DOI: 10.15825/1995-1191-2020-1-59-71

AMBIGUOUS RESULTS OF BALLOON ANGIOPLASTY FOR CENTRAL VEIN STENOSIS IN HEMODIALYSIS PATIENTS WITH NATIVE ARTERIOVENOUS FISTULA

Z.B. Kardanakhishvili[1, 2], A.B. Zulkarnaev[3], B.V. Baykov[1, 4], V.A. Stepanov[3]

[1] War Veterans Hospital No. 2, Moscow, Russian Federation
[2] Zhadkevich City Clinical Hospital, Moscow, Russian Federation
[3] Vladimirsky Moscow Regional Research Clinical Institute, Moscow, Russian Federation
[4] Krasnogorsk City Hospital No. 1, Krasnogorsk, Russian Federation

Objective: to conduct comprehensive comparative analysis of the patency rate of native arteriovenous fistula (AVF) for central vein stenosis (CVS) after endovascular balloon angioplasty and palliative surgery. **Materials and methods.** The retrospective study included 80 patients with confirmed central vein stenosis: subclavian, brachiocephalic veins, inferior vena cava, or multiple lesions. The experimental group included 39 patients who underwent percutaneous balloon angioplasty. The control group included 41 patients who, for various reasons, did not do balloon angioplasty, but underwent palliative interventions: thrombectomy, proximalization of arteriovenous anastomosis, AVF blood flow-reducing surgical procedures. **Results.** Primary patency (time interval between the first intervention for CVS and the second intervention) in the experimental group was 61.5% [95% CI 44.5; 74.7] and 15.4% [95% CI 6.2; 28.3] at 6 and 12 months, respectively. In the control group, it was 39% [95% CI 24.3; 53.4] and 0% respectively. Hazard ratio (HR) 0.5337 [95% CI 0.3381; 0.8427], log-rank test p = 0.0011. No differences in functional primary patency (time interval between the start of using AVF and the first intervention for CVS) were found: 89.7% [95% CI 74.9; 96] and 30.8% [95% CI 17.3; 45.4] at 1 year and 3 years, respectively, in the experimental group, and 80.5% [95% CI 64.8; 89.7] and 24.4% [95% CI 12.7; 38.2] in the control group. There were no differences between the groups HR 0.7695 [95% CI 0.4952; 1.196], log-rank p = 0.2259. In the experimental group, strong negative correlation between primary patency and functional primary patency was detected: r = –0.627 [95% CI –0.787; –0.388], p < 0.0001. In the control group, no such correlation was found: r = 0.049 [95% CI –0.262; –0.351], p = 0.7599. Thus, the later CVS developed, the less effective balloon angioplasty was. Balloon angioplasty significantly increased duration of AVF use after first intervention for CVS (secondary patency): 84.6% [95% CI 68.9; 92.8], 66.7% [95% CI 49.6; 79.1] and 17.9% [95% CI 7.9; 31.3] at 6, 12 and 24 months, respectively in the experimental group. In the control group, it was 56.1% [95% CI 39.7; 69.6], 19.5% [95% CI 9.2; 32.7] and 0%. HR 0.4009 [95% CI 0.2481; 0.6477], log-rank p < 0.0001. Functional secondary patency (total duration of AVF use) was: 100%, 74.4% [95% CI 57.6; 85.3] and 12.8% [95% CI 4.7; 25.2] at 1, 3 and 5 years in the experimental group, and 95.1% [95% CI 81.9; 98.8], 36.6% [95% CI 22.3; 51] and 4.9% [95% CI 0.9; 14.5] in the control group. HR 0.5661 [95% CI 0.3598; 0.8906], log-rank p = 0.0067. **Conclusions.** 1. Central vein stenosis inevitably cuts vascular access from the ipsilateral side. 2. Balloon angioplasty allows to slightly prolong AVF use but it cannot radically change the long-term results of CVS treatment. 3. The outcome of balloon angioplasty greatly depends on the length of the period from the time the use of AVF started to the time CVS developed. 4. Multiple repeated balloon angioplasties are apparently justified in patients for whom creating a new vascular access might not be possible. 4. AVF volumetric blood flow velocity is an important factor determining the severity of CVS clinical manifestations and whether repeated surgical interventions are needed.

Keywords: central vein stenosis, arteriovenous fistula, hemodialysis vascular access, balloon angioplasty, percutaneous transluminal angioplasty, endovascular surgery.

INTRODUCTION

Vascular access is one of the key aspects in the survival of patients with chronic kidney disease (CKD) receiving treatment with long term hemodialysis (HD). From year to year, there has been a monotonous increase in the number of patients with stage 5 CKD. The rate of increase is gradually rising [1]. It is generally accepted that arteriovenous fistula (AVF) is the preferred vascular access for HD. Initiating HD with an AVF is associated with better survival compared to other types of vascular access [2–4].

Central vein stenosis (CVS) is one of the severe complications in patients on HD. It is known that CVS significantly increases the risk of loss of ipsilateral access. It

Corresponding author: Alexey Zulkarnaev. Address: 61/2, Schepkina str, block 6, Moscow, 129110, Russian Federation. Tel. (916) 705-98-99. E-mail: 7059899@gmail.com

also has many adverse manifestations from subclinical venous hypertension to superior vena cava syndrome [5]. CVS prevalence varies widely: from 2 to 40% [6–8]. An important aspect is the fact that CVS not only leads to loss of functioning vascular access, but also makes it impossible to create a new access from the ipsilateral side [6, 7]. This significantly reduces the "vascular resource" of formation of not only native AVF, but also of any type of vascular access.

Implantation of the central venous catheter (CVC) is a major etiological factor for CVS [9–11]. Despite the "fistula first" principle [12, 13], 21% of prevalent hemodialysis patients in the US were dialyzed with a CVC [14], in Europe – 28% [15], in Russia – 12% [16]. At the same time, the need for CVC is highest at the beginning of renal replacement therapy: 80% of HD patients in the USA [14] start dialyzing with a CVC, in Europe – 61% [15]. According to our data [17] (registry of CKD patients in the Moscow region) – 43% of patients in Moscow and Moscow Oblast start dialyzing with a CVC. Due to widespread high demand for CVCs, one cannot but hope for a spontaneous solution to the problem of CVS.

Endovascular surgery is one of the fastest growing areas of reconstructive vascular access surgery for HD. Despite the enthusiasm generated after the first reports on successful percutaneous balloon angioplasty in stenosis and recanalization of occluded central veins [18, 19], and the high probability of technical success (which, according to many authors, reaches 100% [20–22]), it was later established that long-term AVF patency is low [5, 23]. The revealed contradictions contributed to the rethinking of approaches to plastic surgery in central vein stenoses and occlusions, which, presumably, has not ended today. As experience was gained and clinical trials were completed, approaches to improving the outcomes of endovascular interventions were proposed. Thus, the use of stents [24] and stent grafts [25], drug-coated balloon catheters [26] and high-pressure balloon catheters [27] has been suggested. Such a variety of available methods of influencing the affected vein segment is compensated by the absence of specific indications that make it possible to choose an optimal method, which significantly compromises endovascular interventions. At the same time, percutaneous balloon angioplasty remains the most accessible method for restoring central vein patency in HD patients. We dedicated our research to analyzing the characteristics of the outcomes of primary angioplasty without stenting.

Objective: to conduct a comprehensive comparative analysis of the patency of native AVF for CVS after endovascular balloon angioplasty and palliative surgery.

MATERIALS AND METHODS

The study protocol was endorsed by a local ethics committee and approved by the academic council.

Patients

The retrospective study included 80 patients with confirmed CVS. The experimental group included 39 patients (48.75%) who underwent endovascular balloon angioplasty (BA). Thrombectomy was performed in case of thrombosis, which was supplemented by proximalization of arteriovenous anastomosis (AVA), if necessary. The control group included patients who, for various reasons, did not perform BA. Palliative surgeries were performed in this group: thrombectomy in case of thrombosis, which was supplemented by AVA proximalization if necessary, or by AVF blood flow-reducing surgical procedures by means of formation of a bandage from synthetic vascular prosthesis on the juxta-anastomotic segment of the "fistula" vein, if the indication for operation were clinical manifestations for venous hypertension in the limb. This group was composed of 41 patients (51.25%).

The main inclusion criteria were: above 18 years of age at the time of inclusion in the study, subclavian, jugular, and brachiocephalic vein stenosis, inferior vena cava, or their combination; AVF lasting for at least one month; availability of reliable information on anamnesis and catamnesis; loss of AVF. Patients who used stents, as well as patients who used synthetic vascular prostheses as vascular access, were excluded from the study (such observations were excluded from analysis).

In all patients, except for 5 (3 (7.7%) in the experimental group and 2 (4.9%) in the control group), the first AVF was created before the start of HD. However, a large proportion of patients initiated HD through CVC due to primary dysfunction: 25 (64.1%) in the experimental group and 28 (68.3%) in the control group. Prior to the first CVS intervention, patients underwent one to three interventions. Tunneled CVS was the preferred intervention. In both groups, the need for CVC was high. The main indicators in the groups are summarized in Table. To evaluate the comorbid background, the CIRS (Cumulative Illness Rating Scale) scale [28] in the Miller modification [29] was used as the most convenient for retrospective analysis in our center. When analyzing CKD causes, the "systemic processes" group included patients with vasculitis, myeloma, HIV infection, patients with kidney neoplasm (some of them are renoprival), patients who underwent chemotherapy, having a long history of drug addiction, etc.

In 23 patients of the experimental group and 26 patients of the control group, isolated subclavian, brachiocephalic or superior vena cava stenosis was revealed. In 16 patients of the experimental group and 15 patients in the control group, stenosis of one of these veins was combined with jugular vein stenosis. Central vein stenosis was confirmed by angiography or ultrasound. Moreover, in patients from the control group, stenosis in some cases

Table

Characteristics of the groups

	Experimental group (n = 39)	Control group (n = 41)	Significance of difference
Age (years)	45 [39.25; 50], 23 to 59[1]	47 [41; 55], 26 to 71[1]	p = 0.3999
Gender (M/F)	43.6%/56.4% (17/22)	41.5%/58.5% (17/24)	p = 0.8475
Comorbidity, CIRS scores	14.5 [12; 19.75], 7 to 26[1]	18 [12; 23], 7 to 29[1]	p = 0.0894
Cause of CKD			
Polycystic kidney disease	25.6% (10)	22% (9)	p = 0.993
Pyelonephritis	12.8% (5)	12.2% (5)	
Glomerulonephritis	15.4% (6)	17.1% (7)	
Diabetes	28.2% (11)	31.7% (13)	
System processes	17.9% (7)	17.1% (7)	
Time interval between AVF formation and its use (months)	4 [3; 4.6], 0.7 to 7[1]	3 [3; 4], 2 to 7[1]	p = 0.43
Time interval between the start of using AVF and the first intervention for CVS (months)	29 [18.5; 40.5], 6 to 54[1]	25 [16; 36], 4 to 51[1]	p = 0.2858
Need for reconstructive interventions before using AVF	3.704 [2.79; 4.82][2]	3.841 [2.917; 4.965][2]	0.964 [0.665; 1.396][3] p = 0.845
Need for reconstructive interventions from the start of using AVF to the first intervention for CVS	2.263 [1.478; 3.316][4]	2.241 [1.435; 3.334][4]	1.01 [0.577; 1.773][3] p = 0.9742
Number of CVCs before the first intervention for CVS	3 [2; 5], 0 to 7[1]	3 [2; 5], 0 to 8[1]	p = 0.763
Need for CVC before the first intervention for CVS	1.443 [1.205; 1.713][2]	1.298 [1.091; 1.532][2]	1.112 [0.875; 1.412][3] p = 0.3859
Number of catheters in relation to catheterization duration	4.72 [4.944; 5.605][5]	4.796 [4.032; 5.663][5]	0.984 [0.774; 1.25][3] p = 0.897
Average duration of use of one CVC (months)	1.4 [1.18; 1.8], 0.7 to 5.7[1]	1.3 [1.03; 1.98], 0.6 to 5.6[1]	p = 0.753
Stenosis localization:	(percentage of 39)	(percentage of 41)	
left subclavian vein	46.2% (18)	51.2% (21)	p = 0.9915
right subclavian vein	28.2% (11)	24.4% (10)	
left internal jugular vein	17.9% (7)	14.6% (6)	
right internal jugular vein	23.1% (9)	22% (9)	
left brachiocephalic vein	12.8% (5)	12.2% (5)	
right brachiocephalic vein	7.7% (3)	9.8% (4)	
superior vena cava	5.1% (2)	2.4% (1)	
Need for open reconstructive interventions after the first intervention for CVS	0.374 [0.24; 0.556][2]	2.451 [1.963; 3.023][2]	0.153 [0.095; 0.237][3] p < 0.0001
Need for balloon angioplasty	1.137 [0.89134 1.43][2]	–	–
General need for surgical interventions after the first intervention for CVS	1.511 [1.225; 1.843][2]	2.451 [1.963; 3.023][2]	0.617 [0.461; 0.825][3] p = 0.0011

Note. [1] Median, interquartile range. [2] Number of operations per 10 patient-months and 95% confidence interval. [3] Incidence rate ratio (intensity of occurrence of events) and 95% confidence interval. [4] Operations per 100 patient-months and 95% confidence interval. [5] Number of CVCs per 100 catheter days and 95% confidence interval.

was revealed during angiographic examination performed in connection with CVC implantation difficulties.

Estimated indicators

In accordance with the latest clinical recommendations [30], we evaluated the following indicators:

– Primary patency – the time interval between the first intervention for CVS and the first repeated surgical intervention (eventless survival of vascular access from the moment of first intervention for CVS);
– Assisted primary patency – the time interval between the first intervention for CVS and the first AVF thrombosis, including surgical open or endovascular interventions to maintain its function (non-occlusive vascular access survival from the moment of the first intervention for CVS);
– Secondary patency – the time interval between the first intervention for CVS and complete cessation of the use of AVF, including all surgical interventions.
– Functional primary patency – the time interval between the start of AVF and the first surgical intervention.

These indicators are similar to those described above with the only difference being that the start of measurement of the corresponding period was considered as the start of using AVF.

Statistical analysis methods

For quantitative features (e.g. age, average duration of CVC use), the median and interquartile range (first and third quartiles) were calculated. Comparisons were performed using the Mann–Whitney U test. For nominal values (e.g. gender, localization of stenosis), fractions were calculated. Comparisons were performed using the chi-square test.

Patency was assessed using the Kaplan–Meier estimate. The significance of differences was assessed using the Mantel–Cox Logrank test (long term) and Gehan–Breslow–Wilcoxon (short term). Point estimates and 95% confidence intervals (95% CI) were calculated. In addition, the survival median (and 95% CI) was calculated, i.e. point in time when the event did not occur in 50% of subjects. Relative risk of event was assessed using the hazard ratio – HR (log-rank).

In order to take the total number of events into account when doing risk assessment, the incidence rate ratio was determined, which is the intensity of the onset of events: the number of events for a standardized time interval (for example, the number of operations of 10 patient-months of follow-up). The ratio of the two incidence rate ratios (IRRs) was interpreted as relative risk.

Calculations were performed in GraphPad v.8 and OpenEpi v.3. A two-sided level of significance was evaluated. Values $p < 0.05$ were considered statistically significant.

RESULTS

Indicators of functional patency, as well as patency after the first intervention for CVS are shown in Figure.

We did not notice any significant differences in the functional primary patency (Fig., a) between the groups: 89.7% [95% CI 74.9; 96] and 30.8% [95% CI 17.3; 45.4] after one year and three years, respectively, in the experimental group, 80.5% [95% CI 64.8; 89.7] and 24.4% [95% CI 12.7; 38.2] – in the control group, HR 0.7695 [95% CI 0.4952; 1.196], $p = 0.2259$; median in the experimental group – 29 months [95% CI 22.9; 35.1], in the control group – 25 months [95% CI 19.8; 30.2].

Moreover, there was lesser need for second intervention after BA compared with palliative "open" interventions, as evidenced by primary patency rates (Fig., b): 61.5% [95% CI 44.5; 74.7] and 15.4% [95% CI 6.2; 28.3] after 6 and 12 months, respectively, in the experimental group, 39% [95% CI 24.3; 53.4] and 0% in the control group, HR 0.5337 [95% CI 0.3381; 0.8427], $p = 0.0011$; median in the experimental group – 8 months [95% CI 6; 10], in the control group – 6 months [95% CI 4.9; 7.1].

In the experimental group, a strong inverse correlation between the primary patency and functional primary patency was found: $r = –0.627$ [95% CI –0.787; –0.388], $r^2 = 0.393$, $p < 0.0001$. In the control group, there was no such correlation: $r = 0.049$ [95% CI –0.262; –0.351], $r^2 = 0.002$, $p = 0.7599$.

The total duration of use of AVF in the experimental group was significantly longer than in the control group, and as evidenced by functional secondary patency rates (Fig., c): 100%, 74.4% [95% CI 57.6; 85.3] and 12.8% [95% CI 4.7; 25.2] after one, three and five years in the experimental group, 95.1% [95% CI 81.9; 98.8], 36.6% [95% CI 22.3; 51] and 4.9% [95% CI 0.9; 14.5] in the control group, HR 0.5661 [95% CI 0.3598; 0.8906], $p = 0.0067$; median in the experimental group – 47 months [95% CI 40.9; 53.1], in the control group – 34 months [95% CI 29.8; 38.2].

At the same time, BA allowed to significantly increase AVF duration after the first operation for CVS, as evidenced by secondary patency rates (Fig., d): 84.6% [95% CI 68.9; 92.8], 66.7% [95% CI 49.6; 79.1] and 17.9% [95% CI 7.9; 31.3] after 6, 12 and 24 months, respectively, in the experimental group, 56.1% [95% CI 39.7; 69.6], 19.5% [95% CI 9.2; 32.7] and 0% in the control group, HR 0.4009 [95% CI 0.2481; 0.6477], $p < 0.0001$; median in the experimental group – 16 months [95% CI 12.5; 19.5], in the control group – 7 months [95% CI 4.9; 9.1].

The occlusion-free period from the moment the use of AVF was stated was longer in the experimental group than in the control group, as evidenced by the functional primary assisted patency rate (Fig., e): 100%, 61.5% [95% CI 44.5; 74.7] and 2.6% [95% CI 0.2; 11.5] after one, three and five years, respectively, in the experimen-

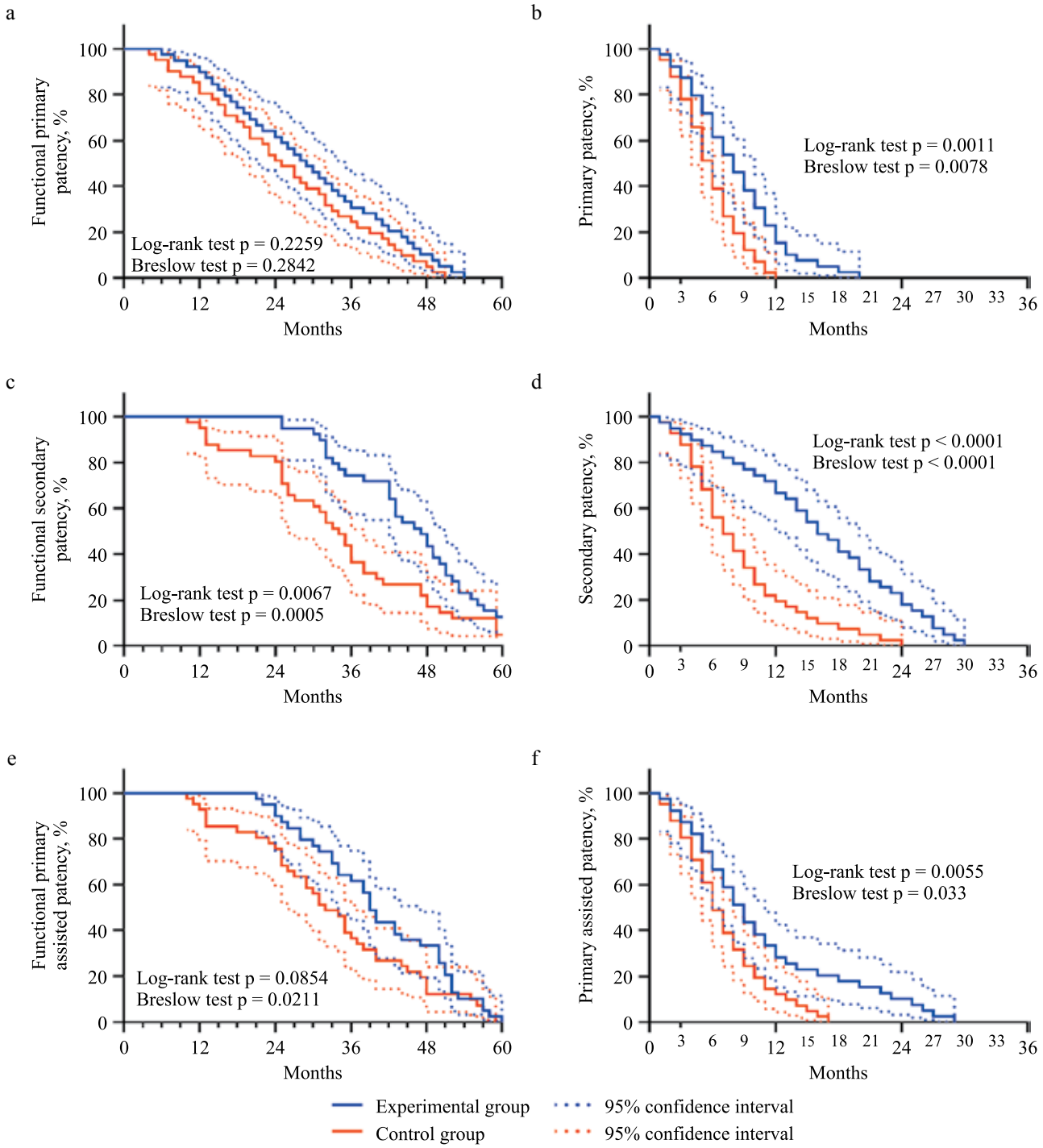

Fig. Functional patency rates – primary (a), secondary (c) and assisted primary (e); patency indicators after the first intervention for CVS – primary (b), secondary (d) and assisted primary (f). Red indicates a group of patients after balloon angioplasty, blue – after palliative "open" surgical interventions, the dots indicates 95% confidence intervals (Kaplan–Meier estimate)

tal group, 92.7% [95% CI 79; 97.6], 36.6% [95% CI 22.3; 51] and 0% in the control group, HR 0.7212 [95% CI 0.4633; 1.123], p = 0.1193; median in the experimental group – 39 months [95% CI 36.5; 41.5], in the control group – 32 months [95% CI 27.5; 36.5].

The occlusal period from the moment of the first surgical intervention was also significantly longer in the experimental group, as evidenced by the primary assisted patency rate (Fig., f): 66.7% [95% CI 49.6; 79.1], 28.2% [95% CI 15.3; 42.7] and 10.3% [95% CI 3.3; 22] after 6, 12 and 24 months, respectively, in the experimental group, 48.8% [95% CI 32.9; 62.9], 12.2% [95% CI 4.5; 24.1] and 0% in the control group, HR 0.5758 [95% CI 0.3664; 0.905], p = 0.0055; median in the experimental group – 9 months [95% CI 7; 11], in the control group – 7 months [95% CI 5.6; 8.4].

DISCUSSION

To increase the objectivity of the study, the sample was deliberately formed in such a way that all subjects had an outcome – AVF failure.

From Table it can be seen that both samples were obtained from the same set: we did not note any differences between the groups by main parameters. However, this population differs from the general HD patient population by distribution of CKD causes [17]. It is logical that the proportion of patients with CKD causes that predetermine the difficulties of providing constant vascular access (polycystic kidney disease, diabetes and systemic processes) were significantly higher. Although the first AVF creation in most patients was done before HD, a larger proportion of patients initiated HD via CKD. As a result, in 3–4 months before a stable vascular access was created and AVF was started, patients underwent an average of 3 reconstructive interventions performed in connection with primary failure: early thrombosis or delayed fistula vein maturation. Since the vast majority of these interventions consisted of AVA proximalization, by the time the use of AVA was started, most patients had AVF in the middle or upper third of the forearm (proximal AVF). We consider this an important factor in both the development of central venous stenosis and in its rapid clinical manifestation. It is known that the AVF high volumetric flow rate (which is characteristic of proximal AVF) leads to abnormal shear stress and turbulence. Non-physiological hemodynamics promotes endothelial dysfunction, activation of endotheliocytes and platelets, and neointimal hyperplasia. The vein walls thicken due to remodeling and fibrosis, [31–34]. At the same time, increased volumetric flow rate quickly leads to depletion of the functional-compensatory capabilities of the vein and its collaterals.

In the vast majority of cases, the main initiating factor for CVS is the use of CVC [9–11]. Indeed, the subjects had a high need for CVC (Table). Moreover, despite the fact that preference was given to permanent CVCs, the average duration (median) of using one was approximately 1.3–1.4 months. CVC dysfunctions or infectious complications required implantation of a new catheter. Despite the fact that this was not the immediate goal of our analysis, based on our own experience, we are inclined to conclude that the number of CVC implants is a more important risk factor for CVS development than catheterization duration. This has been confirmed by a number of studies [35–38]. Nevertheless, it should be noted that there is no consensus among researchers on this issue: some have the opposite opinion [36–39].

It is curious that in 4 patients (1 in the experimental group and 3 in the control group), CVCs were not used. It is known that idiopathic CVS is described, which nevertheless is extremely rare [40]. In this case, stenosis could occur in the area of confluence or branching, and in the area of twisted vein segments and anatomical bends, subject to increased pressure and sustained turbulent blood flow due to the presence of AVF from the ipsilateral side. [41, 42].

Symptoms of venous hypertension can occur in the absence of organic stenosis due to damaged vein walls. Functional stenosis may develop as a result of external compression of the vein by anatomical structures in the thoracic outlet [43–45]. This phenomenon is known as thoracic outlet syndrome [46]. Such observations in patients on hemodialysis are fairly well described [40, 47, 48]. In the most severe cases, Paget–Schroetter syndrome develops [49, 50]. The AVF high volumetric flow rate significantly promotes early clinical manifestation of vein compression (or organic stenosis) [40, 43, 44, 51–53].

External vein compression (as well as its physiological bends) against the background of direct arteriovenous discharge of a large volume of blood (as a result of AVF creation) can cause blood flow turbulence and promote neointimal hyperplasia and fibrosis [51, 54] of the vein wall. In this regard, chronic venous compression at the thoracic outlet level can be an important potential factor for CVS [55].

We noted a high incidence of subclavian vein stenosis, especially on the left. This agrees well with the data from other authors: the use of subclavian veins for catheterization (compared with the internal jugular veins) [7, 35, 37, 56, 57] and the use of left subclavian veins [7, 58] are associated with increased risk of stenosis. This can be explained by anatomical features: a more winding path to the right atrium, as well as a smaller vein diameter on the left [7, 10]. Nevertheless, such localization distribution of stenosis can distort reality, as localization of stenosis was one of the inclusion criteria. The study did not include patients with isolated jugular vein stenosis. This was done deliberately, since isolated jugular vein stenosis creates objective difficulties in CVC implantation, but affects AVF patency to a lesser extent.

A comparative analysis of treatment outcomes (comparing the patency rates with and functional patency rates) reveals some interesting nuances that allow to somewhat differently evaluate BA outcomes and supplement essentially the idea about optimal provision of stable vascular access for CVS patients.

Primary patency rates (Fig., b) are traditional: in the control group, one year after the first operation for CVS, all patients required another surgery. In the experimental group, by 15 months, 92% of patients needed another operation. Only a small group of patients required repeated surgical intervention at a later date. However, primary patency did not exceed 20 months.

Functional primary patency curves, on one hand, indicate that CVS manifestations requiring surgical treatment, can, with the same probability, occur at any time of its use. In both groups, the survival curves decrease almost linearly, which is an indirect sign that the intensity

of the onset of the event (the first repeated operation) is relatively constant. On the other hand, it is rather difficult to predict when the CVS clinical manifestation would occur. Obviously, this depends on many factors, among which we believe the main ones are the initial state of the patient's veins, AVF blood flow rate and a history of CVC use. The first surgeries for CVS in the experimental group were performed already after 6 months of AVF use, and in the control group – after 4. The combined influence of a number of factors contributed to the fact that clinically significant CVS had already formed in the patients within this period. In this regard, it is very important not only to identify significant risk factors for CVS (which is the subject of most of these studies), but also to assess with reasonable accuracy the unique impact of each of them on CVS *incidence* at different periods of treatment. It is likely that the impact of various factors on CVS risk will be different. For example, a patient who has successfully used one tunneled CVC for six months, and a patient who, for various reasons, underwent several catheterizations within one month, may have different risks of CVS. Results of such an analysis can serve as a reason for conducting, for example, angiography (an invasive and expensive method) for vascular access dysfunction in a patient at risk until clinically significant symptoms of venous hypertension appear.

We identified very important features when comparing primary patency with functional primary patency (Fig., a). These two indicators are inextricably linked. The endpoint for functional primary patency (first repeated operation after AVF formation) is the starting point for primary patency. In the experimental group, there was a strong inverse correlation between functional primary patency and primary patency: the later the first intervention for CVS was required, the earlier a repeated intervention would be required. This is understandable because hemodynamic disturbances against the background of a long-functioning AVF, on one hand, apparently lead to formation of the corresponding morphological substrate – change in the vein wall. On the other hand, a gradual increase of AVF blood flow (especially with proximal AVF) leads to manifestation of clinical signs of CVS. Since, as we have established, "late" stenoses are less treatable and after surgical resolution of CVS with BA, recurrence develops faster, the compensatory potential of venous collaterals does not have time to be fully achieved. No such dependence was revealed in the control group: duration of the first intervention for CVS to the second one did not depend on the length of time between AVF formation and CVS appearance. This can be explained by the fact that the essence of operations in the control group consisted of thrombectomy in case of thrombosis, which was supplemented by AVA proximalization if necessary or by reducing blood flow through AVF by forming a bandage from a synthetic vascular prosthesis on the juxtaanastomotic segment of the "fistula" vein, if the indication for the operation consisted of clinical manifestations of venous hypertension in the limb. Both the formation of a bandage and formation of a new AVA led to reduced blood flow through AVF. This, on one hand, indicates the important influence of this parameter on the clinical manifestations of CVS. On the other hand, the lack of significant correlation between functional primary patency and primary patency indicates that reducing AVF blood flow is an effective palliative operation for any CVS formation period (within our study), in contrast to the effectiveness of balloon angioplasty.

As follows from Fig., d, balloon angioplasty can significantly increase the secondary patency, i.e. the period between the first intervention for CVS and the complete loss of AVF function. Nevertheless, even in the experimental group, secondary patency does not exceed 30 months. In addition, differences between functional secondary patency curves are not so pronounced. In both groups, AVF function was completely lost 70 months after the start of AVF in the experimental group and 66 months in the control group. Despite the fact that differences between the groups were statistically significant (even in the long-term period, as evidenced by the P value for the log rank test), after 54 months, the confidence intervals cross the alternative survival curves. In other words, whenever CVS develops, its function will most likely be lost by 5 years after the start of AVF, regardless of the treatment method used. Given the fact that, according to the data in Table and Fig., a, the time interval between the start of using AVF and the first intervention for CVS (i.e., in fact, the duration for development of clinically significant CVS) did not differ between the groups. This can be explained by the fact that the effectiveness of balloon angioplasty decreases as the duration of AVF use increases. This is also confirmed by the presence of a significant inverse correlation between primary patency and functional primary patency. As a result, differences in secondary patency are partially offset.

At the same time, BA allowed to more than halve the risk of losing AVF function in the early stages of its use: in the control group, the first AVF was lost after 10 months, while in the experimental group – after 25 months (functional secondary patency – Fig., c).

Differences in primary and secondary AVF patency in the control group indicate that blood flow reduction is an effective palliative method for increasing AVF patency. However, there is no consensus on the optimal value of AVF volumetric blood flow rate. It must be remembered that significant decrease in this rate may increase the risk of AVF thrombosis [59–62]. In our study, whenever blood flow reduction was necessary, the target values were in the range of 1–1.5 liters per minute.

One of the important reasons for the higher functional secondary survival of AVF in patients from the experimental group is the fact that in the experimental

group, "open" surgical interventions only supplemented endovascular interventions if necessary, while in the control group, "open" interventions were the only option for surgical interventions. Moreover, since AVA proximalization was often required, it is natural that in the control group the "vascular resource" was exhausted more quickly.

Analysis of primary assisted patency (Fig., f) showed that the probability of AVF thrombosis is much lower in the late stages after the first intervention for CVS in the experimental group: repeated operations were performed in connection with increasing manifestations of venous hypertension (clinical manifestations, indirect "dialysis" signs: decreased HD effectiveness, increased pressure in the venous line, increased circulation in the vascular access). If the second operation was performed shortly after the first intervention for CVS, the differences between the groups are not so obvious: the P value is very close to the threshold of statistical significance ($p = 0.033$ according to the Breslow-Day test). When analyzing the functional primary assisted patency (i.e., when the starting point for the period corresponds with the start of AVF use), the results are somewhat different (Fig., e): the time interval between the start of AVF use and the first intervention for CVS compensates to some extent the differences between the groups. Differences in the long-term period are statistically insignificant (log-rank test $p = 0.0854$), but significant in the short term (Breslow-Day test $p = 0.0211$). However, both estimates are on the threshold of statistical significance. In other words, BA allows to slightly reduce the risk of AVF thrombosis. However, their effectiveness decreases as the duration of AVF use increases. At the same time, BA more than halved the risk of thrombosis in the early stages of its use – in the control group, the first AVF thrombosis occurred after 10 months, in the experimental group – after 21 months.

STUDY LIMITATIONS

First, the study was retrospective. Secondly, inclusion and exclusion criteria were determined to best achieve the research objective but limit the specific sample. Care should be taken when attempting to interpolate the resulting AVF patency estimates to the total HD patient population. The work was carried out to investigate the peculiarities of cause-effect relationships (which, in general, are relevant for the general HD patient population), and not to conduct a general assessment of the effectiveness of balloon angioplasty. Thirdly, the study did not include patients who used various stenting options. There is convincing evidence in favor of the fact that the use of stents can significantly increase patency [25, 63–68]. The main deterrent to the use of stents is the limited increase in primary patency, lack of clear indications for the use of stent and the choice of stent, as well as high cost of treatment [69]. Analysis of the effectiveness of angioplasty using stents requires a separate thorough investigation, which will be presented by us later. Fourth, we did not take into account the type of balloon, its working pressure and the extent of stenosis. There is reason to believe that these factors also have clinical significance [66, 70–72]. Fifth, we did not include in the study patients in whom AVF was created using a synthetic vascular prosthesis, as well as those patients in whom prosthesis was used during reconstructions (such patients were excluded from the study). This is an important factor in the context of our study, since it is obvious that a vascular prosthesis has less potential for significant increase in arteriovenous blood flow compared to native AVF.

CONCLUSION

Unfortunately, it must be recognized that CVS inevitably leads to loss of vascular access from the ipsilateral side. Balloon angioplasty, at the moment, is virtually a non-alternative way to quickly restore central vein patency in patients on HD. They allow to slightly extend the period of AVF use. However, BA outcomes significantly depend on the time interval between the start of AVF use and CVS appearance. At the same time, percutaneous balloon angioplasty is not able to radically change the long-term outcomes of CVS. If this complication develops, it is necessary to assess the possibility of forming a new vascular access from the contralateral side. Multiple repeated balloon angioplasties are apparently justified in patients in whom the possibility of creating a new vascular access is doubtful.

AVF volumetric flow rate is an important factor determining the severity of clinical manifestations of CVS and the need for repeated surgical interventions. AVF blood flow reduction is an effective palliative treatment for CVS.

The authors declare no conflict of interest.

REFERENCES

1. *Tomilina NA, Andrusev AM, Peregudova NG, Shinkarev MB*. Renal replacement therapy for End Stage Renal Disease in Russian Federation, 2010–2015. Russian National Renal Replacement Therapy Registry Report of Russian Public Organization of Nephrologists "Russian Dialysis Society", Part 1. *Nefrologiya i dializ [Nephrology and dialysis]*. 2017; 19 (4, supplement): 1–95. [In Russ, English abstract]. doi: 10.28996/1680-4422-2017-4suppl-1-95.
2. *Ravani P, Palmer SC, Oliver MJ, Quinn RR, MacRae JM, Tai DJ et al*. Associations between hemodialysis access type and clinical outcomes: a systematic review. *J Am Soc Nephrol*. 2013; 24 (3): 465–473. doi: 10.1681/ASN.2012070643.
3. *Almasri J, Alsawas M, Mainou M, Mustafa RA, Wang Z, Woo K et al.* Outcomes of vascular access for hemodialysis: A systematic review and meta-analysis. *J Vasc Surg*. 2016; 64 (1): 236–243. doi: 10.1016/j.jvs.2016.01.053.

4. *Arhuidese IJ, Orandi BJ, Nejim B, Malas M*. Utilization, patency, and complications associated with vascular access for hemodialysis in the United States. *J Vasc Surg*. 2018; 68 (4): 1166–1174. doi: 10.1016/j.jvs.2018.01.049.
5. *Miller LM, MacRae JM, Kiaii M, Clark E, Dipchand C, Kappel J* et al. Hemodialysis Tunneled Catheter Noninfectious Complications. *Can J Kidney Health Dis*. 2016; 3: 2054358116669130. doi: 10.1177/2054358116669130.
6. *Mansour M, Kamper L, Altenburg A, Haage P*. Radiological central vein treatment in vascular access. *J Vasc Access*. 2008; 9: 85e101.
7. *Agarwal AK, Patel BM, Haddad NJ*. Central vein stenosis: a nephrologist's perspective. *Semin Dial*. 2007; 20: 53e62.
8. *Kundu S*. Review of central venous disease in hemodialysis patients. *J Vasc Intervent Radiol JVIR*. 2010; 21: 963e8. doi: 10.1016/j.jvir.2010.01.044.
9. *MacRae JM, Ahmed A, Johnson N, Levin A, Kiaii M*. Central vein stenosis: a common problem in patients on hemodialysis. *ASAIO J*. 2005; 51 (1): 77–81.
10. *Agarwal AK*. Central vein stenosis. *Am J Kidney Dis*. 2013; 61 (6): 1001–1015. doi: 10.1053/j.ajkd.2012.10.024.
11. *Tedla FM, Clerger G, Distant D, Salifu M*. Prevalence of Central Vein Stenosis in Patients Referred for Vein Mapping. *Clin J Am Soc Nephrol*. 2018; 13 (7): 1063–1068. doi: 10.2215/CJN.14001217.
12. *Brown RS, Patibandla BK, Goldfarb-Rumyantzev AS*. The Survival Benefit of "Fistula First, Catheter Last" in Hemodialysis Is Primarily Due to Patient Factors. *J Am Soc Nephrol*. 2017; 28 (2): 645–652.
13. *Sequeira A, Naljayan M, Vachharajani TJ*. Vascular Access Guidelines: Summary, Rationale, and Controversies. *Tech Vasc Interv Radiol*. 2017; 20 (1): 2–8.
14. USRDS.org [Internet]. United States Renal Data System. 2016 USRDS annual data report. Volume 2 – End-stage Renal Disease (ESRD) in the United States: 1 · Incidence, Prevalence, Patient Characteristics, and Treatment Modalities 2016; Available at: https://www.usrds.org/2016/view/Default.aspx.
15. ERA-EDTA-reg.org [Internet]. European Renal Association – European Dialysis and Transplant Association (ERA-EDTA) Registry Annual Report 2015. 2017; Available at: https://www.era-edta-reg.org/files/annualreports/pdf/AnnRep2015.pdf.
16. *Bikbov BT, Tomilina NA*. Renal replacement therapy for ESRD in Russian Federation, 1998–2013 Report of the Russian Renal Replacement Therapy Registry. Part 1. *Nefrologiya i dializ [Nephrology and dialysis]*. 2015; 17 (3, supplement): 5–111. [In Russ, English abstract]. doi: 10.28996/1680-4422-2017-4suppl-1-95.
17. *Vatazin AV, Zulkarnaev AB, Fominykh NM, Kardanakhishvili ZB, Strugailo EV*. The creation and maintenance of vascular access for chronic hemodialysis in the Moscow region: a five-year experience of a regional center. *Russian Journal of Transplantology and Artificial Organs*. 2018; 20 (4): 44–53. [In Russ, English abstract]. doi: 10.15825/1995-1191-2018-4-44-53.
18. *Fant GF, Dennis VW, Quarles LD*. Late vascular complications of the subclavian dialysis catheter. *Am J Kidney Dis*. 1986; 7 (3): 225–228.
19. *Schwab SJ, Quarles LD, Middleton JP, Cohan RH, Saeed M, Dennis VW*. Hemodialysis-associated subclavian vein stenosis. *Kidney Int*. 1988; 33 (6): 1156–1159.
20. *Massmann A, Fries P, Obst-Gleditsch K, Minko P, Shayesteh-Kheslat R, Buecker A*. Paclitaxel-coated balloon angioplasty for symptomatic central vein restenosis in patients with hemodialysis fistulas. *J Endovasc Ther*. 2015; 22 (1): 74–79. doi: 10.1177/1526602814566907.
21. *Aj A, Razak Uk A, R P, Pai U, M S*. Percutaneous intervention for symptomatic central vein stenosis in patients with upper limb arteriovenous dialysis access. *Indian Heart J*. 2018; 70 (5): 690–698. doi: 10.1016/j.ihj.2018.01.013.
22. *Shi YX, Ye M, Liang W, Zhang H, Zhao YP, Zhang JW*. Endovascular treatment of central venous stenosis and obstruction in hemodialysis patients. *Chin Med J (Engl)*. 2013; 126 (3): 426–430.
23. *Surowiec SM, Fegley AJ, Tanski WJ, Sivamurthy N, Illig KA, Lee DE et al*. Endovascular management of central venous stenoses in the hemodialysis patient: results of percutaneous therapy. *Vasc Endovascular Surg*. 2004; 38 (4): 349–354.
24. *Massara M, De Caridi G, Alberti A, Volpe P, Spinelli F*. Symptomatic superior vena cava syndrome in hemodialysis patients: mid-term results of primary stenting. *Semin Vasc Surg*. 2016; 29 (4): 186–191. doi: 10.1053/j.semvascsurg.2017.05.001.
25. *Haskal ZJ, Trerotola S, Dolmatch B, Schuman E, Altman S, Mietling S et al*. Stent graft versus balloon angioplasty for failing dialysis-access grafts. *N Engl J Med*. 2010; 362 (6): 494–503. doi: 10.1056/NEJMoa0902045.
26. *Hongsakul K, Bannangkoon K, Rookkapan S, Boonsrirat U, Kritpracha B*. Paclitaxel-Coated Balloon Angioplasty for Early Restenosis of Central Veins in Hemodialysis Patients: A Single Center Initial Experience. *Korean J Radiol*. 2018; 19 (3): 410–416. doi: 10.3348/kjr.2018.19.3.410.
27. *Aftab SA, Tay KH, Irani FG, Gong Lo RH, Gogna A, Haaland B et al*. Randomized clinical trial of cutting balloon angioplasty versus high-pressure balloon angioplasty in hemodialysis arteriovenous fistula stenoses resistant to conventional balloon angioplasty. *J Vasc Interv Radiol*. 2014; 25 (2): 190–198. doi: 10.1016/j.jvir.2013.10.020.
28. *Linn BS, Linn MW, Gurel L*. Cumulative illness rating scale. *J Amer Geriatr Soc*. 1968; 16: 622–626.
29. *Miller MD, Paradis CF, Houck PR, Mazumdar S, Stack JA, Rifai AH et al*. Rating chronic medical illness burden in geropsychiatric practice and research: application of the Cumulative Illness Rating Scale. *Psychiatry Res*. 1992; 41: 237–248.
30. *Schmidli J, Widmer MK, Basile C, de Donato G, Gallieni M, Gibbons CP et al.* Editor's Choice – Vascular Access: 2018 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS). *Eur J Vasc Endovasc Surg*. 2018; 55 (6): 757–818. doi: 10.1016/j.ejvs.2018.02.001.

31. *Kundu S*. Central venous disease in hemodialysis patients: prevalence, etiology and treatment. *J Vasc Access*. 2010; 11 (1): 1–7.
32. *Roy-Chaudhury P, Spergel LM, Besarab A, Asif A, Ravani P*. Biology of arteriovenous fistula failure. *J Nephrol.* 2007; 20 (2): 150–163.
33. *Browne LD, Bashar K, Griffin P, Kavanagh EG, Walsh SR, Walsh MT*. The Role of Shear Stress in Arteriovenous Fistula Maturation and Failure: A Systematic Review. *PLoS One*. 2015; 10 (12): e0145795. doi: 10.1371/journal.pone.0145795.
34. *Fitts MK, Pike DB, Anderson K, Shiu YT*. Hemodynamic Shear Stress and Endothelial Dysfunction in Hemodialysis Access. *Open Urol Nephrol J*. 2014; 7 (Suppl 1 M5): 33–44.
35. *Osman OO, El-Magzoub AR, Elamin S*. Prevalence and Risk Factors of Central Venous Stenosis among Prevalent Hemodialysis Patients, a Single Center Experience. *Arab J Nephrol Transplant*. 2014; 7 (1): 45–47.
36. *Agarwal AK*. Central vein stenosis: current concepts. *Adv Chronic Kidney Dis*. 2009; 16 (5): 360–370. doi: 10.1053/j.ackd.2009.06.003.
37. *Naroienejad M, Saedi D, Rezvani A*. Prevalence of central vein stenosis following catheterization in patients with end-stage renal disease. *Saudi J Kidney Dis Transpl*. 2010; 21 (5): 975–978.
38. *Yardim H, Erkoc R, Soyoral YU, Begenik H, Avcu S*. Assessment of internal jugular vein thrombosis due to central venous catheter in hemodialysis patients: a retrospective and prospective serial evaluation with ultrasonography. *Clin Appl Thromb Hemost*. 2012; 18 (6): 662–665. doi: 10.1177/1076029611432739.
39. *Gonsalves CF, Eschelman DJ, Sullivan KL, DuBois N, Bonn J*. Incidence of central vein stenosis and occlusion following upper extremity PICC and port placement. *Cardiovasc Intervent Radiol*. 2003; 26 (2): 123–127.
40. *Oguzkurt L, Tercan F, Yildirim S, Torun D*. Central venous stenosis in haemodialysis patients without a previous history of catheter placement. *Eur J Radiol*. 2005; 55 (2): 237–242.
41. *Shi Y, Zhu M, Cheng J, Zhang J, Ni Z.* Venous stenosis in chronic dialysis patients with a well-functioning arteriovenous fistula. *Vascular*. 2016; 24 (1): 25–30. doi: 10.1177/1708538115575649.
42. *Dixon BS*. Why don't fistulas mature? *Kidney Int*. 2006; 70 (8): 1413–1422.
43. *Horita Y*. Percutaneous transluminal angioplasty for central venous stenosis or occlusion in hemodialysis patients. *J Vasc Access*. 2019; 20 (1_suppl): 87–92. doi: 10.1177/1129729817747545.
44. *Hall HC, Moudgill N, Kahn M, Burkhart R, Eisenberg J, Rao A et al.* An unusual cause of venous hypertension after dialysis access creation. *Ann Vasc Surg*. 2011; 25 (7): 983.e1-4.
45. *Collin G, Jones RG, Willis AP*. Central venous obstruction in the thorax. *Clin Radiol*. 2015; 70 (6): 654–660. doi: 10.1016/j.crad.2015.01.014.
46. *Murtazina AF, Nikitin SS, Naumova ES*. Thoracic outlet syndrome: clinical and diagnostic features. Neuromuscular Diseases. 2017; 7 (4): 10–19. [In Russ, English abstract]. doi: 10.17650/2222-8721-2017-7-4-10-19.
47. *Itkin M, Kraus MJ, Trerotola SO*. Extrinsic compression of the left innominate vein in hemodialysis patients. *J Vasc Interv Radiol*. 2004; 15 (1 Pt 1): 51–56.
48. *Glass C, Dugan M, Gillespie D, Doyle A, Illig K*. Costoclavicular venous decompression in patients with threatened arteriovenous hemodialysis access. *Ann Vasc Surg*. 2011; 25 (5): 640–645. doi: 10.1016/j.avsg.2010.12.020.
49. *Wooster M, Fernandez B, Summers KL, Illig KA*. Surgical and endovascular central venous reconstruction combined with thoracic outlet decompression in highly symptomatic patients. *J Vasc Surg Venous Lymphat Disord*. 2019; 7 (1): 106–112.e3. doi: 10.1016/j.jvsv.2018.07.019.
50. *Vemuri C, Salehi P, Benarroch-Gampel J, McLaughlin LN, Thompson RW*. Diagnosis and treatment of effort-induced thrombosis of the axillary subclavian vein due to venous thoracic outlet syndrome. *J Vasc Surg Venous Lymphat Disord*. 2016; 4 (4): 485–500. doi: 10.1016/j.jvsv.2016.01.004.
51. *Jennings WC, Miller GA, Coburn MZ, Howard CA, Lawless MA*. Vascular access flow reduction for arteriovenous fistula salvage in symptomatic patients with central venous occlusion. *J Vasc Access*. 2012; 13 (2): 157–162. doi: 10.5301/jva.5000020.
52. *Jennings WC, Maliska CM, Blebea J, Taubman KE*. Creating arteriovenous fistulas in patients with chronic central venous obstruction. *J Vasc Access*. 2016 7; 17 (3): 239–242. doi: 10.5301/jva.5000507.
53. *Sequeira A, Tan TW*. Complications of a High-flow Access and Its Management. *Semin Dial*. 2015; 28 (5): 533–543. doi: 10.1111/sdi.12366.
54. *Trerotola SO, Kothari S, Sammarco TE, Chittams JL*. Central venous stenosis is more often symptomatic in hemodialysis patients with grafts compared with fistulas. *J Vasc Interv Radiol*. 2015; 26 (2): 240–246. doi: 10.1016/j.jvir.2014.10.048.
55. *Kotoda A, Akimoto T, Sugase T, Yamamoto H, Kusano E*. Is there a link between the structural impact of thoracic outlet and the development of central venous stenosis? *Med Hypotheses*. 2013; 80 (1): 29–31. doi: 10.1016/j.mehy.2012.09.023.
56. *Bozof R, Kats M, Barker J, Allon M*. Time to symptomatic vascular stenosis at different locations in patients with arteriovenous grafts. *Semin Dial*. 2008; 21: 285e8. doi: 10.1111/j.1525-139X.2008.00436.x.
57. *Ruesch S, Walder B, Tramèr MR*. Complications of central venous catheters: internal jugular versus subclavian access – a systematic review. *Crit Care Med*. 2002; 30 (2): 454–460.
58. *Gibson F, Bodenham A*. Misplaced central venous catheters: applied anatomy and practical management. *Br J Anaesth*. 2013; 110 (3): 333–346. doi: 10.1093/bja/aes497.
59. Kidney.org [Internet]. KDOQI. Clinical practice guidelines for vascular access. 2006; Available at: https://www.kidney.org/sites/default/files/docs/12-50-0210_jag_dcp_guidelines-pd_oct06_sectionb_ofc.pdf.

60. *Benaragama KS, Barwell J, Lord C, John BJ, Babber A, Sandoval S et al.* Post-operative arterio-venous fistula blood flow influences primary and secondary patency following access surgery. *J Ren Care*. 2018. doi: 10.1111/jorc.12238.
61. *Polkinghorne KR, Kerr PG*. Epidemiology and blood flow surveillance of the native arteriovenous fistula: a review of the recent literature. *Hemodial Int*. 2003; 7 (3): 209–215. doi: 10.1046/j.1492-7535.2003.00039.x.
62. *Aragoncillo I, Abad S, Caldés S, Amézquita Y, Vega A, Cirugeda A et al.* Adding access blood flow surveillance reduces thrombosis and improves arteriovenous fistula patency: a randomized controlled trial. *J Vasc Access*. 2017; 18 (4): 352–358. doi: 10.5301/jva.5000700.
63. *Miquelin DG, Reis LF, da Silva AA, de Godoy JM*. Percutaneous transluminal angioplasty in the treatment of stenosis of arteriovenous fistulae for hemodialysis. *Int Arch Med*. 2008; 1 (1): 16. doi: 10.1186/1755-7682-1-16.
64. *Haskal ZJ, Saad TF, Hoggard JG, Cooper RI, Lipkowitz GS, Gerges A et al*. Prospective, Randomized, Concurrently-Controlled Study of a Stent Graft versus Balloon Angioplasty for Treatment of Arteriovenous Access Graft Stenosis: 2-Year Results of the RENOVA Study. *J Vasc Interv Radiol*. 2016; 27 (8): 1105–1114.e3. doi: 10.1016/j.jvir.2016.05.019.
65. *Abreo K, Sequeira A*. Role of stents in hemodialysis vascular access. *J Vasc Access*. 2018; 19 (4): 341–345. doi: 10.1177/1129729818761280.
66. *Agarwal SK, Nadkarni GN, Yacoub R, Patel AA, Jenkins JS, Collins TJ et al*. Comparison of Cutting Balloon Angioplasty and Percutaneous Balloon Angioplasty of Arteriovenous Fistula Stenosis: A Meta-Analysis and Systematic Review of Randomized Clinical Trials. *J Interv Cardiol*. 2015; 28 (3): 288–295. doi: 10.1111/joic.12202.
67. *Kim CY, Guevara CJ, Engstrom BI, Gage SM, O'Brien PJ, Miller MJ et al*. Analysis of infection risk following covered stent exclusion of pseudoaneurysms in prosthetic arteriovenous hemodialysis access grafts. *Journal of vascular and interventional radiology: JVIR*. 2012; 23 (1): 69–74.
68. *Jones RG, Willis AP, Jones C*. Long-term results of stent-graft placement to treat central venous stenosis and occlusion in hemodialysis patients with arteriovenous fistulas. *J. Vasc. Interv. Radiol*. 2011; 22 (9): 1240–1245.
69. *Schmidli J, Widmer MK, Basile C, de Donato G, Gallieni M, Gibbons CP et al*. Editor's Choice – Vascular Access: 2018 Clinical Practice Guidelines of the European Society for Vascular Surgery (ESVS). *Eur J Vasc Endovasc Surg*. 2018; 55 (6): 757–818. doi: 10.1016/j.ejvs.2018.02.001.
70. *Mickley V*. Central vein obstruction in vascular access. *Eur J Vasc Endovasc Surg*. 2006; 32 (4): 439–444.
71. *Khawaja AZ, Cassidy DB, Al Shakarchi J, McGrogan DG, Inston NG, Jones RG*. Systematic review of drug eluting balloon angioplasty for arteriovenous haemodialysis access stenosis. *J Vasc Access*. 2016; 17 (2): 103–110. doi: 10.5301/jva.5000508.
72. *Hongsakul K, Bannangkoon K, Rookkapan S, Boonsrirat U, Kritpracha B*. Paclitaxel-Coated Balloon Angioplasty for Early Restenosis of Central Veins in Hemodialysis Patients: A Single Center Initial Experience. *Korean J Radiol*. 2018; 19 (3): 410–416. doi: 10.3348/kjr.2018.19.3.410.

The article was submitted to the journal on 29.08.2019