Natural Resources for Human Health
eISSN: 2583-1194

Original Research

**Received** 08 May 2026
**Revised** 19 June 2026
**Accepted** 03 July 2026

Natural Resources for Human Health 2026, Vol. 6 (11s): 628–636

**KEYWORDS:**
hemodialysis; vascular access; arteriovenous fistula; thrombosis; stenosis; access failure; recurrence; Saudi Arabia

# Who Develops Recurrent Hemodialysis Vascular Access Failure? Clinical, Biochemical, and Access-Related Determinants in a Multicenter Cohort

## Recurrent Hemodialysis Vascular Access Failure

Mohammed Yousef Almulhim (MD)[1]

[1] Department of Internal Medicine, College of Medicine, King Faisal University, Al-Ahsa, Saudi Arabia, ORCID: 0009-0001-6347-4743,
E-mail: malmulhim@kfu.edu.sa
Corresponding author: Mohammed Yousef Almulhim, MD; Department of Internal Medicine, College of Medicine, King Faisal University, Al-Ahsa, Saudi Arabia;
E-mail: malmulhim@kfu.edu.sa

**ABSTRACT:**

**Background:** Recurrent vascular access failure imposes repeated procedures and threatens continuity of hemodialysis, yet recurrence phenotypes are less well characterized than single access failure.

**Methods:** We performed a retrospective secondary cohort analysis of 381 adults receiving maintenance hemodialysis at two centers in Al-Ahsa, Saudi Arabia. Recurrent failure/immaturation was defined a priori as at least two documented access failure or immaturation episodes. Modified Poisson regression with robust variance estimated adjusted risk ratios (aRRs). Biochemical associations were examined in prespecified center-, age-, and sex-adjusted models with false-discovery-rate correction.

**Results:** Recurrent failure/immaturation occurred in 38 patients (10.0%; 95% CI 7.4%-13.4%). Among 102 patients with any failure history, 37.3% had recurrence. Thrombosis was recorded in 63.2% of recurrent cases and stenosis in 47.4%. Treatment center was associated with recurrence in the primary model (aRR 2.62, 95% CI 1.19-5.79; p=0.017), but this association weakened in sensitivity analyses. No biochemical marker remained associated after multiplicity correction.

**Conclusions:** Recurrent access failure affected one in ten patients and was dominated by thrombotic and stenotic mechanisms. Recurrence should trigger structured access review and documentation, while center-level variation requires prospective validation before being interpreted as a quality signal.

## INTRODUCTION

Reliable vascular access is essential for maintenance hemodialysis, but access durability remains limited by maturation failure, stenosis, thrombosis, infection, and the need for repeated interventions. Contemporary guidance emphasizes that vascular access should be managed as part of an individualized kidney failure Life-Plan, with explicit contingency and succession strategies when an access becomes dysfunctional [1-3].

Arteriovenous fistulas (AVFs) generally offer favorable long-term infection and patency profiles once successfully matured, but initial nonmaturation and later dysfunction are common. A large systematic review reported substantial fistula

abandonment and incomplete functional maturation, illustrating that creation of an AVF does not guarantee durable clinical use [4].

Stenosis and thrombosis remain the dominant flow-related mechanisms of arteriovenous access dysfunction, and recurrence after successful salvage can lead to a cycle of angioplasty, thrombectomy, revision, catheter exposure, and eventual access abandonment [1,3,5].

Most vascular-access studies classify patients according to a single episode of primary failure, current patency, or time to a first intervention. This approach can obscure a clinically important subgroup: patients who experience repeated episodes of failure or immaturation. Recurrent events may reflect persistent anatomic lesions, unfavorable access configuration, low-flow states, cannulation-related injury, systemic vascular disease, or unmeasured center-level processes. Recent evidence also suggests that a history of multiple prior AVF failures is itself an important marker of subsequent thrombosis risk [6].

Risk-factor literature is heterogeneous. Systematic reviews and meta-analyses have implicated age, sex, diabetes, vessel diameter, fistula location, low blood flow, albumin, and other patient or access factors in primary AVF failure or thrombosis [7-10]. However, published prediction models frequently have methodological limitations, including small event counts, inconsistent outcome definitions, and lack of external validation [11]. Evidence from Saudi Arabia is particularly limited.

The underlying Al-Ahsa cohort has previously been used to describe overall access patterns, current patency/failure, complications, and barriers to AVF/AVG establishment [12]. The present analysis addresses a distinct question by using the documented number of historical access failure/immaturation episodes to identify a recurrent-failure phenotype and by examining clinical, biochemical, and access-related factors associated with recurrence.

The objectives were to quantify the burden of recurrent vascular access failure/immaturation, characterize the mechanisms recorded in recurrent cases, identify independently associated clinical and access-related factors, and explore biochemical markers while explicitly accounting for multiplicity and the temporal limitations of retrospective laboratory data.

## METHODS

### Study design and setting

This was a retrospective multicenter secondary cohort analysis of adults receiving maintenance hemodialysis at Almoosa Specialist Hospital and Aljaber Kidney Center in Al-Ahsa, Eastern Province, Saudi Arabia. The underlying data were collected during 2024 and included demographic, clinical, biochemical, echocardiographic, and vascular-access variables. Reporting was guided by the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement [13].

### Relationship to the previous cohort report

The source cohort was previously analyzed to describe current vascular-access types and current access outcomes [12]. That report analyzed 378 patients because three records lacked the current access-type information required for that analysis.

The updated source files available for the present secondary analysis contained 381 records; the three additional records had documented historical failure/immaturation episode counts and were therefore informative for the recurrence outcome. The current analysis uses a different outcome definition, hypothesis, and statistical framework from the previous publication.

### Participants and outcome definition

All 381 adults in the updated files had an available value for the number of documented access failure or immaturation episodes and were included in the primary analysis.

The primary outcome was recurrent vascular access failure/immaturation, defined a priori as at least two documented episodes. Values recorded as 'multiple times' were classified as recurrent because they unambiguously represented more than one episode, but were not assigned an exact numerical count. A history of any failure/immaturation was defined as at least one episode. Patients with no recorded episode constituted the reference group for the primary cohort analysis.

This outcome was intentionally broader than current access failure. It captured repeated historical dysfunction across the patient's access course, including failure and immaturation, and did not require the current access to be dysfunctional at the time of data abstraction.

**Failure mechanisms**

Recorded free-text causes were harmonized into non-mutually-exclusive mechanism categories: thrombosis, stenosis, immaturation, venous hypertension, aneurysm/pseudoaneurysm, infection, other specified mechanisms, and unknown/unspecified. Because a patient could have more than one mechanism documented, percentages across mechanism categories can exceed 100%.

**Candidate clinical and access-related variables**

The primary multivariable model was deliberately parsimonious because only 38 recurrent events were observed. Six variables were specified on clinical grounds before model fitting: age (per 10-year increase), sex, diabetes mellitus as the primary etiology of end-stage kidney disease (ESKD), cardiovascular comorbidity, catheter versus arteriovenous access as the first documented dialysis access, and treatment center. Cardiovascular comorbidity was identified from documented ischemic heart disease, coronary disease, heart failure, or cardiomyopathy.

Current access status was not entered into the etiologic model because recurrent failure can itself lead to current dysfunction or access abandonment, creating substantial risk of reverse causation.

**Biochemical variables**

Available biochemical and physiologic variables included hemoglobin category, albumin, creatinine, total cholesterol, triglycerides, calcium, phosphorus, parathyroid hormone (PTH), ferritin, and left ventricular ejection fraction. Hemoglobin entries were harmonized as below 12 g/dL versus within/above the center's documented target. Creatinine, PTH, and ferritin were log2-transformed for regression because of right-skewed distributions. Ejection fraction values stored as proportions were converted to percentage units for presentation.

Because laboratory measurements could not be confirmed to precede historical access failures in every patient, biochemical analyses were explicitly treated as exploratory associations rather than causal predictors.

**Statistical analysis**

Continuous variables were summarized using medians and interquartile ranges (IQRs), and categorical variables using frequencies and percentages. Recurrent-event prevalence was reported with a Wilson 95% confidence interval. Unadjusted group comparisons used the Mann-Whitney U test or Fisher exact test as appropriate.

Modified Poisson regression with robust sandwich variance was used for the primary multivariable analysis to estimate adjusted risk ratios (aRRs) and 95% confidence intervals directly [14]. No automated stepwise selection was performed. For the exploratory biochemical screen, each biomarker was assessed in a separate modified Poisson model adjusted for age, sex, and center. The Benjamini-Hochberg false discovery rate (FDR) procedure was applied across the ten biochemical tests; both nominal p-values and FDR-adjusted q-values were considered.

Two sensitivity analyses were prespecified. First, records described only as 'multiple times' were excluded to test dependence on qualitative episode coding. Second, analysis was restricted to patients with at least one documented failure/immaturation episode, comparing recurrent versus single-event cases. Statistical tests were two-sided.

**RESULTS**

**Cohort and recurrence burden**

The analytic cohort included 381 patients. Overall, 279 (73.2%) had no documented failure/immaturation episode, 64 (16.8%) had one episode, and 38 (10.0%; 95% CI 7.4%-13.4%) met the definition of recurrent failure/immaturation. Thus, among the 102 patients with any documented failure history, 38 (37.3%; 95% CI 28.5%-46.9%) had recurrence.

The recurrent group was older on unadjusted comparison (median 63.5 years, IQR 54.5-72.0) than the nonrecurrent group (59.0 years, IQR 42.5-69.0; p=0.047). Recurrent cases also had higher median creatinine, calcium, and ferritin in unadjusted analyses; these associations were substantially attenuated after adjustment and multiplicity correction.

Recurrent failure was documented in 19 of 106 patients at Almoosa (17.9%) and 19 of 275 at Aljaber (6.9%). This center difference was investigated in adjusted and sensitivity analyses rather than interpreted as a direct measure of care quality.

**Table 1.** Clinical and biochemical characteristics according to recurrent vascular access failure/immaturation

| Characteristic | No recurrence (n=343) | Recurrent (n=38) | Unadjusted p |
|---|---|---|---|
| Age, years, median (IQR) | 59.0 (42.5-69.0) | 63.5 (54.5-72.0) | 0.047 |
| Female sex | 132 (38.5%) | 17 (44.7%) | 0.486 |
| Diabetes as primary ESKD etiology | 242 (70.6%) | 29 (76.3%) | 0.572 |
| Cardiovascular comorbidity | 86 (25.1%) | 11 (28.9%) | 0.563 |
| First dialysis access was a catheter | 240 (70.0%) | 24 (63.2%) | 0.458 |
| Almoosa treatment center | 87 (25.4%) | 19 (50.0%) | 0.002 |
| Hemoglobin <12 g/dL | 242 (70.6%) | 30 (78.9%) | 0.346 |
| Albumin, g/L, median (IQR) | 36.4 (33.6-38.9) | 35.8 (33.5-37.5) | 0.526 |
| Creatinine, umol/L, median (IQR) | 342.3 (230.3-552.3) | 451.9 (269.3-783.3) | 0.019 |
| Cholesterol, mmol/L, median (IQR) | 3.67 (3.05-4.46) | 3.28 (2.84-4.08) | 0.055 |
| Triglycerides, mmol/L, median (IQR) | 1.27 (0.90-1.88) | 1.46 (0.73-1.77) | 0.550 |
| Calcium, mmol/L, median (IQR) | 2.10 (2.00-2.26) | 2.20 (2.10-2.31) | 0.005 |
| Phosphorus, mmol/L, median (IQR) | 1.60 (1.22-1.97) | 1.46 (1.18-1.91) | 0.269 |
| PTH, pmol/L, median (IQR) | 50.7 (27.2-89.8) | 46.9 (26.7-79.9) | 0.757 |
| Ferritin, ng/mL, median (IQR) | 466.8 (235.8-844.0) | 650.4 (404.0-1179.6) | 0.038 |
| Ejection fraction, %, median (IQR) | 55 (50-55) | 55 (50-55) | 0.772 |

IQR, interquartile range; ESKD, end-stage kidney disease; PTH, parathyroid hormone. P-values are unadjusted and descriptive; biochemical inference is based on the multiplicity-adjusted models in Table 4.

**Failure and immaturation mechanisms**

Among all 102 patients with at least one failure/immaturation episode, thrombosis was documented in 43 (42.2%), stenosis in 39 (38.2%), immaturation in 13 (12.7%), venous hypertension in 7 (6.9%), and aneurysm/pseudoaneurysm in 6 (5.9%).

The recurrent phenotype was particularly thrombotic: thrombosis was recorded in 24 of 38 recurrent cases (63.2%) compared with 19 of 64 single-event cases (29.7%; p=0.002). Stenosis was also common in recurrent cases (47.4%), although its frequency did not differ significantly from single-event cases.

As expected, a history of recurrence was overrepresented among patients whose current access was documented as dysfunctional or failed: 15 of 29 (51.7%) had recurrent episodes compared with 21 of 255 (8.2%) whose current access was

documented as well functioning. Current status was not modeled as a determinant because it may lie downstream of prior recurrent failure.

**Table 2.** Recorded mechanisms among patients with any access failure/immaturation history (n=102)

| Mechanism | Single event (n=64) | Recurrent (n=38) | p |
|---|---|---|---|
| Thrombosis | 19 (29.7%) | 24 (63.2%) | 0.002 |
| Stenosis | 21 (32.8%) | 18 (47.4%) | 0.206 |
| Immaturation | 7 (10.9%) | 6 (15.8%) | 0.545 |
| Venous hypertension | 4 (6.3%) | 3 (7.9%) | 1.000 |
| Aneurysm/pseudoaneurysm | 3 (4.7%) | 3 (7.9%) | 0.668 |
| Infection | 1 (1.6%) | 0 | 1.000 |
| Other specified | 2 (3.1%) | 2 (5.3%) | 0.627 |
| Unknown/unspecified | 13 (20.3%) | 4 (10.5%) | 0.275 |

Mechanism categories are non-mutually-exclusive because multiple mechanisms could be recorded for one patient; column percentages therefore do not sum to 100%.

**Clinical and access-related factors associated with recurrence**

In the prespecified modified Poisson model, treatment center was the only variable with evidence of an independent association with recurrent failure/immaturation. After adjustment for age, sex, diabetic ESKD, cardiovascular comorbidity, and first-access type, treatment at Almoosa was associated with a 2.62-fold higher recorded recurrence risk than treatment at Aljaber (aRR 2.62, 95% CI 1.19-5.79; p=0.017).

Age, sex, diabetes as the primary ESKD etiology, cardiovascular comorbidity, and catheter-based dialysis initiation were not independently associated with recurrence. Because center differences can reflect documentation intensity, referral patterns, access complexity, or residual case mix, this association was treated as a contextual signal rather than a causal center effect.

**Table 3.** Prespecified multivariable model for recurrent vascular access failure/immaturation

| Variable | Adjusted risk ratio | 95% CI | p |
|---|---|---|---|
| Age, per 10-year increase | 1.12 | 0.90-1.40 | 0.318 |
| Female sex | 1.14 | 0.61-2.15 | 0.679 |
| Diabetes as primary ESKD etiology | 0.88 | 0.40-1.93 | 0.743 |
| Cardiovascular comorbidity | 0.77 | 0.39-1.54 | 0.459 |
| First dialysis access was a catheter | 1.15 | 0.53-2.52 | 0.724 |
| Almoosa vs Aljaber center | 2.62 | 1.19-5.79 | 0.017 |

Modified Poisson regression with robust variance. ESKD, end-stage kidney disease.

**Exploratory biochemical associations**

No biochemical marker retained evidence of association after FDR correction. A nominal inverse association was observed for cholesterol (aRR 0.75 per 1 mmol/L increase; p=0.036), and calcium showed a borderline positive association (aRR 1.14 per 0.1 mmol/L increase; p=0.075), but the corresponding FDR q-values were 0.355 and 0.373. Hemoglobin below 12 g/dL, albumin, creatinine, triglycerides, phosphorus, PTH, ferritin, and ejection fraction were not associated after adjustment for age, sex, and center.

These results indicate that the unadjusted differences in creatinine, calcium, and ferritin should not be interpreted as independent biochemical determinants of recurrent access failure.

**Table 4.** Exploratory biochemical associations with recurrent access failure/immaturation

| Biochemical factor | n | Adjusted RR | 95% CI | p | FDR q |
|---|---|---|---|---|---|
| Hemoglobin <12 g/dL | 381 | 1.39 | 0.67-2.91 | 0.380 | 0.636 |
| Albumin, per 5 g/L higher | 379 | 1.10 | 0.81-1.49 | 0.541 | 0.750 |
| Creatinine, per doubling | 380 | 1.32 | 0.79-2.21 | 0.287 | 0.636 |
| Cholesterol, per 1 mmol/L higher | 371 | 0.75 | 0.58-0.98 | 0.036 | 0.355 |
| Triglycerides, per 1 mmol/L higher | 366 | 0.85 | 0.58-1.23 | 0.382 | 0.636 |
| Calcium, per 0.1 mmol/L higher | 378 | 1.14 | 0.99-1.33 | 0.075 | 0.373 |
| Phosphorus, per 0.5 mmol/L higher | 379 | 0.98 | 0.73-1.33 | 0.904 | 0.904 |
| PTH, per doubling | 371 | 1.12 | 0.89-1.39 | 0.330 | 0.636 |
| Ferritin, per doubling | 378 | 1.06 | 0.84-1.32 | 0.629 | 0.750 |
| Ejection fraction, per 10 percentage-point higher | 264 | 1.07 | 0.79-1.43 | 0.675 | 0.750 |

Each biomarker was modeled separately and adjusted for age, sex, and treatment center. Creatinine, PTH, and ferritin were log2-transformed. FDR q-values use the Benjamini-Hochberg procedure across the 10 biochemical tests. PTH, parathyroid hormone; RR, risk ratio.

### Sensitivity analyses

Two records had episode frequency documented qualitatively as 'multiple times.' When these records were excluded, 36 of 379 patients met the recurrent definition. The center association attenuated to aRR 2.17 (95% CI 0.96-4.92; p=0.064), while other clinical covariates remained non-significant.

When analysis was restricted to the 102 patients with at least one documented failure/immaturation episode, comparing recurrent (n=38) with single-event (n=64) cases, the center association was also attenuated (aRR 1.49, 95% CI 0.80-2.78; p=0.209). These analyses support cautious interpretation of center-level differences.

## DISCUSSION

### Principal findings

This multicenter secondary analysis identifies recurrent vascular access failure/immaturation as a clinically important phenotype affecting approximately one in ten patients in the overall hemodialysis cohort and more than one third of patients

who had experienced any access failure. The recurrent phenotype was dominated by thrombosis and stenosis. Thrombosis was more than twice as frequent among recurrent than single-event cases, supporting a mechanistic pattern in which repeated flow-limiting lesions and thrombotic events contribute disproportionately to recurrent access morbidity.

The analysis also demonstrates why recurrence should not be reduced to a single laboratory abnormality. Although recurrent cases had higher creatinine, calcium, and ferritin in unadjusted comparisons, none of the biochemical associations survived center adjustment and FDR correction. The strongest primary-model signal was center-level variation, but its attenuation in sensitivity analyses argues against interpreting this as evidence of a causal institutional effect.

**Recurrent failure as a distinct vascular-access phenotype**

Vascular-access research has historically emphasized first failure, nonmaturation, primary patency, or current access status. These outcomes are clinically important, but they may not identify patients entering a cycle of repeated salvage procedures and access loss. Contemporary reviews emphasize that recurrent stenosis and thrombosis are common threats to arteriovenous access durability and require an explicit contingency and succession plan [1,3,5].

The finding that thrombosis occurred in 63% of recurrent cases is consistent with the central role of stenosis-thrombosis pathways in access failure. A 2024 systematic review identified multiple patient and access characteristics associated with dialysis-access thrombosis, including graft access, age, diabetes, access location, and low access flow [8]. More recently, a 2026 prediction study of mature autogenous AVFs identified a history of two or more prior AVF failures as a predictor of subsequent thrombosis, directly supporting the clinical relevance of recurrent-event history as a risk marker [6].

**Clinical and biochemical factors**

Age, sex, diabetes, cardiovascular comorbidity, and initial catheter use did not independently distinguish patients with recurrent failure in the present cohort. This does not imply that these factors are irrelevant to vascular-access outcomes. Rather, the risk factors for initial AVF maturation, thrombosis, restenosis, and repeated failure are not necessarily identical.

Recent meta-analyses of primary AVF failure report associations with female sex, diabetes, low albumin, distal fistula location, and smaller vessel diameter [7], while restenosis after angioplasty has been associated with diabetes, upper-arm fistula location, longer stenotic lesions, and multiple lesions [10]. The current dataset did not contain detailed vessel diameter, lesion length, access flow, serial imaging, cannulation technique, or intervention-level data. These unmeasured access characteristics may be more proximal to recurrence than the general demographic variables available here.

The biochemical findings should also be interpreted cautiously. Current laboratory values may reflect nutritional status, dialysis adequacy, inflammation, mineral-bone disorder, or treatment intensity, but the retrospective dataset does not establish that each measurement preceded the recurrent access events. Our use of center-adjusted models and FDR correction therefore intentionally favors conservative interpretation. The nominal cholesterol and calcium associations should be viewed as hypothesis-generating rather than clinically actionable.

**Center-level variation**

Recorded recurrence was more frequent at Almoosa than Aljaber in the primary model. Several explanations are possible, including differences in documentation completeness, referral of complex access cases, case mix, access surveillance, procedural thresholds, or true differences in recurrent-event burden. The association weakened when qualitative 'multiple times' entries were removed and when the analysis was conditioned on having experienced at least one failure. Accordingly, the present data cannot support a conclusion that one center performs better or worse than another.

This finding instead highlights an important methodological point for vascular-access registries: recurrence estimates depend heavily on how previous interventions and failures are captured. Standardized prospective definitions and event-level documentation are essential before recurrence can be used as a benchmarking metric.

**Clinical implications**

A documented second failure or immaturation episode should function as a trigger for structured multidisciplinary review rather than simply another isolated access event. Such review should reassess inflow and outflow stenosis, central venous disease, access configuration, cannulation practices, intradialytic hemodynamics, previous intervention durability, and the

patient's remaining access options. This approach aligns with KDOQI and contemporary PLAN concepts, which require a contingency strategy for a failing access and a succession strategy for the patient's next access [1-3].

For quality improvement, dialysis programs should distinguish three levels of access history: no previous failure, single failure, and recurrent failure. Recording the mechanism, date, intervention, and subsequent patency after each event would allow more meaningful recurrence surveillance and time-to-event modeling than a cross-sectional count alone.

**Strengths and limitations**

The principal strength of this study is its focus on recurrent rather than single access failure, using a multicenter real-world cohort with clinical, biochemical, and access-history data. The statistical strategy was deliberately conservative: the primary model was parsimonious, biochemical analyses were separated from causal interpretation, and multiplicity was addressed with FDR correction. Sensitivity analyses tested the robustness of the qualitative episode coding and center association.

Several limitations are important. First, the study is retrospective and the timing of individual failure episodes relative to laboratory values was not consistently available, precluding causal inference. Second, the outcome combined failure and immaturation and did not distinguish whether repeated episodes occurred in the same access or sequential accesses. Third, two records used the qualitative category 'multiple times,' although sensitivity analysis excluding them was performed. Fourth, the dataset lacked vessel diameter, blood-flow measurements, cannulation technique, detailed lesion characteristics, and complete intervention histories. Fifth, center-level variation may reflect documentation or referral patterns rather than biological or care-quality differences. Sixth, some laboratory variables had missing data, particularly ejection fraction. Finally, both centers were located in Al-Ahsa, limiting geographic generalizability.

## CONCLUSIONS

Recurrent vascular access failure/immaturation affected 10.0% of adults in this two-center hemodialysis cohort and 37.3% of patients with any documented failure history. Thrombosis and stenosis were the dominant recurrent mechanisms, with thrombosis substantially more common in recurrent than single-event cases.

No patient-level clinical or biochemical factor demonstrated a robust independent association with recurrence after conservative adjustment and multiplicity control. A center-level signal was observed but was not stable across sensitivity analyses and should not be interpreted as a quality comparison.

Prospective vascular-access registries should record event timing, lesion characteristics, access flow, interventions, and whether recurrence occurs in the same or a subsequent access. Clinically, the second documented failure should prompt structured multidisciplinary reassessment and an explicit access contingency and succession plan.

## REFERENCES

[1] Lok CE, Huber TS, Orchanian-Cheff A, Rajan DK. Arteriovenous access for hemodialysis: a review. JAMA. 2024;331(15):1307-1317. doi:10.1001/jama.2024.0535. PMID: 38497953.

[2] Lok CE, Huber TS, Lee T, et al; National Kidney Foundation. KDOQI Clinical Practice Guideline for Vascular Access: 2019 Update. Am J Kidney Dis. 2020;75(4 Suppl 2):S1-S164. doi:10.1053/j.ajkd.2019.12.001. PMID: 32778223.

[3] Lok CE, Yuo T, Lee T. Hemodialysis vascular access: Core Curriculum 2025. Am J Kidney Dis. 2025;85(2):236-252. doi:10.1053/j.ajkd.2024.05.021. PMID: 39625430.

[4] Bylsma LC, Gage SM, Reichert H, Dahl SLM, Lawson JH. Arteriovenous fistulae for haemodialysis: a systematic review and meta-analysis of efficacy and safety outcomes. Eur J Vasc Endovasc Surg. 2017;54(4):513-522. doi:10.1016/j.ejvs.2017.06.024. PMID: 28843984.

[5] Dalton-Petillo SN, Siracuse JJ. Management of thrombosed or failed dialysis access. Semin Vasc Surg. 2024;37(4):394-399. doi:10.1053/j.semvascsurg.2024.10.006. PMID: 39675847.

[6] Zhang Y, Liu F, Li X, Xu Y, Jiang H. Predictive model for thrombosis in mature autologous arteriovenous fistula. Ann Vasc Surg. 2026;132:345-356. doi:10.1016/j.avsg.2026.06.038. PMID: 42365896.

[7] Salih SSM, Mohamed KO, Mohamedali AOO, et al. Predictors of early arteriovenous fistula failure in patients with end stage renal disease on hemodialysis: a systematic review and meta-analysis. Patient Saf Surg. 2025;19(1):24. doi:10.1186/s13037-025-00449-9. PMID: 40898218.

[8] Shiri P, Rezaeian S, Abdi A, Jalilian M, Khatony A. Risk factors for thrombosis in dialysis patients: a comprehensive systematic review and meta-analysis. J Vasc Nurs. 2024;42(3):165-176. doi:10.1016/j.jvn.2024.05.002. PMID: 39244328.

[9] Bashar K, Conlon PJ, Kheirelseid EAH, Aherne T, Walsh SR, Leahy A. Arteriovenous fistula in dialysis patients: factors implicated in early and late AVF maturation failure. Surgeon. 2016;14(5):294-300. doi:10.1016/j.surge.2016.02.001. PMID: 26988630.

[10] Fan Y, Luo X, Wang X, Luo H, Du J, Mou J, Lin L, Deng F. A meta-analysis of risk factors for restenosis following percutaneous transluminal angioplasty in arteriovenous fistulas of maintenance hemodialysis patients. Ann Vasc Surg. 2026;122:912-922. doi:10.1016/j.avsg.2025.08.033. PMID: 40992495.

[11] Han M, Zhao Q, Zhao J, Xue X, Wu H. Risk prediction models for autogenous arteriovenous fistula failure in maintenance hemodialysis patients: a systematic review and meta-analysis. World J Surg. 2024;48(10):2526-2542. doi:10.1002/wjs.12335. PMID: 39304914.

[12] Alhubail FM, Al Mousa AM, Alhassan GM, et al. Patterns and outcomes of permanent vascular access in end-stage kidney disease: a multicenter experience. Cureus. 2025;17(10):e94950. doi:10.7759/cureus.94950. PMID: 41267729.

[13] von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. J Clin Epidemiol. 2008;61(4):344-349. doi:10.1016/j.jclinepi.2007.11.008. PMID: 18313558.

[14] Zou G. A modified Poisson regression approach to prospective studies with binary data. Am J Epidemiol. 2004;159(7):702-706. doi:10.1093/aje/kwh090. PMID: 15033648.