ORIGINAL ARTICLE

CONTENTS

Indicators of venous blood flow in the long-term period after inferior vena cava thrombosis

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ABSTRACT

Aim: To evaluate central haemodynamic parameters in the long-term period after inferior vena cava (IVC) thrombosis depending on the treatment method.

Materials and Methods: Blood flow parameters were evaluated in 25 patients after IVC thrombosis. All patients formed the I (main) group. The results of the examination of patients in group I were compared with group II (control) consisting of 10 healthy individuals.

Results: Open thromboectomy from the lumen of the IVC combined with incomplete mechanical cavaplication allows not only to restore the lumen of the venous trunk and prevent the development of pulmonary embolism, but also to quickly normalise the parameters of central and regional venous haemodynamics. Analysis of blood flow parameters in the early and late postoperative periods allows us to conclude that partial mechanical cavaplication does not impair venous return to the heart, and open thromboectomy prevents the development of post-thrombotic changes. Conservative treatment of IVC thrombosis accelerates recanalisation processes compared to the natural course of thrombosis, but during this time, natural venous collaterals develop, which ensure venous outflow from the lower half of the body and remain active even after restoration of the IVC lumen.

Conclusions: Open thrombectomy of the inferior vena cava allows restoration of the lumen, ensuring effective venous blood flow, prevent post-thrombotic syndrome. Cavoplication prevents thromboembolic complications, increases blood flow velocity and leads to spontaneous restoration of the lumen of the inferior vena cava within 14.3±2.2 months ($p \leq 0.01$).

KEY WORDS: inferior vena cava, venous hemodynamics, surgical intervention, conservative treatment, pulmonary embolism.

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INTRODUCTION

The annual incidence of deep vein thrombosis (DVT) is 1 case per 10,000 people under the age of 40 and 5–6 cases per 1,000 people over the age of 80. The recurrence rate for DVT reaches 5–10% per year, and the mortality rate reaches 4.6–10.0% within the first month [1]. In 15% of patients with DVT, thrombotic masses are found in the inferior vena cava (IVC) [2].

IVC thrombosis doubles the risk of fatal pulmonary embolism (PE) [3, 4]. Treatment of IVC thrombosis depends on the patient's clinical condition, the cause of thrombosis, the identification of risk factors and the benefits of the proposed treatment method, as well as the treatment methods available at the medical facility [5].

In cases of IVC thrombosis, the authors suggest starting anticoagulant therapy, thromboaspiration and/or thrombolytic therapy [6–8]. The use of cava-filters in IVC thrombosis is quite controversial, as complications after their implantation may exceed the therapeutic effect [9, 10], in particular, recurrent thrombosis is observed in 1.6–33% of patients [6, 11, 12]. In the presence of implant thrombosis, stenting of the IVC with subsequent radio- or chemotherapy has been proposed, which ensures rapid regression of clinical manifestations [13].

In the absence of adequate treatment of IVC thrombosis, post-thrombotic syndrome occurs in 20–90% of patients, leading to severe venous insufficiency in 5–45% of cases and the development of trophic ulcers in 15–18.8% of patients [1, 6, 14–16], causing significant damage to quality of life with professional and financial consequences [1, 15]. The recurrence rate of PE increases to 30% with a fatal outcome in 5.3% of patients [6, 14, 16].

However, according to Klein-Weigel P.F. et al. (2021), despite a number of studies, many clinically important questions in the management of patients with IVC remain unresolved, including optimised strategies for patient selection for interventions, antithrombotic and antiplatelet treatment regimens to prevent thrombosis recurrence [5]. In addition, the available literature does not assess haemodynamic changes in IVC thrombosis.

AIM

To evaluate central haemodynamic parameters in the long term after inferior vena cava thrombosis, depending on the treatment method.

MATERIALS AND METHODS

An assessment of blood flow parameters in the IVC was performed in 25 patients 3–5 years after IVC thrombosis. All patients with long-term consequences of IVC thrombosis were examined on an outpatient basis and formed the first (main) group. The average age of patients in the first group was 53.4±2.2 years ($p \leq 0.01$). There were 4 women (16.0%) and 21 men (84.0%). The results of the examination of patients in group I were compared with group II (control group) consisting of 10 people who had no signs of pathology in the IVC system and voluntarily agreed to undergo examination. The average age of patients in group II was 41.2±2.3 years ($p \leq 0.01$). There were 2 women (20.0%) and 8 men (80.0%).

Patients in group I were divided into the following subgroups depending on their previous treatment for thrombosis in the IVC system:

- Group I A – 8 (32.0%) patients who underwent open thromboectomy and incomplete mechanical cavaplication for IVC thrombosis;
- Group I B – 17 (68.0%) patients who received only conservative treatment for IVC thrombosis.

Laboratory and instrumental methods were used to examine patients, including ultrasound dopplerography and ultrasound duplex scanning, X-ray contrast phlebography, multispiral computed tomography (MSCT) with intravenous contrast, and radioisotope phleboscintigraphy.

Venous blood flow in the IVC was assessed in the immediate (up to 30 days after surgery), early (up to 1 year) and late (up to 10 years) postoperative periods.

Statistical processing of the study results was performed using Microsoft Excel 2010 computer software with the Statistica 5.0 for Windows application package. Comparisons of mean values and relative indicators were performed using the Mann-Whitney criteria and Pearson's parametric correlation analysis.

The study was conducted in accordance with the provisions of the World Medical Association's Declaration of Helsinki 'Ethical Principles for Medical Research Involving Human Subjects' (revised in 2008) and approved by the Bioethics Commission of the Medical Faculty of Uzhhorod National University. No violations of ethical standards were found during the research. All patients signed an informed consent form for participation in the research.

RESULTS

IVC thromboses were divided into tumour and non-tumour thromboses depending on the underlying cause. The cause of tumour thrombosis was the spread of implant thrombosis from the renal vein into the lumen of the IVC. Non-tumour IVC thrombosis was caused by the spread of thrombotic masses from the iliac venous segment towards the path of least resistance – in the direction of the right atrium. At the same time, vigorous blood flow through the renal and hepatic veins slowed the growth of thrombotic occlusion in the proximal direction, which was confirmed by a large number of infrarenal localisations of the apex of thrombotic masses among the examined patients in group I, in particular, infrarenal localisation of the apex of thrombotic masses was found in 18 (72.0%) of 25 patients. However, in 2 cases of non-tumour thrombosis and in all cases (n=5) of implantation thrombosis, thrombotic masses still caused a noticeable obstruction of blood flow from the renal and hepatic veins and contributed to the spread of the pathological process to the suprarenal section of the IVC.

In group I A, open thromboectomy from the IVC lumen and incomplete mechanical cavaplication were performed in 8 (32.0%) patients, including those with non-tumour (n=3) and tumour (n=5) thrombosis of the caval venous segment. When choosing the site for cavaplication, the infrarenal segment of the IVC directly under the renal veins prevailed in 7 (87.5%) patients, and in 1 (12.5%) patient, cavaplication was performed above the renal veins. The indications for cavaplication above the renal vein were old organised parietal layers in the infrarenal segment of the IVC, which could not be removed. A distinctive feature of tumour implantation thrombosis is that the 'thrombotic' masses in the IVC are morphologically tumour tissue, and anticoagulant therapy has no therapeutic effect. Therefore, the only way to combat IVC implantation thrombosis is thromboectomy from the IVC.

Group I B included 17 (68.0%) patients with diagnosed non-tumour IVC thrombosis who underwent conservative treatment.

Conservative treatment of IVC thrombosis in group I involved the administration of heparin at the maximum daily dose subcutaneously or intravenously continuously using an infusion pump, infusion-spasmolytic therapy, phlebotropic agents, and elastic hosiery. To prevent heparin-induced thrombocytopenia, the duration of heparin therapy averaged 9.3 ± 0.7 days. Subsequently, patients were switched to new oral anticoagulants at therapeutic doses. The duration of their administration, depending on the cause of thrombosis and the intensity of recanalisation of the veins of the IVC basin, ranged

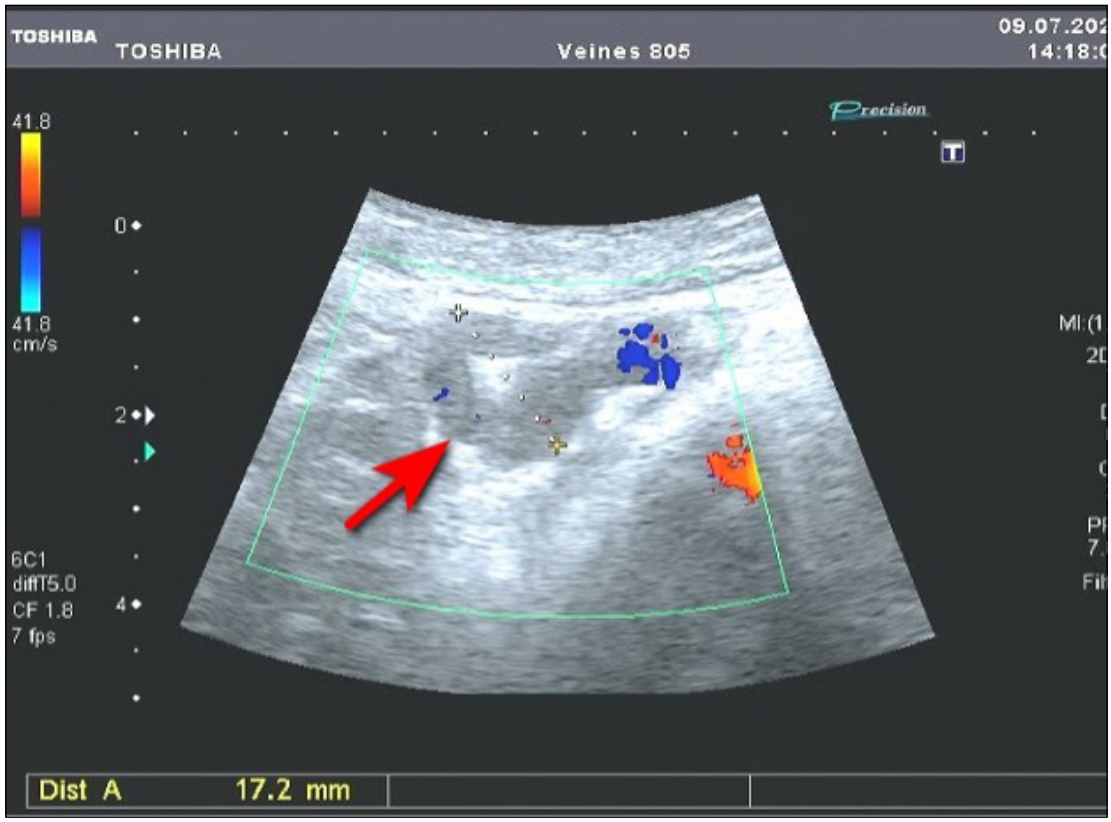

Fig. 1. Ultrasound examination: IVC in the plication area 6 months after surgery: IVC lumen is intact (the arrow indicates the plication area with two formed channels)
Picture taken by the authors

Table 1. Central haemodynamic parameters in the examined patients

Indicators*	I A group			I B group		II group
	Before surgery	After surgery		Before treatment	36 – 60 months	
		12 months	36 – 60 months			
Left ventricular ejection fraction, %	60,50±3,72	65,50±3,52	56,30±5,27	61,23±4,55	57,21±3,82	57,43±2,55
Minute blood flow, l/min	4,83±2,32	5,71±2,10	5,43±3,22	4,79±1,91	5,23±2,78	5,54±2,35
Stroke volume, ml/m²	49,97±3,15	46,54±2,10	55,15±2,14	47,56±1,27	57,21±2,32	60,12±2,34
Cardiac index, l/(min×m²)	3,41±0,14	3,50±0,30	3,61±0,60	3,24±0,20	3,56±0,71	3,67±0,56
Systolic volume, ml	65,6±2,32	67,3±1,32	66,2±1,41	61,1±1,22	66,2±1,23	67,2±1,42
End-systolic volume index of the right ventricle, ml/m²	21,45±1,2	24,52±1,1	25,31±1,1	21,32±1,5	27,23±1,3	27,51±1,6
End-diastolic volume index of the right ventricle, ml/m²	60,77±2,2	61,50±1,7	65,21±1,3	61,02±1,7	67,12±0,9	67,23±1,1
Linear blood flow velocity, cm/sec	3,06±1,5	6,31±1,8	7,13±1,6	2,98±1,7	7,02±1,2	9,01±1,4
Blood flow volume, ml/sec	2,63±0,5	4,6±0,7	5,31±1,2	3,01±0,8	5,11±1,4	5,21±1,1
Average transport time of RP, sec	12,7±0,5	7,9±0,3	5,1±0,4	13,3±0,7	5,2±0,5	4,1±0,3
IVC pressure (mm Hg)	7,73±1,52	5,42±1,73	4,9±1,13	8,21±1,56	6,1±3,21	5,7±1,22
IVC collapse index, %	11,2±2,9	15,4±2,1	35,5±2,0	10,3±1,7	27,2±1,5	36,7±2,5
IVC diameter (infrarenal), mm	24,7±2,3	22,3±1,4	21,5±1,2	25,1±2,2	21,9±1,6	20,9±2,1
Diameter of the left gonadal vein, mm	11,2±2,1	7,5±1,9	6,7±2,3	10,9±2,1	7,4±1,8	3,4±1,2

* - P ≤0,05
Source: compiled by the authors of this study

from 4–6 months to lifelong administration. The average duration of oral anticoagulants in therapeutic doses at the end of the study was 14.7±3.5 months.

In group I A, after incomplete device-based cavaplication, no cases of PE were observed in any of the 8 patients in the early and late postoperative period. It should be noted that the absence of a foreign body in the lumen of the IVC contributed to early recanalisation and improved outflow from the IVC. Staple eruption and gradual restoration of the IVC lumen were observed after 10.2 ± 2.7 months. This is the main advantage of direct methods of PE prevention over indirect methods. The characteristic ultrasound image is shown below (Fig. 1).

To determine individual indicators of central haemodynamics in patients with IVC thrombosis, echocardiography, ultrasound examination and radioisotope phleboscintigraphy were performed in the preoperative and postoperative periods. In all patients of group I, during the manifestation of IVC thrombosis, changes in central haemodynamic parameters were noted, indicating the absence of adequate venous return to the heart (Table 1).

Thus, in patients of groups I A and I B, before the start of surgical or conservative treatment of IVC thrombosis, due to its thrombotic occlusion, an increase in the diameter of the IVC to 24.7±2.3 mm ($p \leq 0.05$) and 25.1±2.2 mm ($p \leq 0.05$), respectively, compared with the control group of 20.9±2.1 mm ($p \leq 0.05$). At the same time, a sharp decrease in the IVC collapse index was observed, in particular 11.2±2.9% ($p \leq 0.05$) in group I A and 10.3±1.7% ($p \leq 0.05$) in group I B compared with the norm of 36.7±2.5% ($p \leq 0.05$). One of the compensatory mechanisms for activating collateral blood flow in IVC thrombosis was an increase in the diameter of the left gonadal vein to 11.2±2.1 mm ($p \leq 0.05$) in group I A and 10.9±2.1 mm ($p \leq 0.05$) in group I B compared with the norm of 3.4±1.2 mm ($p \leq 0.05$). In addition, ultrasound examination revealed compensatory dilation of the pelvic veins, which also indicated an increase in the volume of collateral venous blood flow in the IVC basin. At the same time, the pressure in the IVC practically did not exceed the upper limit of the norm and amounted to 7.73±1.52 mm Hg ($p \leq 0.05$) in group I A and 8.21±1.56 mm Hg ($p \leq 0.05$) in group I B.

Due to thrombotic occlusion of the IVC, a decrease in the main blood flow parameters was noted, in particular, the linear and volumetric blood flow velocity during ultrasound examination and the average radio-pharmaceutical (RP) transport time during radioisotope phleboscintigraphy. The linear blood flow velocity in the IVC above the apex of the thrombotic masses was reduced by almost three times compared to the control group, in particular 3.06±1.5 cm/sec ($p \leq 0.05$) in group I A and 2.98±1.7 cm/sec ($p \leq 0.05$) in group I B at 9.01±1.4 cm/sec ($p \leq 0.05$) in the control group. The volumetric blood flow velocity in the IVC above the apex of the thrombotic masses was reduced by half compared with the control group, namely 2.63±0.5 ml/sec ($p \leq 0.05$) in group IA and 3.01±0.8 ml/sec ($p \leq 0.05$) in group IB, compared to 5.21±1.1 ml/sec ($p \leq 0.05$) in the control group.

The average RP transport time due to thrombotic occlusion of the IVC increased threefold compared to the control group, in particular 12.7±0.5 sec ($p \leq 0.05$) in group I A and 13.3±0.7 sec ($p \leq 0.05$) in group I B compared to 4.1±0.3 sec ($p \leq 0.05$) in the control group.

During echocardiography, no significant changes in cardiac performance indicators such as left ventricular ejection fraction, minute blood flow volume, stroke and systolic volume, cardiac index, end-systolic and end-diastolic volumes of the right ventricle were observed. This is associated with the enormous compensatory capacity of collateral blood flow in the IVC basin.

Restoration of IVC lumen at the site of its thrombotic occlusion in patients of group I A after open thromboectomy allowed rapid improvement of central haemodynamic parameters. Return of blood flow parameters to normal values was observed only after staples were removed at the site of cavaplication and complete restoration of IVC lumen. Restoration of IVC lumen at the site of cavaplication in patients of group I A was noted during ultrasound examination after 1–1.5 years (14.3±2.2 months ($p \leq 0.01$)). At the same time, thickening and rigidity of the IVC wall were noted in the cavaplication area (Fig. 2).

After thromboectomy, in the early postoperative period, there was also an improvement in regional haemodynamic parameters, in particular a decrease in the average RP transport time during radioisotope phleboscintigraphy from 12.7±0.5 ($p \leq 0.05$) seconds to 7.9±0.3 seconds ($p \leq 0.05$), and after restoration of IVC patency at the site of cavaplication, the mean RP transport time decreased to 5.1±0.4 with values of 4.1±0.3 sec ($p \leq 0.05$) in the control group. At the same time, at the site of cavaplication, with preserved IVC patency on phleboscintigrams, a slight delay of the RP was observed (Fig. 3). There were also no areas of venous stasis and dilated collateral veins (Fig. 4). The images of the IVC and contrasted iliac veins are clear, homogeneous, with no collateral outflow. The patency of the IVC and recanalisation of the distal bed, with preserved volumetric blood flow, allow us to predict satisfactory functional results of treatment in the long term.

Thus, the average RP transit time during IVC restoration was close to that of the control group (Fig. 5). Radioisotope phleboscintigraphy data confirmed the

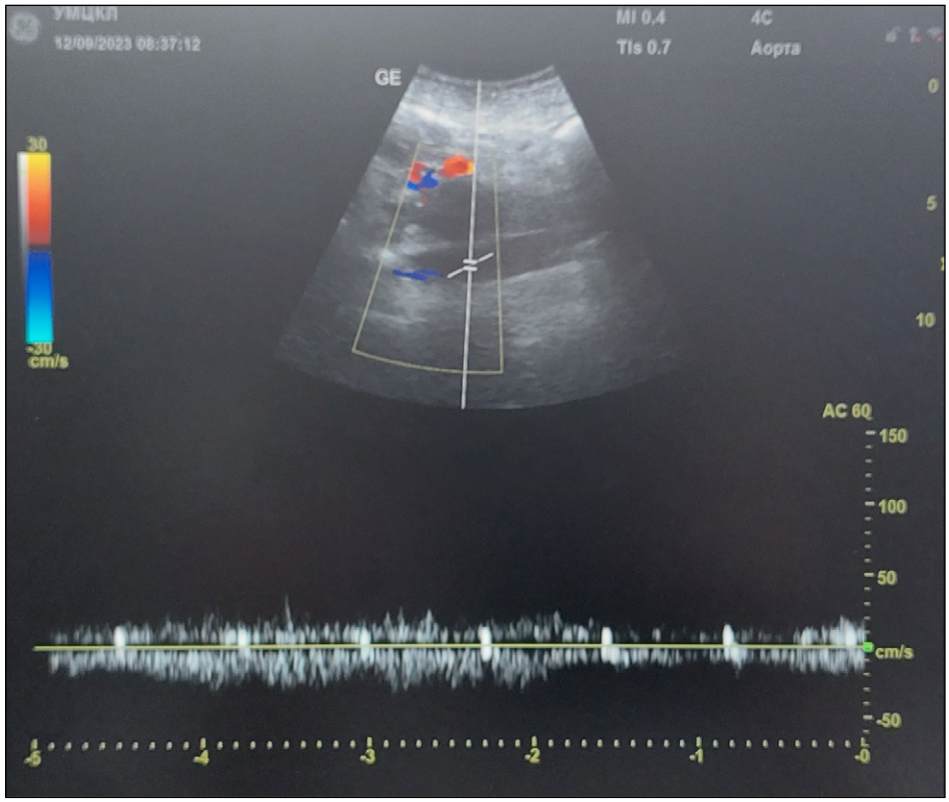

Fig. 2. Ultrasound examination: venous blood flow spectrum in the IVC 1.5 years after cavaplication is not disturbed
Picture taken by the authors

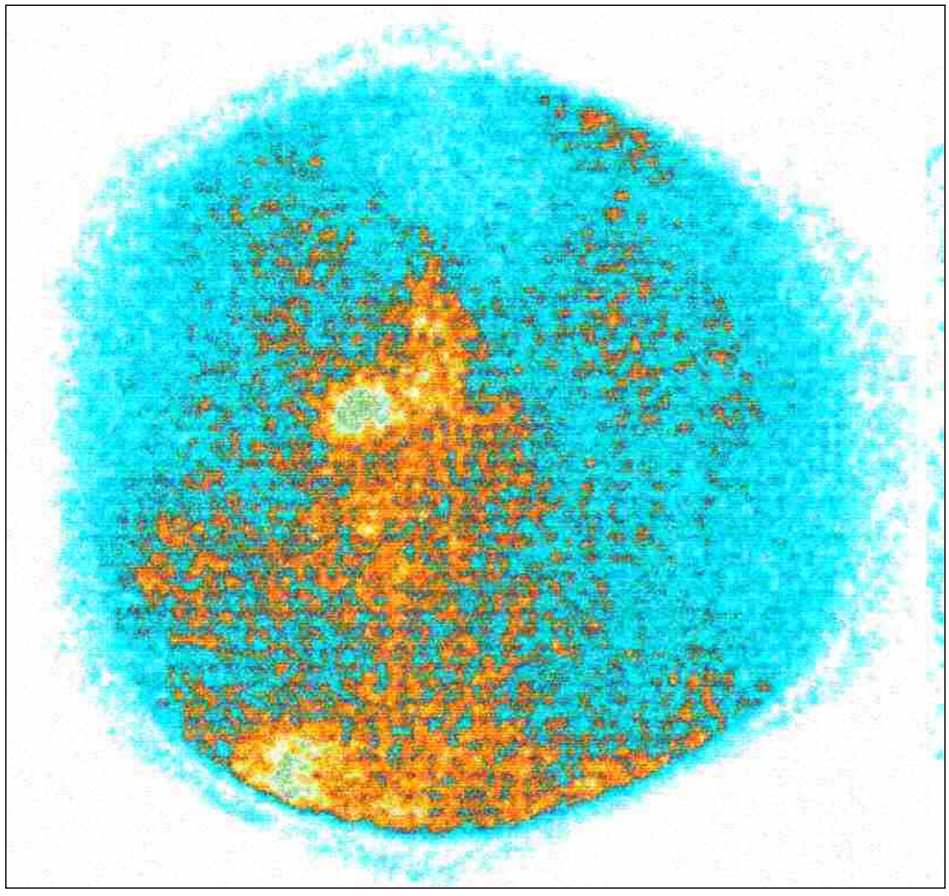

Fig. 3. Radioisotope phleboscintigraphy in a horizontal position: patent iliac-caval segment, slight delay in the evacuation of the radiopharmaceutical at the level of cavaplication
Picture taken by the authors

results of ultrasound and X-ray contrast studies in the long-term follow-up period (Fig. 6).

In group I, a gradual recovery of central and regional haemodynamic parameters was observed. This situation was associated with the processes of recanalisation of the IVC lumen against the background of prolonged anticoagulant therapy. The average recanalisation time with conservative treatment in group I B was 4.74±2.35

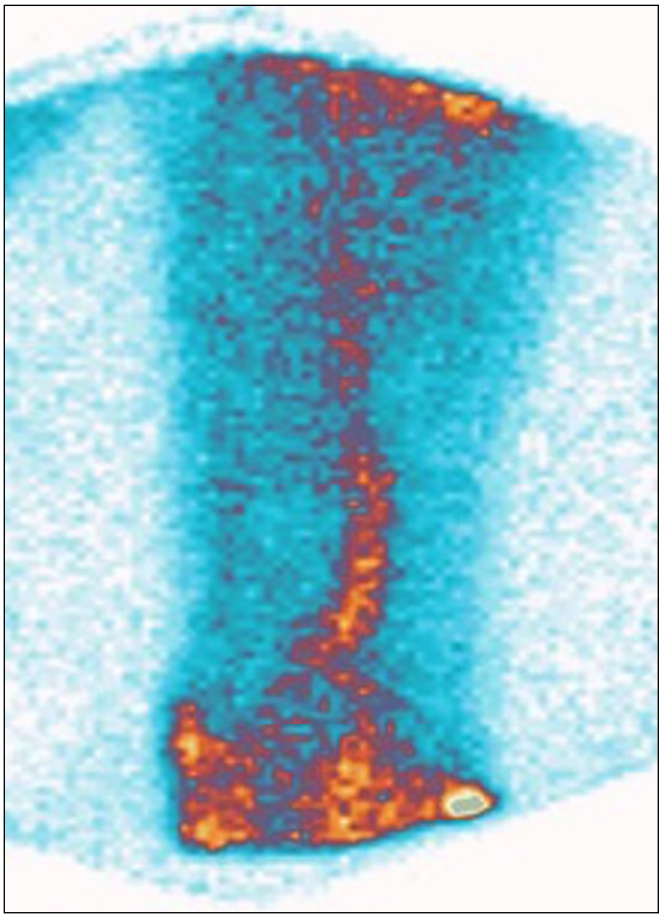

Fig. 4. Radioisotope phleboscintigraphy in a horizontal position: patent IVC after cavaplication
Picture taken by the authors

months (p≤0.01). Central haemodynamic parameters in group I B differed from blood flow parameters in patients in groups I A and II, which was caused by incomplete recanalisation and the occurrence of post-thrombotic changes in the IVC walls. Thus, the IVC diameter in patients of the I B group in the long term was 21.9±1.6 mm (p≤0.05) compared to 21.5±1.2 mm (p≤0.05) in the I A group and 20.9±2.1 mm (p≤0.05) in the control group. The IVC collapse index increased gradually and amounted to 27.2±1.5% (p≤0.05) and was still significantly lower than in group I A (35.5±2.0%, p≤0.05) and the control group (36.7±2.5%, p≤0.05), which was also associated with post-thrombotic changes in the IVC wall. The diameter of the left gonadal vein, even after IVC recanalisation, decreased insignificantly, which was due to its prolonged participation in the compensatory mechanisms of collateral venous blood flow. We observed preservation of varicose transformation of the left gonadal vein, in particular, its diameter was 7.4±1.8 mm (p≤0.05) versus 6.7±2.3 mm (p≤0.05) in group I A and 3.4±1.2 mm (p≤0.05) in the control group. In addition, during ultrasound examination, varicose venous plexuses of the small pelvis and retroperitoneal space were visualised, and clinically, varicose superficial veins of the abdominal wall were detected in patients (Fig. 7).

IVC pressure in patients of group I B in the remote period was slightly higher (6.1±3.21 mm Hg (p≤0.05)) compared to similar indicators in group I A (4.9±1.13 mm Hg (p≤0.05)) and the control group (5.7±1.22 mm Hg (p≤0.05)). The linear blood flow velocity in patients of the I B group after IVC recanalisation was 7.02±1.2 cm/sec (p≤0.05) and practically did not differ from the indicators of the I A group (7.13±1.6 cm/sec (p≤0.05)), although it was lower than that of the control group (9.01±1.4 cm/sec (p≤0.05)). At the same time, the volumetric blood flow velocity in all groups was practically the same, in particular 5.11±1.4 ml/sec (p≤0.05) in patients of the I B group compared to 5.31±1.2 ml/sec (p≤0.05) in patients of the I A group and 5.21±1.1 ml/sec (p≤0.05) in the control group. The average RP transport time during radioisotope phleboscintigraphy in groups I B (5.2±0.5 sec (p≤0.05)) and I A (5.1±0.4 sec

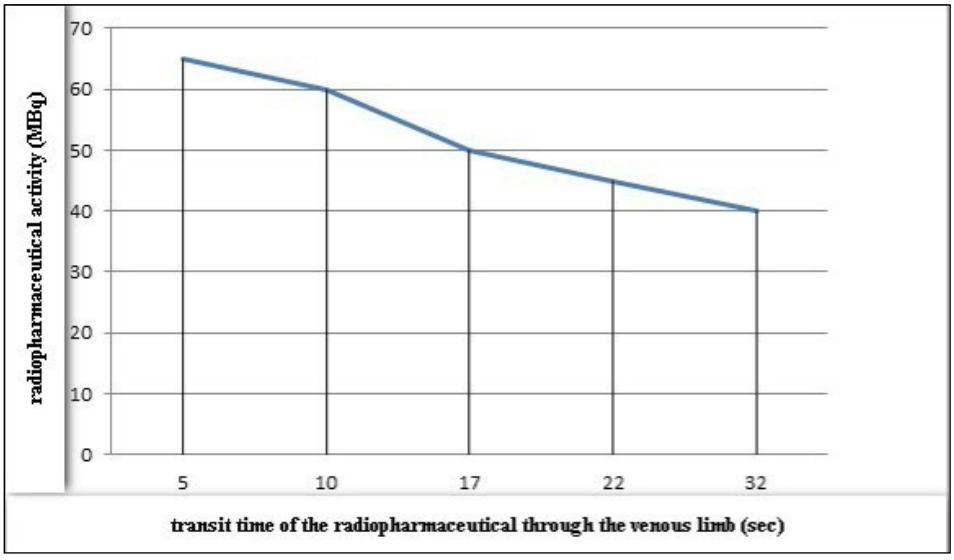

Fig. 5. Graph showing the dependence of the transport time of the radiopharmaceutical Tc-99 (70–40 MBq) on IVC 1 year after cavaplication
Picture taken by the authors

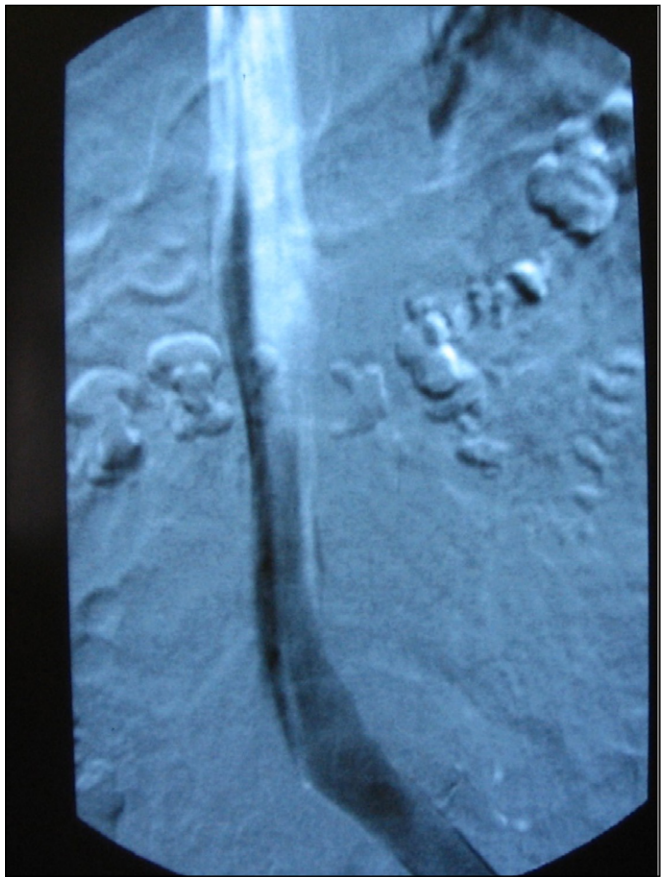

Fig. 6. X-ray contrast cavography: preservation of the patency of the inferior vena cava 3 years after plication
Picture taken by the authors

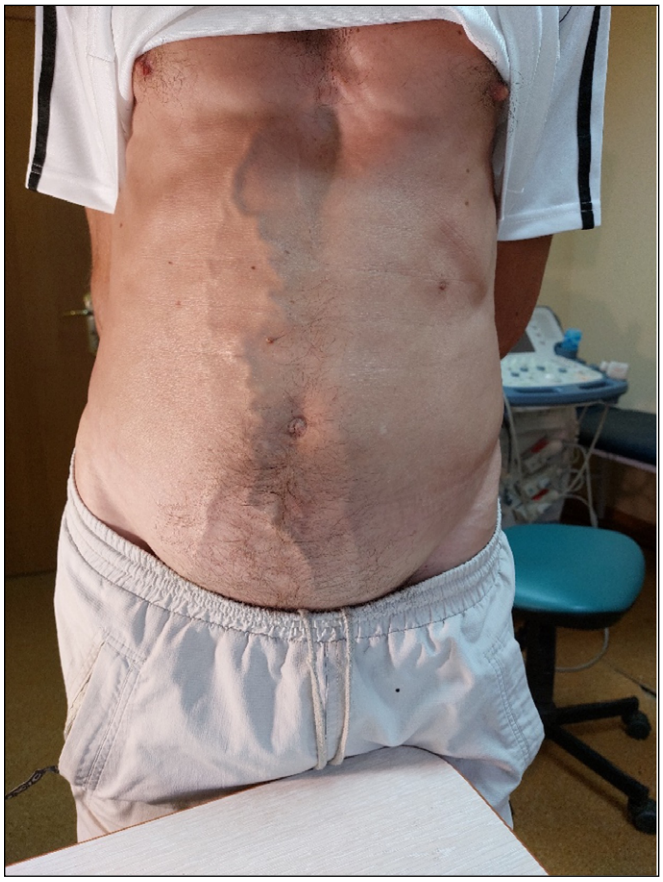

Fig. 7. Patient X: varicose veins of the anterior abdominal wall in post-thrombotic syndrome of the inferior vena cava
Picture taken by the authors

(p≤0.05)) increased to almost the same values, although it was lower than that of the control group (4.1±0.3 sec (p≤0.05)).

During echocardiography, no significant changes in cardiac function were observed in patients of group I B compared to patients of groups I A and II.

DISCUSSION

Thrombosis of the IVC significantly remodels venous return to the heart, changing central haemodynamic parameters during ultrasound examination of the heart. Compensation of venous return to the heart from the lower half of the body in IVC thrombosis is provided by venous collaterals, in particular the lumbar venous plexus, ascending lumbar, gonadal, diaphragmatic, unpaired and semi-unpaired veins [17, 18]. At the same time, the principles of diagnosis and treatment of DVT are mainly based on morphological criteria, and changes in peripheral and central venous haemodynamics are practically not assessed [19]. According to studies by Antignani P.L. et al. (2020), venous pressure in individuals with chronic lower limb vein disease is significantly higher in superficial and deep veins compared to healthy individuals [20]. In post-thrombotic syndrome, pressure in superficial and deep veins reaches the highest possible values, causing severe pain [20]. At the same time, deep venous hypertension is more pronounced than superficial hypertension, with pressure rising to 130–140 mm Hg [20]. The authors note that venous pressure values do not always correspond to the severity of the disease [20].

An important parameter of central haemodynamics is central venous pressure or right atrial pressure, which requires mainly invasive examination [21]. At the same time, the invasive method of measuring central venous pressure is dangerous due to catheter-induced thrombosis, arrhythmia and infectious complications [21]. Therefore, in clinical practice, preference is given to non-invasive methods of assessing central venous pressure [21]. According to the recommendations of the American Society of Echocardiography (2015), the maximum diameter of the IVC should be measured along its axis approximately 1–2 cm caudal to the junction of the IVC and the right atrial appendage [22]. Ciozda W. et al. (2015), based on an analysis of 21 prospective studies involving 1,430 patients, found a direct relationship between central venous pressure and IVC diameter [23].

The difficulty of measuring central venous haemodynamic parameters in vivo led to the study of haemodynamics in an experimental model of the inferior vena cava, which revealed peak blood flow velocities ranging from 4.9 cm/s to 27 cm/s at rest and during physical exertion, respectively [24]. Raju S. et al. (2020) proved that there is no evidence of inspiratory compression of the central veins of the abdominal cavity and refuted the haemodynamic theory of the abdominal pump [25]. Instead, with an increase in the volume of inspiration, an increase in the diameter of the IVC by 32% and a simultaneous decrease in pressure in it were observed [25].

Based on 7 studies involving 395 spontaneously breathing patients, the concept of the IVC collapse index (sensitivity 71%; specificity 81%) was proposed, which involves the ratio of the diameter of the IVC during the respiratory or ventilation cycle during ultrasound examination. specificity 81%), which is the ratio of the IVC diameter during the respiratory or ventilation cycle during ultrasound examination. Similar data were obtained in 9 studies involving 284 patients on artificial ventilation, where the sensitivity and specificity of the IVC collapse index were 75% and 82%, respectively. The IVC collapse index indirectly indicates right atrial pressure and circulating blood volume [26–28]. At the same time, other researchers believe that ultrasound examination of the IVC may be limited by factors affecting the diameter or collapse of the IVC and should take into account the clinical situation [29].

We have not found any studies in the available literature that evaluate changes in venous haemodynamics depending on the treatment of venous thrombosis in the IVC. However, our data suggest that open thromboectomy from the lumen of the IVC combined with incomplete mechanical cavaplication in floating thromboses not only restores the lumen of the venous trunk, prevent the development of PE, but also quickly normalise the parameters of central and regional venous haemodynamics. Analysis of blood flow parameters in the early and late postoperative periods allows us to conclude that partial mechanical cavaplication does not impair venous return to the heart, and open thromboectomy from the IVC lumen prevents the development of post-thrombotic changes in the walls of the venous trunk. Conservative treatment of IVC thrombosis significantly accelerates recanalisation processes compared to the natural course of thrombosis, but during this time, natural venous collaterals arise, which ensure venous outflow from the lower half of the body and remain active even after restoration of the IVC lumen.

CONCLUSIONS

1. Open thromboectomy from the inferior vena cava allows restoration of the lumen and ensures effective main venous blood flow, and is the only effective treatment for implant thrombosis. The introduction of incomplete mechanical cavaplication after thromboectomy from the lumen of the inferior vena cava prevents venous thromboembolic complications.
2. The advantage of incomplete mechanical cavaplication is the independent gradual restoration of the lumen of the inferior vena cava within 14.3±2.2 months ($p \leq 0.01$).
3. Cavaplication not only does not disturb central and regional venous haemodynamics, but also leads to an increase in linear and volumetric blood flow velocities in the inferior vena cava, which prevents recurrence of thrombosis.
4. Open thromboectomy from the lumen of the inferior vena cava, compared with conservative treatment, prevents the development of post-thrombotic syndrome with chronic venous insufficiency and prevent the development of venous collaterals with subsequent remodelling of venous blood flow in the lower hollow vein basin.

REFERENCES

1. Del Pozo MM, Asenjo MM, Moreno FAl et al. Long-term monitoring and treatment of venous thromboembolic disease: recommendations of the Thromboembolic Disease Group of the Spanish Society of Internal Medicine 2024. Rev Clin Esp 2024;224(10):652-63. doi:10.1016/j.rceng.2024.10.004. DOI
2. Shah NG, Wible BC, Paulisin JA et al. Management of inferior vena cava thrombosis with the FlowTriever and ClotTriever systems. J Vasc Surg 2021;9(3):615-20. doi:10.1016/j.jvsv.2020.09.008. DOI
3. Sildiroglu O, Ozer H, Turba CU. Management of the thrombosed filter-bearing inferior vena cava. Semin Intervent Radiol. 2012;29:57-63. doi:10.1055/s-0032-1302453. DOI
4. Girard P, Meyer G, Parent F et al. Medical literature, vena cava filters and evidence of efficacy. A descriptive review. Thromb Haemost. 2014;111:761-9. doi:10.1160/TH13-07-0601. DOI
5. Klein-Weigel PF, Elitok S, Ruttloff A et al. Inferior vena cava-syndrome. Vasa. 2021;50(4):250-64. doi:10.1024/0301-1526/a000919. DOI
6. Alkhouli M, Morad M, Narins CR et al. Inferior vena cava thrombosis. JACC Cardiovasc Interv. 2016;9(7):629–43. doi:10.1016/j.jcin.2015.12/268. DOI

7. Shi W, Dowell JD. Etiology and treatment of acute inferior vena cava thrombosis. Thromb Res. 2017;149:9–16. doi:10.1016/j.thromres.2016.07.010. DOI
8. McAree BJ, O'Donnell ME, Fitzmaurice GJ et al. Inferior vena cava thrombosis: a review of current practice. Vasc Med. 2013;18:32–43. doi:10.1177/1358863X12471967. DOI
9. Qi Yu-H, Wang T, Wang J et al. Inferior vena cava filter reduces pulmonary embolism but does not increase deep venous thrombosis: A systematic review and metaanalysis. Asian J of Surg. 2022;45(1):592–3. doi:10.1016/j.asjsur.2021.10.046. DOI
10. King RW, Wooster MD, Veeraswamy RK et al. Contemporary rates of inferior vena cava filter thrombosis and risk factors. J Vasc Surg. 2022;10(2):313-24. doi:10.1016/j.jvsv.2021.07.016. DOI
11. Van Ha TG. Complications of inferior vena caval filters. Semin Intervent Radiol. 2006;23:150-155. doi: 10.1055/s-2006-941445. DOI
12. Kalva SP. Complications of inferior vena cava filters. Cardiovasc Diagn Ther. 2016;6:632-41. doi:10.21037/cdt.2016.09.08. DOI
13. Friedman T, Quencer KB, Kishore SA et al. Malignant venous obstruction: superior vena cava syndrome and beyond. Semin Intervent Radiol. 2017;34:398–408. doi:10.1055/s-0037-1608863. DOI
14. Teter K, Schrem E, Ranganath N et al. Presentation and Management of Inferior Vena Cava Thrombosis. Ann Vasc Surg. 2019;56:17-23. doi:10.1016/j.avsg.2018.08.082. DOI
15. Popovych YaM, Korsak VV, Boldizhar PO et al. Khirurhichna profilaktyka tromboembolichnykh uskladnen pry transfastsialnomu trombozi [Surgical Prevention of Thromboembolic Complications in Transfascial Thrombosis]. Ukrainskyi zhurnal sertsevo-sudynnoi khirurhii. 2023;31(1):66–73. doi:10.30702/ujcvs/23.31(01)/PK002-6673. (Ukranian) DOI
16. Popovich YaM, Rosul MV, Sich PR et al. Thrombolysis in Pulmonary Embolism Treatment. Wiad Lek. 2023;71(3):604–9. doi:10.36740/WLek202303123. DOI
17. Meyer S, Poryo M. Inferior vena cava thrombosis-rope ladder sign. Wien Med Wochenschr. 2023;173:159–160. doi:10.1007/s10354-021-00886-y. DOI
18. Popovych YaM. Otsinka stanu hemodynamiky u patsiientiv z trombozamy systemy nyzhnoi porozhnystoi veny zalezhno vid sposobu likuvannia [Estimation of hemodynamic parameters in patients with trombosis inferior vena cava system depends on the treatment method]. Khirurhiia Ukrainy 2018;1(65):55–61. doi:10.30978/SU2018155. (Ukranian) DOI
19. Franceschi C. Measurement of venous pressure by Doppler: food for thought. Acta Phlebologica. 2022;23(3):95-6. doi:10.23736/S1593-232X.22.00545-8. DOI
20. Antignani PL, Peruzzi G, Spina T. The measurement of venous pressure by Doppler: is it a hemodynamic evaluation? J of Theoret App Vascul Res. 2020;5(2). doi:10.24019/jtavr.114. DOI
21. Beigel R, Cercek B, Luo H et al. Noninvasive evaluation of right atrial pressure. J Am Soc Echocardiogr. 2013;26:1033–42. doi:10.1016/10.1016/j.echo.2013.06.004. DOI
22. Lang RM, Badano LP, Mor-Avi V et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28:1–39. doi:10.1016/j.echo.2014.10.003. DOI
23. Ciozda W, Kedan I, Kehl DW et al. The efficacy of sonographic measurement of inferior vena cava diameter as an estimate of central venous pressure. Cardiovasc Ultrasound 2015;14(33). doi:10.1186/s12947-016-0076-1. DOI
24. Gallagher MB, Aycock KI, Craven BA et al. Manning Steady Flow in a Patient-Averaged Inferior Vena Cava–Part I: Particle Image Velocimetry Measurements at Rest and Exercise Conditions. Cardiovasc Eng Technol. 2018;9(4):641–53. doi:10.1007/s13239-018-00390-2. DOI
25. Raju S, Walker W, Noel C et al. An alternative explanation for the phasic variations in venous flow. J Vasc Surg Venous Lymphat Disord. 2021;9(4):977-986. doi:10.1016/j.jvsv.2020.11.018. DOI
26. Si X, Cao D, Xu H, Guan X. Meta-analysis of ventilated versus spontaneously breathing patients in predicting fluid responsiveness by inferior vena cava variation. Int J Clin Med. 2018;9:760-777. doi:10.4236/ijcm.2018.910063. DOI
27. Preau S, Bortolotti P, Colling D et al. Diagnostic accuracy of the inferior vena cava collapsibility to predict fluid responsiveness in spontaneously breathing patients with Sepsis and acute Circulatory failure. Crit Care Med. 2017;45(3):e290-e297. doi:10.4236/ijcm.2018.910063. DOI
28. Corl KA, George NR, Romanoff J et al. Inferior vena cava collapsibility detects fluid responsiveness among spontaneously breathing critically-ill patients. J Crit Care. 2017;41:130-137. doi:10.1016/j.jcrc.2017.05.008. DOI
29. Kaptein MJ, Kaptein EM. Inferior Vena Cava Collapsibility Index: Clinical Validation and Application for Assessment of Relative Intravascular Volume. Adv Chronic Kidney Dis. 2021;28(3):218-226. doi:10.1053/j.ackd.2021.02.003. DOI

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CONFLICT OF INTEREST
The Author declare no conflict of interest

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