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Recurrent Intradialytic Hypotension Increases AV Fistula Failure Risk Ten-fold

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ABSTRACT

Background: Arteriovenous fistulas (AVFs) remain the preferred vascular access for hemodialysis (HD) owing to their superior patency and lower complication rates compared to other modalities. yet AVF failure remains a major cause of morbidity and increased healthcare costs.

Aim: This study aimed to identify clinical, cardiovascular, and laboratory risk factors associated with AVF failure in HD patients.

Patients and Methods: A cohort of 64 HD patients were retrospectively assessed for AVF failure risk factors. Patients were classified into two groups: AVF failure (n=26) and functioning AVFs (n=38). then AVF failure group was further subdivided into 2 subgroups (failure with a chance for the patient to have another AVF and failure without a chance to the patient to have another AVF). Data were collected and predictors of AVF failure were evaluated using univariate and multivariate logistic regression, and receiver operating characteristic (ROC) curve analysis.

Results: Intradialytic hypotension (IDH) was identified as the strongest independent predictor of AVF failure (OR = 9.7, p=0.001; AUC = 0.709, p=0.002) increasing the risk ten-fold. Additional significant risk factors included dialysis duration > 2 years and elevated hemoglobin and hematocrit levels. ROC analysis confirmed dialysis duration and IDH as strong predictors, with sensitivities of 88.5% and 65.4% respectively. Elevated white blood cell count and pre-dialysis blood urea nitrogen were also significant correlates.

Conclusion: IDH and dialysis duration were the most potent predictors of AVF failure, highlighting the necessity of close intradialytic BP monitoring to enhance vascular access survival. These findings warrant validation through larger multicenter studies to inform preventive strategies.

Keywords: Arteriovenous fistula, hemodialysis, AVF failure, intradialytic hypotension, predictors.

INTRODUCTION

Chronic kidney disease (CKD) is a major public health concern marked by progressively rising rates of illness and death [(1)].

For patients on hemodialysis (HD), the arteriovenous fistula (AVF) is the vascular access of choice, regarded as the gold standard over arteriovenous grafts and central venous catheters due to its better long-term patency, decreased infection risk, and association with lower mortality. Despite this, AVF dysfunction remains a significant problem in clinical practice, commonly presenting as either primary or secondary failure. Primary failure, means that the AVF does not mature sufficiently for dialysis, is seen in 20–50% of created fistulas. Secondary failure involves a loss of previously established patency and is a major source of access-related complications. These failures adversely affect patient morbidity and quality of life, in addition to placing a substantial financial strain on healthcare services [(2, 3)].

The etiology of AVF failure is multifaceted involving an interplay of clinical, cardiovascular, vascular, and systemic inflammatory pathways. Established clinical risk factors are older age, male gender, and the presence of comorbidities like diabetes mellitus (DM) and hypertension (HTN). DM and HTN promote endothelial dysfunction, vascular calcification, and increased arterial stiffness, all of which negatively impact the chances of AVF maturation. Moreover, the persistent inflammatory milieu in end-stage kidney disease (ESKD) worsens vascular damage, impairs endothelial function, and increases the propensity for thrombosis [(4, 5)].

Factors related to dialysis itself, such as a longer vintage and occurrences of intradialytic hypotension, also predispose the access to stenosis and thrombotic occlusion [(6)].

Cardiovascular changes in individuals with ESRD are now understood to be critical factors influencing AVF success. Chronic fluid overload leads to maladaptive cardiac remodeling, particularly left ventricular hypertrophy (LVH) and a rise in left ventricular mass (LVM). This reduces vascular compliance and causes hemodynamic instability, thereby impairing fistula functionality. Consistent evidence indicates that a high LVM is associated with poorer AVF maturation rates and a higher risk of thrombosis. Therefore, echocardiographic measures offer crucial prognostic information regarding how cardiac health affects AVF performance [(7, 8)].

The utilization of preoperative vascular ultrasound is now considered fundamental for evaluating the viability of an AVF and predicting its clinical success. This assessment focuses on critical parameters such as vessel

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diameter, intimal medial thickness (IMT), and the resistive index (RI). Elevated IMT and RI values are indicative of reduced vascular elasticity and increased resistance, which are risk factors for developing stenosis and thrombosis. The prognostic value of these ultrasound-derived metrics is well-documented in numerous studies, justifying their standard application in preoperative planning [9, 10].

Biochemical and hematologic markers also offer important clues regarding AVF prognosis. Anemia, a frequent complication in ESRD, impairs vascular repair mechanisms and increases susceptibility to thrombosis. Hemoglobin (Hb) and hematocrit (Hct) levels are therefore important predictors of access patency. Similarly, inflammatory markers such as elevated white blood cell (WBC) count and C-reactive protein (CRP) reflect systemic inflammation that contributes to endothelial dysfunction and vascular injury. Blood urea nitrogen (BUN), a measure of dialysis adequacy, has additionally been linked with AVF function and patient outcomes [11, 12].

Combining these multifaceted parameters from clinical, cardiovascular, vascular, and laboratory domains, into consolidated predictive models enhances the precision of forecasting AVF success. Sophisticated statistical techniques, notably multivariate logistic regression and receiver operating characteristic (ROC) curve analysis are employed to isolate independent risk variables and measure their predictive strength. These models facilitate personalized risk assessment, directing clinicians to implement tailored interventions for improving AVF results [13, 14]. Nevertheless, despite considerable research, the specific influence of certain factors, particularly IDH, is not yet fully elucidated. While many research linked post operative hypotension to AVF failure.

Therefore, this study was designed to conduct a comprehensive examination of the factors determining AVF success, with a specific focus on clinical, cardiovascular, vascular, and biochemical predictors. Especially intradialytic blood pressure Through the application of multivariate analytical methods and ROC curve assessment, we aimed to pinpoint independent prognostic factors and determine their diagnostic efficacy. It is expected that the results will advance the current knowledge of AVF pathophysiology and inform more effective strategies for managing vascular access in hemodialysis patients.

PATIENTS AND METHODS

Study Design and Participants: This retrospective cohort study was conducted in the Nephrology Unit, Department of Internal Medicine, Menoufia University, between August 2023 and March 2024.

Participants: A total of 64 adult patients (≥18 years) receiving maintenance HD via AVFs for at least six months were included. Patients with incomplete records or acute illness at the time of evaluation were excluded.

Definition of AVF Failure: AVF failure was defined according to the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines as the loss of access function that prevents its use for effective dialysis, most commonly due to thrombosis, stenosis, or mechanical dysfunction [15]. Failures were further categorized as:

- Non-severe/failure with a chance: Patients eligible for creation of a new AVF at another site.
- Severe/failure without any chance: Patients deemed unsuitable for new AVF creation at any vascular site.

This was determined by a multidisciplinary team of three nephrologists, three vascular surgeons, and two radiologists.

A detailed history-taking and complete clinical evaluation were performed for all patients. This was further verified using data extracted from patient records within the dialysis unit, which encompassed demographics (age and sex), comorbidities (such as diabetes mellitus, hypertension, and bone mineral disease), and dialysis-related parameters (including dialysis vintage, interdialytic weight gain, intradialytic hypertension, and IDH).

Intradialytic hypotension (IDH) was defined per the Kidney Disease Outcomes Quality Initiative (KDOQI) guidelines [15] as a reduction in systolic blood pressure (SBP) by ≥ 20 mmHg or a drop in mean arterial pressure (MAP) by ≥ 10 mmHg during a dialysis session, which was associated with symptoms necessitating clinical intervention (such as saline administration, reduction of ultrafiltration, or termination of dialysis). For the purposes of this research, **a patient was classified as having IDH if they had a minimum of two documented episodes per month,** as noted in the dialysis unit charts.

Laboratory Investigations: Collected values included complete blood count (hemoglobin, hematocrit, platelets and white blood cells), renal function markers (pre-/post-dialysis BUN, serum creatinine and electrolytes), bone mineral parameters (calcium, phosphorus and parathyroid hormone), ferritin, and serum albumin. Dialysis adequacy was evaluated using Kt/V.

Radiology: Echocardiography was done by cardiology consultant to assess left ventricular hypertrophy, diastolic dysfunction, and cavity size. Vascular ultrasonography (performed by a consultant radiologist) assessed carotid intima-media thickness and vascular indices of medium sized vessels.

Ethical approval: The Institutional Review Board of Menoufia University approved the study and all patients provided informed consent (Approval No.: 8/2023/INTM4-1). The study adhered to the Helsinki Declaration throughout its execution.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics, Version 28.0 (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was applied to assess data distribution normality. Continuous variables were presented as medians with their interquartile ranges (25th and 75th percentiles). Parametric tests were used for normally distributed data, while non-parametric alternatives were chosen for data that deviated from normality. Categorical variables were summarized as counts and percentages, with group comparisons made via the Chi-square test.

For inter-group comparisons, the Student's *t*-test or the Mann-Whitney U test was selected based on data characteristics. Receiver operating characteristic (ROC) curve analysis was utilized to evaluate the predictive power of different parameters for AVF failure. Univariate and subsequent multivariate logistic regression analyses were performed to pinpoint independent predictors of AVF failure. For all tests, a two-tailed p-value less than or equal 0.05 was considered statistically significant.

RESULTS

Analysis of patient demographics revealed no notable disparities in age, sex, or clinical history between those who experienced arteriovenous fistula (AVF) failure and those who did not implying that these factors are not primary determinants of failure. In contrast, dialysis-related characteristics showed that the failure group had a significantly extended dialysis history and a greater frequency of intradialytic hypotension identifying these as potential key factors in AVF dysfunction.

The remaining dialysis parameters were comparable across both cohorts.

Echocardiographic evaluation indicated that an enlarged left ventricular (LV) cavity was markedly more common in patients without AVF failure.

This observation could point toward compensatory cardiovascular adaptations in this group. Other echocardiographic measures, however, did not differ significantly, and ultrasonographic scans of the AVF itself also demonstrated no substantial variations between the groups. Laboratory analysis identified significantly elevated concentrations of hemoglobin (Hb) and hematocrit (Hct) in patients with AVF failure.

This association implies a possible involvement of these hematological factors in failure pathogenesis, perhaps mediated by changes in vascular hemodynamics or erythropoiesis. The specificity of this relationship is underscored by the lack of significant differences in all other laboratory values (Table 1).

Table (1): Comparison of patients' data between AVF patients with or without failure

Variable	AVF failure (n =26)	AVF no failure (n =38)	Statistical test	*P*-value
Age (years)	43 (24 – 60)	44 (27 – 59)	U =510	0.685
Sex				
- Male	12 (46.2%)	22 (57.9%)	χ^2 =0.45	0.447
- Female	14 (53.8%)	16 (42.1%)		
DM	4 (15.4%)	10 (26.3%)	χ^2 =1.08	0.299
HTN	18 (69.2%)	30 (78.9%)	χ^2 =0.78	0.378
BMD	15 (57.7%)	15 (39.5%)	χ^2 = 2.1	0.151
Resistant anemia	2 (7.7%)	1 (2.6%)	χ^2 =0.89	0.210
Dialysis data				
- Duration of dialysis (year)	10.5 (5 – 14)	3 (1 – 10)	U =283	**0.004***
- Intradialytic hypertension	3 (11.5%)	10 (26.3%)	χ^2 =2.1	0.347
- Intradialytic hypotension(IDH)	17 (65.4%)	9 (23.7%)	χ^2 =11.1	**<0.001***
- Interdialytic weight gain (kg)	3 (2 – 3.5)	3 (2 – 4)	U =358	0.828
- Residual renal function	7 (26.9%)	15 (39.5%)	χ^2 =1.1	0.299
Echo data				
- Increase in LV cavity size	0 (0%)	7 (63.6%)	χ^2 =8.1	**0.013***
- Diastolic dysfunction	6 (75%)	9 (81.8%)	χ^2 =0.1	1.000
- LVH	4 (50%)	8 (72.7%)	χ^2 =1.03	0.377
US data				
- Rt intimal medial thickness (mm)	0.07 (0.06 – 0.08)	0.08 (0.06 – 0.09)	U =365	0.508
- Lt intimal medial thickness (mm)	0.08 (0.06 – 0.08)	0.075 (0.06 – 0.1)	U =331	0.977
- PSV (cm/s)	83.5 (73 – 142)	115 (85 – 184)	U =419	0.099
- EDV (cm/s)	42 (29 – 77)	62 (40 – 83)	U =405	0.164
- RI	0.47 (0.33 – 0.6)	0.45 (0.4 – 0.53)	U =302	0.604
- PI	0.87 (0.62 – 1.5)	0.64 (0.53 – 0.97)	U =259	0.191
Laboratory data				
- Hb (g/dL)	10.1 (9.4 – 11.5)	9.4 (8.1 - 10.2)	U =249	**0.037***
- Hct (%)	32.2 (29.3 - 36.1)	30 (24 - 32.8)	U =243	**0.029***
- Plat $\times 10^3$ (cells/µL)	202.5 (127 - 260)	166 (127 - 220)	U =320	0.377
- WBCs $\times 10^3$ (cells/µL)	6.1 (4.7 - 8.1)	5.2 (4.7 - 6.2)	U =285	0.142
- AST (U/L)	20 (15 - 23.5)	15 (12 - 22)	U =299	0.217
- ALT (U/L)	15 (10 - 18)	14 (10.5 - 22)	U =374	0.966
- Albumin (g/dL)	4.3 (4.2 - 4.8)	4.2 (3.9 - 4.7)	U =57	0.333
- BUN pre dialysis (mg/dL)	50 (42 - 63)	55 (44 - 61)	t =-0.19	0.628
- BUN post dialysis (mg/dL)	23 (16 - 30)	21.5 (17 - 23)	U =291	0.231
- Serum creatinine (mg/dL)	8.3 (6.5 - 9.6)	9.3 (7.7 - 10.5)	t =-0.69	0.293
- K (mmol/L)	5.1 (4.7 - 5.6)	5.2 (4.8 - 5.5)	U =402	0.610
- Ca (mg/dL)	8.5 (7.6 - 9.4)	8.5 (7.9 - 9)	U =394	0.709
- Po (mg/dL)	4.4 (3.3 - 5)	4.7 (3.3 - 4.9)	t =0.13	0.779
- Na (mg/dL)	138 (135 - 139)	136 (134.5 - 138)	U =290	0.16
- Iron (mg/dL)	32.5 (20 - 56)	32 (31 - 41.5)	U =123	0.892
- Ferritin (ng/mL)	570 (250 - 1859)	485 (315 - 863.4)	U =275	0.665
- PTH (pg/mL)	756 (420 - 1685)	459 (273.2 - 970)	U =150	0.168
- Kt/V	1.3 (1.2 - 1.7)	1.2 (1 - 1.4)	U =87	0.341

Data is presented as median (IQR) or n (%), Data is presented as median (IQR) or n (%), **DM:** Diabetes mellites, **HTN:** Hypertension, **BMD:** Bone mineral disease, **RRF:** Residual renal function, **LV:** Left ventricle, **LVH:** Left ventricular hypertrophy, **US:** Ultrasound, **Rt:** Right**, Lt:** Left, **Hb:** Hemoglobin, **Hct:** Hematocrit, **Plat:** Platelets, **WBCs:** White blood cells, **AST:** Aspartate transaminase, **ALT:** Alanine transaminase, **BUN:** Blood urea nitrogen, **K:** Potassium, **Ca:** Calcium, **Po:** Phosphorus, **PTH:** Parathormone, *****Statistically significant as p value <0.05.

When assessing the potential for recovery among patients with AVF failure, a pronounced divergence in gender distribution was observed. Males predominated in the subgroup with a chance of recovery, whereas females were more prevalent in the group with no recovery prospect. This disparity may be attributable to underlying biological or anatomical gender differences affecting AVF outcomes. Conversely, age, and histories of DM, HTN, or resistant anemia were similar between these subgroups, as were all dialysis and ultrasonographic parameters. Further laboratory comparisons revealed significantly higher WBC counts and pre-dialysis BUN levels in patients with no chance of AVF recovery. These findings suggest that inflammatory states (evidenced by elevated WBCs) and heightened uremic toxicity (reflected by increased BUN) could be implicated in irreversible AVF failure. All other laboratory markers were consistent between the subgroups (Table 2).

Table (2): Comparison of patients' data between AVF failure patients with or without chance for another AVF

Variable	AVF failure with chance (n =19)	AVF failure without chance (n =7)	Statistical test	*P*-value
Age (years)	43 (27.5 - 61)	20 (20 - 53.5)	U =51	0.395
Sex: - Male	12 (63.2%)	0 (0%)	χ^2 =8.2	**0.004***
- Female	7 (36.8%)	7 (100%)		
DM	3 (15.8%)	1 (14.3%)	χ^2 =0.01	0.925
HTN	15 (78.9%)	3 (42.9%)	χ^2 =3.1	0.077
BMD	9 (47.4%)	6 (85.7%)	χ^2 =3.1	0.079
Resistant anemia	2 (10.5%)	0 (0%)	χ^2 =0.79	0.372
Dialysis data				
- Duration of dialysis (year)	11 (4 - 14)	10 (8 - 11)	U =66	1.000
- Intradialytic hypertension	2 (10.5%)	1 (14.3%)	χ^2 =0.07	0.790
- Intradialytic hypotension	13 (68.4%)	4 (57.1%)	χ^2 =0.29	0.592
- Interdialytic weight gain (kg)	2.8 (2 - 3.5)	3 (2.3 - 4.5)	U =62	0.360
- Residual renal function	5 (26.3%)	2 (28.6%)	χ^2 =0.01	0.908
US data				
- Rt intimal medial thickness (mm)	0.1 (0.1 - 0.1)	0.1 (0.1 - 0.1)	t =0.76	0.630
- Lt intimal medial thickness (mm)	0.1 (0.1 - 0.1)	0.1 (0.1 - 0.1)	U =41	0.447
- PSV (cm/s)	84 (73 - 108)	73 (64.5 - 143)	U =52	1.000
- EDV (cm/s)	45 (34 - 63.5)	39 (27.5 - 99.5)	U =55	0.891
- RI	0.5 (0.4 - 0.6)	0.5 (0.4 - 0.6)	t =-0.14	0.837
- PI	1 (0.6 - 1.7)	0.7 (0.6 - 1.1)	t =0.96	0.535
Laboratory data				
- Hb (g/dL)	10.2 (9.4 - 10.7)	10 (9.6 - 11.6)	t =-0.26	0.710
- Hct (%)	32.3 (29.3 - 34.3)	32 (31.2 - 36.1)	t =0.09	0.804
- Plat $\times 10^3$ (cells/µL)	200 (127 - 246)	205 (185 - 251.5)	t =-0.87	0.418
- WBCs $\times 10^3$ (cells/µL)	5.3 (4.6 - 6.4)	8.6 (7 - 8.7)	U =104.5	**0.003***
- AST (U/L)	20 (16 - 25)	18 (16 - 21)	U =51	0.619
- ALT (U/L)	13 (9 - 20)	18 (14 - 18)	U =69.5	0.534
- Albumin (g/dL)	4.2 (4.1 - 4.3)	4.8 (4.6 - 4.8)	U =12.5	0.143
- BUN pre dialysis (mg/dL)	43 (39 - 55)	60 (53 - 70.5)	t =-1.9	**0.024***
- BUN post dialysis (mg/dL)	23 (15 - 35)	23 (22 - 29)	U =60	1.000
- Serum creatinine (mg/dL)	8.4 (6.5 - 9.7)	7.9 (6.7 - 9.1)	t =0.44	0.804
- K (mmol/L)	4.9 (4.6 - 5.3)	5.6 (4.8 - 5.8)	U =76	0.318
- Ca (mg/dL)	8.4 (8 - 8.8)	9.4 (7.1 - 9.5)	t =-0.78	0.576
- Po (mg/dL)	4.4 (3.4 - 4.7)	3.8 (3.2 - 5.4)	t =-0.45	0.852
- Na (mg/dL)	138 (134 - 139)	138 (136.5 - 138.5)	U =58	0.951
- Iron (mg/dL)	30.3 (20 - 55)	48.5 (25 - 56)	U =27	0.770
- Ferritin (ng/mL)	570 (263.5 - 1831)	1054.5 (250 - 1902)	U =47	0.971
- PTH (pg/mL)	1086.5 (574.5 - 2135.5)	420 (315.6 - 942)	U =11	0.177
- Kt/V	1.3 (1.2 - 1.6)	1.5 (1.2 - 1.9)	U =13	0.769

Data is presented as median (IQR) or n (%), Data is presented as median (IQR) or n (%), **DM:** Diabetes mellites, **HTN:** Hypertension, **RRF:** Residual renal function, **LV:** Left ventricle, **LVH:** Left ventricular hypertrophy, **US:** Ultrasound, **Rt:** Right, **Lt:** Left, **Hb:** Hemoglobin, **Hct:** Hematocrit, **Plat:** Platelets, **WBCs:** White blood cells, **AST:** Aspartate transaminase, **ALT:** Alanine transaminase, **BUN:** Blood urea nitrogen, **K:** Potassium, **Ca:** Calcium, **Po:** Phosphorus, **PTH:** Parathormone, *****Statistically significant as p value <0.05.

Evaluation using receiver operating characteristic (ROC) curves revealed multiple factors predictive of AVF failure. Dialysis duration was a significant predictor, showing an area under the curve (AUC) of 0.713 (p = 0.001). A cut-off of more than two years yielded a sensitivity of 88.5% and a specificity of 50%, highlighting the substantial risk associated with prolonged dialysis. Intradialytic hypotension was also a significant predictor (AUC: 0.709, p = 0.002), with 65.4% sensitivity and 76.3% specificity. Furthermore, a normal left ventricular (LV) cavity size, lacking enlargement, was a strong predictor of failure (AUC: 0.818, p = 0.001), with 100% sensitivity and 63.6% specificity, implying a potential protective cardiovascular effect. Significant laboratory predictors included hemoglobin (Hb) (AUC: 0.655, p = 0.026) and hematocrit (Hct) (AUC: 0.673, p = 0.018), with respective cut-offs of >8.8 g/dL and >30.4% (Table 3**).** These results emphasize the value of tracking these variables for the early detection of high-risk individuals.

Table (3): ROC curve analysis of factors that can predict AVF failure

Variable	Cut-off value	Sensitivity	Specificity	AUC	P value
Duration of dialysis (year)	>2	88.5%	50%	0.713	<0.001*
Intradialytic hypotension	Yes	65.4%	76.3%	0.709	<0.002*
Increase in LV cavity size	No	100%	63.6%	0.818	<0.001*
Hb (g/dL)	>8.8	87.5%	38.8%	0.665	<0.026*
Hct (%)	>30.4	66.7%	64.5%	0.673	<0.018*

Hb: Hemoglobin, Hct: Hematocrit, *Statistically significant as p value <0.05.

The duration of dialysis is a significant predictor (AUC: 0.713, p = 0.001), with a sensitivity of 88.5% and specificity of 50% at a cut-off value of >2 years. Intradialytic hypotension is a significant predictor (AUC: 0.709, p = 0.002), demonstrating a sensitivity of 65.4% and specificity of 76.3%. The absence of increased left ventricular (LV) cavity size is also a strong predictor (AUC: 0.818, p = 0.001), with a sensitivity of 100% and specificity of 63.6%. Hemoglobin (Hb) and hematocrit (Hct) levels are additional significant predictors, with respective AUC values of 0.655 (p = 0.026) and 0.673 (p = 0.018), and cut-off values of >8.8 g/dL and >30.4% (figure 1).

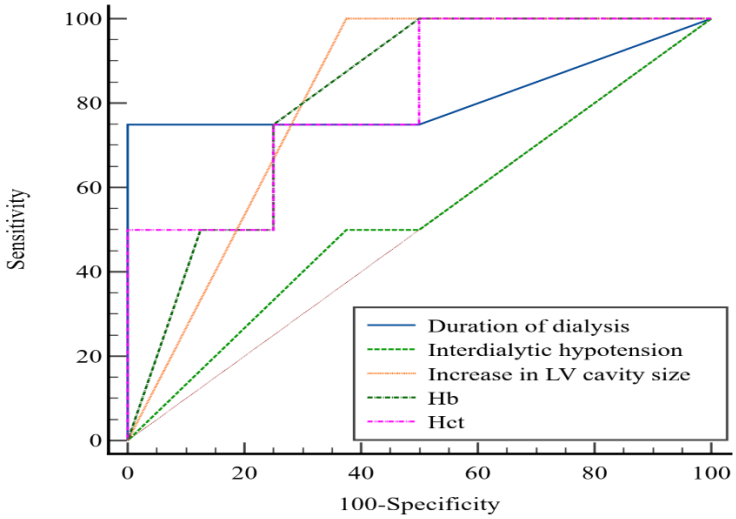

Figure (1): Receiver Operating Characteristic (ROC) curve analysis showing predictive factors for arteriovenous fistula (AVF) failure.

In patients with irreversible AVF failure, WBC counts and pre-dialysis BUN were key predictive factors. According to ROC analysis, WBC count was a strong predictor (AUC: 0.878, p < 0.001). A threshold of >7.3 × 10^3 cells/µL provided a sensitivity of 71.4% and a specificity of 88.2%, underscoring the involvement of a marked inflammatory state in irrecoverable failure. Pre-dialysis BUN was also a significant predictor (AUC: 0.461, p = 0.001). At a cut-off of >46 mg/dL, it achieved 100% sensitivity and 64.7% specificity, indicating that a high uremic load profoundly affects vascular integrity and AVF prognosis (Table 4).

Table (4): ROC curve analysis of factors that can predict AVF failure without chance for another AVF

Variable	Cut-off value	Sensitivity	Specificity	AUC	*P*-value
WBCs $\times 10^3$ (cells/µL)	>7.3	71.4%	88.2%	0.878	<0.001*
BUN pre dialysis (mg/dL)	>46	100%	64.7%	0.794	0.001*

WBCs: White blood cells, BUN: Blood urea nitrogen, *Statistically significant as p value <0.05.

White blood cell (WBC) count is a significant predictor (AUC: 0.878, $p < 0.001$), with a sensitivity of 71.4% and specificity of 88.2% at a cut-off value of >7.3 × 10^3 cells/µL. Pre-dialysis blood urea nitrogen (BUN) level is another significant predictor (AUC: 0.461, $p = 0.001$), demonstrating a sensitivity of 100% and specificity of 64.7% at a cut-off value of >46 mg/dL. These findings emphasize the role of systemic inflammation and uremic burden in irreversible AVF failure (Figure 2).

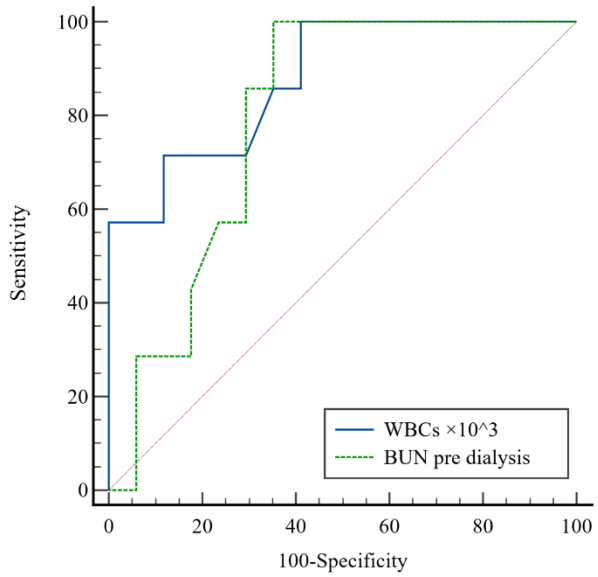

Figure (2): Receiver Operating Characteristic (ROC) curve analysis illustrating predictive factors for arteriovenous fistula (AVF) failure without chance of recovery.

Univariate logistic regression confirmed several factors as significant predictors of AVF failure, namely dialysis duration (OR: 1.13, $p = 0.006$), intradialytic hypotension (OR: 6.08, $p = 0.002$), hemoglobin (Hb) (OR: 1.45, $p = 0.031$), and hematocrit (Hct) (OR: 1.1, $p = 0.029$). Subsequent multivariate analysis solidified intradialytic hypotension as the foremost independent predictor of failure (OR: 9.7, $p = 0.001$), highlighting the imperative for meticulous management of blood pressure stability throughout dialysis (Table 5).

Table (5): Univariate and multivariate logistic regression of factors that can predict AVF failure

Variable	Univariate regression		Multivariate regression	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Duration of dialysis (year)	1.13 (1.02 – 1.24)	0.006*	1.06 (0.94 – 1.18)	0.344
Intradialytic hypotension	6.08 (2.02 – 18.3)	0.002*	9.7 (2.51 – 37.8)	0.001*
Hb (g/dL)	1.45 (1.02 – 2.09)	0.031*	1.19 (0.63 – 2.23)	0.593
Hct (%)	1.1 (1.01 – 1.21)	0.029*	1.02 (0.87 – 1.18)	0.821

Hb: Hemoglobin, Hct: Hematocrit, *Statistically significant as p value <0.05.

In cases of AVF failure deemed irrecoverable, univariate regression analysis identified WBCs count as a significant predictor (OR: 2.54, $p = 0.016$), reinforcing the role that systemic inflammation plays an unfavorable, permanent outcomes (Table 6).

Table (6): Univariate logistic regression of factors that can predict AVF failure without chance for another AVF

Variable	Univariate regression	
	OR (95% CI)	***P*-value**
WBCs $\times 10^3$ (cells/µL)	2.54 (1.19 – 5.45)	**0.016** *
BUN pre dialysis (mg/dL)	1.06 (0.99 – 1.14)	0.076

WBCs: White blood cells, BUN: Blood urea nitrogen, *Statistically significant as p value <0.05

DISCUSSION

Arteriovenous fistulas (AVFs) remain the gold standard for vascular access in hemodialysis (HD) patients because of their superior performance compared to grafts and central venous catheters. They are associated with longer patency, lower infection rates, fewer complications, and improved patient survival. Moreover, AVFs are linked with reduced hospitalization and better quality of life, supporting their preference as the access of choice for HD worldwide. Global prevalence studies report that AVF utilization ranges from 49% to 92% among dialysis-dependent patients, reflecting their widespread acceptance [16, 17].

Despite these advantages, AVF failure remains a critical concern, affecting 51–60% of patients over the course of treatment. Such failures contribute to increased morbidity, extended hospitalizations, repeated interventions, and higher healthcare expenditures [18].

The pathophysiology of AVF failure is complex and multifactorial. The most widely accepted model suggests that after AVF creation, several factors including thrombus formation, uremic toxins, hemodynamic shear stress, hypoxia, and systemic inflammation contribute to endothelial dysfunction. These factors alter gene and protein expression, stimulating neointimal hyperplasia, which thickens the vascular wall and leads to stenosis. This progressive narrowing disrupts blood flow and increases the risk of occlusion and access loss [19]. Neointimal hyperplasia is considered the central mechanism driving AVF dysfunction.

we looked for controllable risk factors that we can manage in day after day follow up of our patients. We aimed to identify clinical, cardiovascular, and laboratory risk factors associated with AVF failure in HD patients.

Sex-related differences in AVF outcomes have also been extensively studied. Multiple reports confirm that female sex is associated with a higher risk of AVF failure [20-23]. In our cohort, gender distribution significantly differed between subgroups: Men predominated in the failure group with a chance of recovery, whereas women were more frequent in the group without recovery. A commonly cited explanation is the smaller vessel caliber in women, which may predispose them to poorer outcomes. However, retrospective studies have not consistently shown significant differences in vessel diameter between sexes [24]. In our study, females more frequently underwent upper-arm AVF placement, likely reflecting a clinical strategy to overcome potential vessel size limitations. Nonetheless, the absence of standardized preoperative vascular imaging limits firm conclusions regarding the role of vessel caliber in these outcomes.

Our findings also demonstrated that intradialytic hypotension (IDH) was an independent predictor of AVF failure (OR: 9.7, p = 0.001). This is consistent with previous studies linking low perioperative blood pressure to increased risk of thrombosis and early primary AVF failure [25].

Pandey *et al.* [25] prospectively followed 224 patients and reported significantly lower diastolic blood pressure (DBP) in those with early AVF failure (88.4 mmHg) compared to successful cases (91.2 mmHg). Similarly, a large retrospective cohort of 1051 patients identified lower DBP in early AVF failure (79.7 mmHg) versus successful outcomes (83.1 mmHg) [26]. Our data suggests that maintaining higher DBP and mean arterial pressure (MAP) may be protective, as low DBP can promote venous stasis, reduce vascular compliance, and increase thrombotic risk. Additionally, low DBP may correspond to wider pulse pressure, which has been associated with impaired vascular remodeling and decreased vessel compliance [27].

These findings highlight the importance of careful blood pressure management during the dialysis sessions after successful AVF maturation, though prospective trials are needed to establish optimal targets.to the most of our knowledge we are the first to clear this strong relationship between intradialytic hypotension and AVF failure lighting the road for a preventive factor in managing AVF of ESRD patients.

We also observed significant associations between AVF failure and elevated WBC counts as well as higher pre-dialysis BUN levels. These results point to the contribution of systemic inflammation and impaired renal function in predicting AVF dysfunction. Prior studies similarly linked inflammation and malnutrition to reduced AVF survival, emphasizing the importance of optimizing systemic health prior to access creation [15].

Echocardiographic analysis revealed that patients without AVF failure more frequently exhibited enlarged left ventricular (LV) cavity size compared to those with failure. This may suggest compensatory cardiovascular mechanisms in the non-failure group. Also, this may point that the patients with enlarged left ventricular cavity size could be linked to elevated blood pressure and this is in consistence with the theory of linking of hypotension with AVF failure. Moreover, preoperative vascular ultrasound assessing arterial and venous calibers has been shown to improve AVF success rates by optimizing vessel selection

[28-30]. Postoperative surveillance strategies, although variably effective, may still play a role in early identification of access dysfunction [31]. Incorporating these cardiovascular and vascular parameters into predictive models could refine risk stratification and clinical decision-making.

While AVF remains the preferred access, alternative modalities should also be considered when indicated. Options such as arteriovenous grafts [32], peritoneal dialysis, or tunneled dialysis catheters may be appropriate in patients with poor vascular anatomy, prior failed AVFs, limited life expectancy, or personal preference [1]. An individualized approach ensures that vascular access decisions align with patient-specific factors and optimize clinical outcomes. Peritoneal dialysis centers should be increased at least one in every governorate as this may be the only solution remaining for some ESRD patients.

In summary, our study identified multiple predictors of AVF failure, including frequent IDH episodes, elevated Hb and Hct levels, dialysis duration exceeding two years, increased inflammatory markers, and impaired renal function. Strategies such as optimizing dialysis sessions blood pressure, frequent monitoring of blood pressure during dialysis sessions and rapid correction of intradialytic hypotension specially for ESRD patients with a history of IDH or at risk for developing IDH are mandatory to enhance AVF survival. Also strict management to antihypertensive drugs at day of dialysis session will be of great benefit. Managing all factors that may increase systemic inflammation may help improve AVF outcomes. Future multicenter studies with larger cohorts are warranted to confirm these associations and to develop effective preventive interventions.

CONCLUSIONS

Intradialytic hypotension is the strongest independent predictor of AVF failure, alongside dialysis duration, elevated Hb/Hct, WBC counts, and pre-dialysis BUN. Early identification and proactive management of these risk factors may improve AVF survival and patient outcomes. Larger multicenter studies are warranted to validate these findings.

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