effected by other treatments (9/10, 4/8), and the healing time is significantly reduced as well.

Several limitations of our study are discussed as follows. First, due to the limited study population collected in a single institution, the power of the analysis may be restricted by selection bias. Second, CTV is not preferred for studying hemodynamics and internal lesions, and patients are exposed to radiation. Third, the evaluation of blood vessel diameter using CTV is dependent on the subjective judgement of the image. Fourth, CT venography should prompt a search for evaluation of blood vessel area. Some patients with CEAP C3 and C4 will need further interventions on iliac systems. Despite the longer scan time, magnetic resonance venography mitigates the risk of ionizing radiation exposure compared with CT venography, and is usually able to obtain the optimal venous contrast timing. Magnetic resonance flow-sensitive sequences allow predicting the hemodynamic significance of compressive lesions, by assessing the direction of venous flow.[13] However, because of the lack of equipment, magnetic resonance venography examination was not performed in our hospital. In addition, IVCS not only leads to venous hemodynamic changes and partial venous occlusion, but also is considered a probable risk factor for developing DVT of the limbs.[14] The rate of ulcer healing via stent is higher than that treated by other methods. Nevertheless, further research is needed to confirm the mechanism, therapeutic effects, and safety.

5. Conclusion

CTV can provide an overview, as well as the details of relevant anatomic structures and help diagnose the recurrence of varicose veins during follow-up. In addition, iliac vein stent implantation promotes the healing of venous ulcer caused by IVCS.

Author contributions

PL carried out the data analyses and wrote the manuscript. JP, LZ, HL, WY, XJ, LZ, HS helped perform the analysis with constructive discussions. ZZ conceived of the study, and participated in its design and coordination and helped to draft the manuscript. All authors read and approved the final manuscript.

Conceptualization: Peng Liu, Zengren Zhao.
Data curation: Peng Liu, Junlu Peng, Lihua Zheng, Weifang Yu, Xia Jiang, Lei Zhang, Haixia Song, Zengren Zhao.
Formal analysis: Junlu Peng, Zengren Zhao.
Funding acquisition: Zengren Zhao.
Investigation: Junlu Peng, Haili Lu, Lei Zhang, Zengren Zhao.
Methodology: Junlu Peng, Lihua Zheng, Lei Zhang, Haixia Song, Zengren Zhao.
Project administration: Lihua Zheng, Haili Lu, Weifang Yu, Xia Jiang, Zengren Zhao.
Resources: Lihua Zheng, Haili Lu, Haixia Song, Zengren Zhao.
Software: Haixia Song, Zengren Zhao.
Supervision: Lei Zhang, Zengren Zhao.
Validation: Lihua Zheng, Zengren Zhao.
Visualization: Zengren Zhao.
Writing – original draft: Peng Liu, Junlu Peng, Haili Lu, Lei Zhang, Zengren Zhao.
Writing – review & editing: Zengren Zhao.

References

[1] Shebel ND, Whalen CC. Diagnosis and management of iliac vein compression syndrome. J Vasc Nurs 2005;23:10–7.
[2] May R, Thurner J. The cause of predominantly sinistral occurrence of thrombosis of the pelvic veins. Angiology 1957;8:419–27.
[3] Lugo-Fagundo C, Nance JW, Johnson PT, et al. May-Thurner syndrome: MDCT findings and clinical correlates. Abdom Radiol (NY) 2016;41:2026–30.
[4] Moudgill N., Hager E., Gonsalves C., et al. May-Thurner syndrome: case report and review of the literature involving modern endovascular therapy. Vascular 2009;17:330–5.
[5] Mickley V, Schwagierek R, Rilinger N, et al. Left iliac venous thrombosis caused by venous spur: treatment with thrombectomy and stent implantation. J Vasc Surg 1998;28:492–7.
[6] Liu Z, Gao N, Shen L, et al. Endovascular treatment for symptomatic iliac vein compression syndrome: a prospective consecutive series of 48 patients. Ann Vasc Surg 2014;28:695–704.
[7] Brazeau NF, Harvey HB, Pinto EG, et al. May-Thurner syndrome: diagnosis and management. Vasa 2013;42:96–105.
[8] Organization WH. Gluidelines for Controlling and Monitoring the Tobacco Epidemic. Tobacco or Health Programme, WHO, Geneva:1997.
[9] Brinegar KN, Sheth RA, Khademhosseini A, et al. Iliac vein compression syndrome: clinical, imaging and pathologic findings. World J Radiol 2015;7:375–81.
[10] van Neer PA, Veraart JC, Neumann HA. Venae perforantes: a clinical review. Dermatol Surg 2003;29:931–42.
[11] Hulsberg PC, McLoney E, Partovi S, et al. Minimally invasive treatments for venous compression syndromes. Cardiovasc Diagn Ther 2016;6:582–92.
[12] Harlander-Locke M, Lawrence PF, Alktaifi A, et al. The impact of ablation of incompetent superficial and perforator veins on ulcer healing rates. J Vasc Surg 2012;55:458–64.
[13] Zucker EJ, Suvranu G, Brian B. Imaging of venous compression syndrome. Cardiovasc Diagn Tber 2016;6:519–32.
[14] Oguzkurt L, Ozkan U, Ulusan S, et al. Compression of the left common iliac vein in asymptomatic subjects and patients with left iliofemoral deep vein thrombosis. J Vasc Interv Radiol 2008;19:366–70.