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RISK FACTORS FOR THE FORMATION OF ARTERIOVENOUS FISTULA THROMBOSIS IN PATIENTS TREATED WITH CHRONIC HEMODIALYSIS

FAKTORI RIZIKA ZA NASTANAK TROMBOZE ARTERIOVENSKE FISTULE KOD BOLESNIKA LEČENIH HRONIČNIM HEMODIJALIZAMA

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Summary

Introduction. Complications related to vascular access, alongside cardiovascular diseases, constitute the primary cause of hospitalization among patients undergoing hemodialysis. Thrombosis stands out as the most prevalent cause of arteriovenous fistula dysfunction. The research aimed to identify the risk factors contributing to thrombosis formation of the first arteriovenous fistula. **Material and Methods.** The study spanned one year and involved 40 patients who initiated hemodialysis through their first arteriovenous fistula at the University Clinical Centre of Vojvodina. The parameters analyzed included demographic, biochemical, and clinical factors, as well as the therapy given. **Results.** Among the 40 patients, 67% were male. The majority (57.5%) were aged over ≥ 60 years. Hypertension was the most prevalent comorbidity, affecting 35% of patients. Arteriovenous fistula thrombosis was diagnosed in 32% of patients. A logistical regression model was employed to determine predictors of arteriovenous fistula thrombosis. The analysis revealed that the patients with a body mass index greater than > 25 kg/m^2, had a 1.5 times higher risk of thrombosis formation. Similarly, individuals with blood pressure exceeding > 140/90 mmHg had nearly a twofold increased risk, while those in the 45-59 age group had a fourfold higher risk. Patients who received anticoagulant therapy before arteriovenous fistula formation had a 16 times lower risk of thrombosis, and nearly 33 times lower risk with the application of angiotensin–converting–enzyme inhibitors/angiotensin II receptor blocker after arteriovenous fistula formation. **Conclusion.** Significant predictors of thrombosis formation of the first arteriovenous fistula among hemodialysis patients included body mass index greater than > 25 kg/m^2, blood pressure values exceeding > 140/90 mmHg, and age group of 45 – 69 years.

Key words: Arteriovenous Fistula; Thrombosis; Risk Factors; Renal Dialysis; Treatment Outcome

Sažetak

Uvod. Komplikacije vaskularnih pristupa, pored kardiovaskularnih bolesti predstavljaju vodeći uzrok hospitalizacija kod bolesnika koji se leče hemodijalizama. Tromboza je najčešći uzrok afunkcije arteriovenske fistule. Cilj ovog istraživanja bio je da se utvrde faktori rizika za nastanak tromboze prve arteriovenske fistule. **Materijal i metode.** Istraživanjem u trajanju od godinu dana obuhvaćeno je 40 bolesnika koji su započeli hemodijalizu preko prve arteriovenske fistule u Univerzitetskom kliničkom centru Vojvodine. Analizirani su demografski, biohemijski, klinički parametri i primenjena terapija. **Rezultati.** Od ukupno 40 bolesnika, 67% su bili muškog pola. Najzastupljenija starosna kategorija od 57,5% bila je ≥ 60 godina. Od komorbiditeta hipertenzija je na prvom mestu sa 35%. Tromboza arteriovenske fistule dijagnostikovana je kod 32% bolesnika. Radi utvrđivanja prediktora tromboze arteriovenske fistule urađen je logistički regresioni model koji je pokazao da su bolesnici sa indeksom telesne mase > 25 kg/m² imali 1,5 puta veću šansu za nastanak tromboze, skoro dva puta veću šansu sa krvnim pritiskom > 140/90 mmHg i četiri puta veću šansu za starosnu kategoriju 45−59 godina. Bolesnici koji su bili na antikoagulantnoj terapiji pre kreiranja arteriovenske fistule imali su 16 puta manju šansu za nastanak tromboze i čak 33 puta manju šansu za nastanak tromboze sa primenom inhibitora angiotenzin–konvertujućeg enzima/blokatora receptora za angiotenzin II posle kreiranja arteriovenske fistule. **Zaključak.** Značajni prediktori nastanka tromboze prve arteriovenske fistule kod bolesnika na hemodijalizi bili su: indeks telesne mase > 25 kg/m², vrednost krvnog pritiska > 140/90 mmHg i starosna kategorija 45−59 godina.

Ključne reči: arteriovenska fistula; tromboza; faktori rizika; hemodijaliza; ishod lečenja

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Abbreviations

CKD – chronic kidney disease
AVF – arteriovenous fistula
BMI – body mass index
DM – diabetes mellitus
HD – hemodialysis
ARB – angiotensin II receptor blockers
TT – thrombin time
PT – prothrombin time
OR – odds ratio
CRP – C-reactive protein
CI – confidence intervals
ACE – angiotensin-converting enzyme

Introduction

In terminal stadium chronic kidney disease (CKD), it is necessary to commence healing with one of the kidney function replacement methods: hemodialysis (HD), peritoneal dialysis, or kidney transplant [1]. Arteriovenous fistula (AVF) is advocated as the preferred vascular access option for HD due to its longevity and fewer complications compared to other vascular access types [2]. However, despite having the fewest complications, arteriovenous fistula can still be associated with intraoperative, early, and later postoperative complications [3]. Thrombosis, along with stenosis, ranks among the most common later complication of AVF, with thrombosis incidence ranging from 17% to 25%. It stands as one of the leading causes of AVF dysfunction, necessitating the placement of a dialysis catheter to continue HD. Patients with hematocrit levels higher than 40%, lower level of protein C and S, and confirmed mutations of factors V Leiden and lupus anticoagulant are at higher risk of thrombosis [4]. The aim of this study was to identify the risk factors contributing to thrombosis formation in the first arteriovenous fistula. We also expected that arteriovenous fistula thrombosis would occur more frequently in female patients aged over 60 years, and that the most common clinical parameters such as diabetes mellitus and hyperlipidemia would be prevalent risk factors.

Material and Methods

The retrospective study was conducted at the Hemodialysis Department of the Clinic of Nephrology and Clinical Immunology of the University Clinical Centre of Vojvodina from 2017 to 2018. The study period predates the Covid-19 pandemic, as patients were subsequently transferred to other satellite dialysis centers and a new COVID hospital, resulting in incomplete patient data access. The study included 40 patients who initiated HD with their first AVF during 2017, and their data were followed until the end of 2018, kidney transplant, transfer to another dialysis center, or death. Patients permanently transferred from peritoneal dialysis to HD within the first year were included. The patients underwent dialysis 1-3 times a week for 4 hours, utilizing bicarbonate on polysulfonic capillary membranes with a surface ranging from 1.1 to 1.3 m^2 and a blood flow rate of 300 ml/min. Inclusion criteria comprised age ranging from 18 to 80 years, initiation of HD with the first created AVF, and absence of evidence of secondary hemophilia or heparin-induced thrombocytopenia. Patients immediately transferred to another satellite dialysis center upon initiating treatment at the Clinical Centre of Vojvodina, as well as those with diagnosed malignant disseminated disease, were excluded from the study. Through the research period, the following biochemical parameters were analyzed monthly: complete blood analyses, hemostatic mechanism, prothrombin time (PT), thrombin time (TT), fibrinogen (g/l).

Data analyzed every three months included nutritional, lipid, and inflammation parameter: albumin (g/l), total cholesterol, triglycerides, and C-reactive protein (CRP) (mg/l). AVF functionality was evaluated through clinical examination and Doppler ultrasound of the upper extremities. Complete blood analyses with a differential blood count were conducted on SYSMEX XN-1000 apparatus employing the flow cytometry method and commercial kits from the same manufacturer. Serum albumin concentration was determined using a photometric color test on the OLYMPUS analyzer with Beckman Coulter kits (Ireland). Total cholesterol and triglycerides were measured using standard enzyme procedure with reagents from BIOMERIEUX. Serum CRP concentration was determined through immune turbidimetric testing on the Siemens Dimension Xpand analyzer with commercial tests from Siemens. Parameters of the hemostatic mechanism (PT, TT, fibrinogen) were determined using a coagulant method on Siemens apparatus. HD adequacy was analyzed based on the Kt/Vsp index, calculated using the formula: $Kt/Vsp = \ln(C2/C1 - 0.008 \times T) + (4 - 3.5 \times C2/C1) \times UF/W$, where: C1 represents the redialysis value of urea (mmol/l), C2 represents the post-dialysis value of urea (mmol/l), T is the dialysis time span (hours), UF is the inter-dialysis proceeds (liters), W is the post-dialysis body mass (kg).

Statistical data analyses included descriptive statistics methods, with numerical data presented as mean values (arithmetic mean) and measures of variability (standard deviation), while categorical data were presented as frequencies and percentages. Differences in analyzed parameters between two or more groups were assessed using appropriate Student t-test, actually nonparametric Mann-Whitney test, and Kruskal-Wallis tests. Differences in categorical data were assessed using the chi-square test. Multiple analyses utilized binary logistic regression models, and coefficient significance assessed using the Wald test, odds ratio (OR), and 95% confidence intervals (CI). Values of $p<0.05$ were considered statistically significant.

Results

This research included 40 patients, with 27 (67%) being men. The oldest age category (>60) was the most prevalent. Hypertension was the most common comorbidity (35%), followed by diabetes mellitus (25%), glomerulonephritis (10%), polycystic kidney disease (10%), tubulointerstitial nephritis (5%), and other co-

Table 1. Demographic, clinical parameters and therapy of patients after the first AVF creation
Tabela 1. *Demografski, klinički parametri i terapija bolesnika posle kreiranja prve AVF*

	N/*Br.*
Age/*Starost*	
30-44	4 (10.0)
45-59	13 (32.5)
≥60	23 (57.5)
Comorbidities/*Komorbiditeti*	
Diabetes mellitus/*Dijabetes melitus*	10 (25)
Arterial hypertension/*Arterijska hipertenzija*	14 (35)
Hyperlipidemia/*Hiperlipidemija*	7 (18)
Glomerulonephritis/*Glomerulonefritis*	4 (10)
PKD/*PBB*	4 (10)
TIN/*TIN*	2 (5)
Other diseases/*Drugo oboljenje*	6 (15)
Therapy/*Terapij*a	
UFH	6 (15)
LMWH	22 (55)
Statins/*Statini*	19 (47.5)
ACE/ARBs	17 (42.5)
ESA	11 (27.5)
Without therapy change/*Bez promene terapije*	14 (35)

Legend: UFH – unfractionated heparin; LMWH – low-molecular-weight heparin; ACE –angiotensin-converting enzyme; ARBs – angiotensin II receptor blockers; ESA – erythropoietin-stimulating agent; PKD – Polycystic kidney disease, TIN –tubulointerstitial nephritis
Legenda: UFH − nefrakcionisani heparin; LMWH − niskomolekularni heparin; ACE − angiotenzin-konvertujući enzim; ARB − blokatori receptora angiotenzina II; ESA − eritropoetin; PBB − policistična bolest bubrega, TIN − tubulointersticijalni nefritis

Table 2. Laboratory and clinical parameters of patients with and without thrombosis
Tabela 2. *Laboratorijski i klinički parametri bolesnika sa trombozom i bez nje*

	Patients with thrombosis *Bolesnici sa trombozom* N/*Br.* 13	Patients without thrombosis *Bolesnici bez tromboze* N/*Br.* 27
Blood pressure/*Krvni pritisak* (mmHg) (N/*Br.* %)		
>140/90 mmHg	9 (30)	21 (70)
<140/90 mmHg	4 (40)	6 (60)
BMI/*Indeks telesne mase* (kg/m^2) (n%)		
Malnutrition/*Pothranjenost*	0 (0)	2 (100)
Normal nutrition/*Normalna uhranjenost*	4 (30.8)	9 (69.2)
Pre-obesity/*Predgojaznost*	6 (37.5)	10 (62.5)
Obesity/*Gojaznost*	3 (33.3)	6 (66.7)
Lab parameters[a] *Laboratorijski parametri*[a]	Arithmetic mean *Aritmetička sredina*	Standard deviation *Standardna devijacija*
Hemoglobin/*Hemoglobin* (g/l)	89.08 ± 27.11	101.59 ± 9.55
Erythrocytes/*Eritrociti* (x 10^{12})	3.01 ± 0.33	3.23 ± 0.39
Plateles/*Trombociti* (x 10^9)	196.31 ± 78.91	206.48 ± 71.47
Albumins/*Albumini* (g/l)	39.15 ± 6.46	36.74 ± 7.73
CRP/C *reaktivni protein* (mg/l)	9.63 ± 5.96	10.18 ± 9.87
Cholesterol/*Holesterol* (mmol/l)	4.39 ± 1.07	4.28 ± 0.96
Triglycerides/*Trigliceridi* (mmol/l)	1.87 ± 1.08	1.68 ± 1.17
PT (R)	0.87 ± 0.82	0.69 ± 0.56
TT (R)	0.72 ± 0.67	0.63 ± 0.49
Fibrinogen/*Fibrinogen* (g/l)	5.18 ± 2.67	5.03 ± 1.71

Legend: CRP – C-reactive protein; PT – Prothrombin time; TT – Thrombin time;
Legenda: CRP − C-reaktivni protein; PT − protrombinsko vreme; TT − trombinsko vreme;

morbidities (15%). After the creation of AVF, low molecular heparin therapy was initiated in 55% of patients, statins in 47.5%, and angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARB) in 42.5% of patients **(Table 1).**

Among patients with AVF thrombosis, 30% had blood pressure values >140/90 mmHg, while 40% of patients had values <140/90 mmHg. Additionally, 33.5% of pre-obese patients and 33.3% of obese patients experienced thrombosis. Patients with AVF thrombosis

Table 3. Predictors of AVF thrombosis
Tabela 3. *Prediktori tromboze arteriovenske fistule (AVF)*

Independent factors *Nezavisni faktori*	B	Wald	p/*p*	OR	95% CI za OR	
					Lower/*Nizak*	Upper/*Visok*
Gender/*Pol* Male/*Muški* Female/*Ženski*	-0.863	1.149	0.284	0.422	0.087	2.045
Age/*Starost* 30-44 45-59 ≥60	 1.472 0.788	 1.382 0.487	 0.040* 0.485	 4.366 2.197	 0.020 0.050	 2.671 4.165
Constant/*Konstanta*	0.432	0.154	0.695	1.540		
Hemoglobin/*Hemoglobin* (g/l)	-0.050	1.537	0.215	0.951	0.879	1.029
Erythrocytes/*Eritrociti* (x 10^{12})	-1.759	1.717	0.190	0.172	0.012	2.392
Plateles/*Trombociti* (x 10^{9})	-0.007	0.877	0.349	0.994	0.980	1.007
Albumins/*Albumini* (g/l)	0.050	0.809	0.368	1.051	0.943	1.173
CRP (mg/l)/*CRP* (mg/l)	0.023	0.184	0.668	1.024	0.920	1.139
Cholesterol/*Holesterol* (mmol/l)	0.721	1.044	0.307	2.056	0.516	8.196
TG/*TG* (mmol/l)	-0.223	0.877	0.684	0.800	0.273	2.341
PT (R)/*PT (R)*	-0.566	0.169	0.681	0.568	0.038	8.456
TT (R)/*TT (R)*	-1.487	1.074	0.300	0.226	0.014	3.762
Fibrinogen/*Fibrinogen* (g/l)	0.312	1.611	0.204	1.367	0.844	2.214
Constant/*Konstanta*	5.524	0.876	0.349	250.519		
Comorbidities/*Komorbiditeti*	0.566	2.828	0.039*	1.761	0.911	3.404
DM Yes/No/*Da/Ne*	0.637	0.524	0.469	2.529	0.094	2.970
HLP Yes/No/*Da/Ne*	0.447	0.297	0.586	1.564	0.313	7.809
OAC[a] Yes/No/*Da/Ne*	-2.793	4.159	0.041*	0.061	0.004	0.897
OAC[b] Yes/No/*Da/Ne*	-0.081	0.003	0.959	0.823	0042	0.140
APT[b] Yes/No/*Da/Ne*	-0.304	0.065	0.799	0.738	0.071	7.696
ACE/ARB[b] Yes/No/ *Da/Ne*	-3.490	4.397	0.036*	0.030	0.001	0.796
Statins[b] Yes/No/*Statini Da/Ne*	-0.052	0.609	0.435	0.592	0.237	8.352
Constant/*Konstanta*	0.415	0.050	0.824	1.515		
BP/*KP* (mmHg) ≥140/90 <140/90	0.304	0.146	0.002*	1.738	0.155	3.508
Kt/V	-1.234	0.510	0.475	0.291	0.010	8.605
BMI/*Indeks telesne mase* (kg/m^2) <25 >25	0.439	0.363	0.047*	1.551	0.372	6.463
Number of HDs a week/*Broj Hd nedeljno*	0.613	0.659	0.862	1.864	0.166	4.496
Constant/*Konstanta*	-1.597	0.291	0.590	0.202		

Legend: CRP – C-reactive protein; ACE/ARBs – Angiotensin-converting enzyme/angiotensin II receptor blockers; BMI – body mass index; APTT – Activated partial thromboplastin time; PT – Prothrombin time; TT – Thrombin time; HD – hemodialysis; BP – blood pressure; AVF – arteriovenous fistula; TG – triglycerides; DM – Diabetes mellitus; HP – hyperlipoproteinemia; OAC – anticoagulation therapy; APT – antiplatelet therapy
[a]before AVF creation; [b]after AVF creation
Legenda: CRP – C-reaktivni protein; ACE/ARB – angiotenzin-konvertujući enzim/blokatori angitenzin II receptora; BMI – indeks telesne mase; APTT – aktivirano parcijalno tromboplastinsko vreme; PT – protrombinsko vreme; TT – trombinsko vreme; HD – hemodijaliza; KP – krvni pritisak; AVF – arteriovenska fistula: TG – trigliceridi; DM – dijabetes melitus; HP – hiperlipoproteinemija; AOK – antikoagulantna terapija; AKS – antiagregaciona terapija; CI – interval poverenja; PR – odds ratio
[a]pre kreiranja AVF; [b]posle kreiranja AVF

exhibited significantly lower hemoglobin values (89.08 +27.11 vs. p=0.030) compared to those without thrombosis **(Table 2).**

Table 3 displays the predictors of AVF thrombosis. Patients aged 45-59 years had a fourfold higher chance of developing AVF thrombosis (OR = 4.366, 95% CI:0.020 - 2,671, p=0.040) compared to the youngest patients (30-44 years old). To identify predictors of AVF thrombosis initiation, a multivariable logistic regression analysis was performed with AVF thrombosis as the dependent parameter and hemoglobin, erythrocytes, platelets, albumin, CRP, cholesterol, triglycerides, PT, TT, and fibrinogen as independent variables.

Higher PT values were associated with a nearly twofold smaller chance of AVF thrombosis development (OR=0.568, 95% CI:0.038-8.456), while higher TT values were associated with approximately 4.5 times smaller chance (OR=0.226, 95% CI:0.014-3.762). An increase in fibrinogen values was associated with a nearly 1.4 times higher chance of thrombosis development (OR=1.367, 95% CI:0.844-2.214). Moreover, an increase in the number of comorbidities was associated with an almost twofold higher chance of AVF thrombosis (OR=1.761, 95% CI:0.911-3.404, p=0.039). The use of anticoagulant therapy before AVF creation decreased the chance of thrombosis by 16 times (OR=0.061, 95% CI:0.004-897, p=0.031), while the use of ACE/ARB upon AVF creation decreased the chance by 33 times (OR=0.030, 95% CI:0.001-796, p=0.036). Patients with blood pressure ≥140/90 mmHg had almost double the chance of developing AVF thrombosis (OR=1.738, 95% CI:0.155–3.508 p=0.002), while pre-obese and obese patients (BMI >25 kg/m^2) had a 1.5 greater chance of thrombosis (OR=1.551; 95% CI:0.372–6.463, p=0.047) **(Table 3).**

Discussion

In our research, we analyzed the influence of potential independent risk factors on the development of thrombosis in the first AVF. When considering the influence of gender on the creation of AVF thrombosis, our findings did not reveal a significant difference between men and women. This contrasts the previous research where AVF complications were predominantly associated with female patients, as demonstrated in various studies including that of Rodriguez et al., which involved 1033 patients over 13 years [5]. However, a doctoral thesis by Jankovic A. conducted an 18-month study at the Clinical Hospital Center Zvezdara in Belgrade, which included 314 patients, and concluded that AVF complications were more prevalent among male patients [6]. Considering that the latter research studies were of non-homogenized types, with varying designs and time span, it is not surprising that different results were observed. Research findings regarding the impact of age on AVF function were not consistent with previous studies. Two studies indicated that age over 65 years resulted in poorer AVF function and suggested earlier planning of vascular access [5, 7]. In contrast, Jankovic A.'s results did not demonstrate a significant impact of age on the development of AVF complications [6]. This aligns with the research by Lock et al., who followed AVF function for six months with 248 patients under 65 year old and 196 patients over 65 year old, finding no difference between these groups. Therefore, they concluded that age should not influence the function and decision-making regarding AVF creation [8]. Although more than 50% of our patients with AVF thrombosis were aged 60 years or older, upon comparison across age groups, it was determined that patients aged 45-59 years had a fourfold greater chance of developing AVF thrombosis, which is significantly more compared to the youngest patients (30-44 years old). In our research, when examining the impact of laboratory parameters on the development of AVF thrombosis, the only significant finding was lower hemoglobin values in patients with thrombosis compared to those without thrombosis. Given that thrombosis accounts for 80% of AVF dysfunction cases, the pharmacological prevention of thrombosis is crucial. This involves the use of antithrombotic medications such as acetylsalicylic acid, clopidogrel, dipyridamole, or parenteral anticoagulants (low molecular weight and unfractionated heparin). Opinions regarding the application of anticoagulant therapy vary, but based on existing research, it can be concluded that anticoagulant therapy in AVF patients contributes to thrombosis prevention and the long-term functionality of AVF without the risk of bleeding [9]. The results of our research revealed that after AVF creation, 55% of patients were prescribed low molecular weight heparin therapy, 47.5% were prescribed statins, and 42.5% were prescribed ACE inhibitors. Before AVF creation, 75% of patients were not receiving anticoagulant therapy, 15% received anticoagulant therapy for up to a year, and 10% received it for more than a year. One in four patients was on some form of anticoagulant therapy before AVF creation. The logistic regression model demonstrated that patients who received anticoagulant therapy before AVF creation had significantly lower chance (16 times less) of developing thrombosis compared to those who did not receive this therapy. Furthermore, patients who were prescribed ACE/ARB upon fistula creation had an even greater reduction in the risk of AVF thrombosis, with a 33 times lower chance. Filipov P., in his doctoral thesis, conducted a five-year study involving 121 patients and found that AVF thrombosis occurred in 17.4% of patients. Among the 40 patients not taking medication, thrombosis occurred in 30% of cases, whereas in the group receiving antithrombotic therapy, thrombosis occurred in only 9.5% of cases [9]. These results highlight the variability in study outcomes and the need for precise guidelines on medication use to maintain AVF patency. In a two-year retrospective study involving 349 patients, none of the cardiovascular drugs including antiplatelet agents, dipyridamole and others demonstrated a positive effect on maintaining AVF patency. How-

ever, contrasting results were observed in a 10-year study involving 34354 patients with short-lasting AVF (≤1 year) and long–lasting AVF (>1 year). The study indicated a significant reduction in the risk of AVF failure with an increase in the cumulative daily dosage of antiplatelet drugs [10]. Similarly, a multicenter study involving 338 patients did not examine the dosage of antiplatelet drugs but showed that their use for 30 to 90 days after AVF creation significantly prolonged its duration, helping to overcome the period of common expected complications [11]. Among the investigated clinical predictors, an increase in the number of comorbidities was shown to significantly increase the risk of AVF thrombosis. Any additional comorbidity doubled the likelihood of AVF thrombosis development. Among the patients with DM, AVF thrombosis occurred in 22% of cases, a condition that, along with hyperlipidemia, was present in over 50% of patients. Our research findings indicate that patients with DM had a 2.5 times greater chance of developing AVF thrombosis, although this association did not reach statistical significance. Various studies have yielded differing results regarding the impact of DM on the function and complication development of arteriovenous fistula [6]. Alongside DM, hyperlipidemia represents another common cause of AVF thrombosis. In our study, hyperlipidemia was present in 18% of patients, and these patients had a 1.5 times greater chance of developing thrombosis compared to others, although this association did not reach statistical significance. Previous researched studies have shown varied results [3, 12]. Our patients were analyzed for HD parameter impact on AVF thrombosis development. We found that 40% of patients had a BMI of 25–29.9 kg/m^2, and 22.5% had a BMI ≥30 kg/m^2 among patients with thrombosis. Pre-obese and obese patients (BMI >25 kg/m^2) had a 1.5 times greater chance of developing AVF thrombosis. For obese patients, decreased AVF function may result from heavy cannulation due to deeply situated blood vessels. In such cases, AVF with surface transposition of arterialized vein is typically the first choice, followed by arteriovenous graft creation or the placement of a long-lasting dialysis catheter if unsuccessful [13]. Jemcov T., et al. in a two-year study involving 122 patients, found a six times higher risk of poor AVF function in malnourished and obese patients [14]. However, Lee et al. in a three-year survey of 173 patients did not find a significant effect of BMI on AVF outcomes [15]. Additionally, a study by Chan et al. indicated that only patients with BMI >35 kg/m² had poor AVF maturation [16]. Arterial blood pressure values are another risk factor for AVF thrombosis development. We analyzed both pre-dialysis and post-dialysis arterial blood pressure values. Pre-dialysis arterial blood pressure values >140/90 mmHg were present in 77.5% of patients, and among patients with AVF thrombosis, 69.2% had pre-dialysis pressure values of >140/90 mmHg. Those patients had nearly twice the significantly higher chance of developing AVF thrombosis compared to those with values of <140/90 mmHg. Siddiquia et al. also concluded that hypertension significantly impacts thrombosis development and weakens AVF function [17]. In contrast to our results, the cohort study by Kim et al. did not confirm the negative effect of arterial hypertension on AVF thrombosis development [18]. The hypothesis that arteriovenous thrombosis most often occurs in patients over 60 years of age was confirmed, as well as the finding that the dominant clinical parameters, such as DM and hyperlipidemia, are significant risk factors for thrombosis occurrence.

Conclusion

Significant predictors for arteriovenous fistula thrombosis development included a body mass index >25 kg/m², an increase in number of comorbidities, blood pressure >140/90 mmHg, and age group 45-59 years old. We found that the application of anticoagulant therapy before arteriovenous fistula development and angiotensin-converting enzyme inhibitors/angiotensin II receptor blockers after arteriovenous fistula development can significantly decrease the chance for arteriovenous fistula thrombosis development.

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