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ORIGINAL RESEARCH

Menopausal Status and Thrombosis Risk in Hemodialysis Catheter Use: A Secondary Analysis of a Prospective Cohort

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Background: Current literature lacks research on the link between menopause and central venous catheter (CVC) thrombosis risk. The aim of the study is to evaluate the association between menopausal status and catheter thrombosis.

Methods: This study analyzed a subset of 322 female patients with tunneled CVC placement from a previously published cohort that was conducted at Hasheminejad Kidney Center, Tehran, Iran (2015–2019). Menopausal status was determined based on clinical documentation and menstrual history (cessation of menses for ≥12 months, history of bilateral oophorectomy, or documented clinical diagnosis. Catheter outcomes were classified as dysfunction due to thrombosis or infection.

Results: Postmenopausal women were significantly older than premenopausal women (62.9 vs 38.6 years, p<0.001), and analyses were adjusted for age to account for this confounding. Catheter thrombosis occurred more frequently among postmenopausal women (68.3% vs 37.9%, p < 0.001), while infection was more common in premenopausal patients (38.9% vs 18.5%, p < 0.001). In the adjusted competing risk model for thrombosis, postmenopausal status was associated with a significantly higher subdistribution hazard (SHR = 2.54; 95% CI: 1.30–4.94; p = 0.006), along with higher BMI (SHR = 1.04 per unit; p = 0.017) and hypertension (SHR = 1.62; p = 0.029). In contrast, menopausal status was not significantly associated with infection risk (SHR = 0.52; p = 0.146), although diabetes showed a borderline association (SHR = 1.81; p = 0.071).

Conclusion: Postmenopausal status was associated with increased risk of catheter thrombosis in this hypothesis-generating analysis. These findings highlight the need for prospective studies investigating hormonal influences on catheter-related complications.

Keywords: menopausal status, hemodialysis catheter thrombosis, central venous catheter complications, CVC, catheter dysfunction in hemodialysis, hormonal influence on thrombosis

Introduction

Central venous catheters (CVC) are indicated for patients who need hemodialysis to bridge the gap until arteriovenous fistula (AVF) maturation, kidney transplantation, or the resolution of AVF complications. Additionally, CVC can be utilized long term for patients who have had multiple prior failed vascular access, prefer this option, or have a limited life expectancy.[1] Research from the Dialysis Outcomes and Practice Patterns Study (DOPPS) shows considerable variation in CVC prevalence, with rates between 1% and 45% across different nations.[2] In the United States, CVC is still utilized by up to 80% of hemodialysis patients.[3]

The dysfunction of catheters can cause serious health issues and increase mortality rates, typically due to thrombosis or infections, which play a major role in patient morbidity and create a notable financial burden on the healthcare system.

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Research indicates that the prevalence of thrombosis among hemodialysis patients varies between 1.8% and 53.3%.[4] Additionally, catheter-related infections have an incidence rate of 1.1 to 5.5 cases per 1000 CVC days.[1]

We have previously reported that women, patients with hypertension, and those who are obese are at a notably higher risk for catheter thrombosis he multivariate competing risk analysis indicated a hazard ratio of 1.82 for catheter thrombosis in females. Furthermore, previous study showed that 70.5% of these women were in menopause.[5] There is evidence that venous thromboembolism is a significant factor contributing to cardiovascular burden, with an incidence of about 1 per 1000 person-years in postmenopausal women.[6] In hemodialysis patients, vascular access failure is multifactorial, involving uremia-related endothelial dysfunction, chronic inflammation, coagulation abnormalities, and repeated vessel injury. The menopausal transition may exacerbate these pathways through estrogen decline, which affects nitric oxide bioavailability, platelet aggregation, fibrinolytic activity and an accelerated rate of vascular aging.[7] Another study found that arterial stiffness increases, and endothelial function decreases throughout the menopause transition in healthy women.[8]

While existing literature has highlighted female sex as a possible risk factor for catheter dysfunction, the specific role of menopausal status in central venous catheter (CVC) thrombosis has not been well investigated. While age-related vascular changes are well-documented, the menopausal transition introduces distinct hormonal alterations—including estrogen decline and procoagulant shifts—that may confer additional thrombosis risk beyond chronological aging. This study specifically aims to evaluate whether menopausal status independently predicts catheter thrombosis after adjusting for age.Clinical guidelines suggest that antiplatelet or anticoagulant therapy may benefit patients at elevated thrombosis risk, yet no studies have explored whether menopausal women represent such a high-risk subgroup.[1] Motivated by this gap, and preliminary findings from our previous cohort suggesting increased thrombosis rates in postmenopausal patients, this study aimed to evaluate the association between menopausal status and CVC thrombosis using a competing risks framework.

Methods

Study Design and Data Source

This subgroup analysis was nested within a previously published cohort study investigating tunneled hemodialysis catheter dysfunction at Hasheminejad Kidney Center, Tehran, Iran, between 2015 and 2019.[5] The original study included 466 patients undergoing their first tunneled dialysis catheterization and evaluated risk factors for catheter dysfunction using competing risk analysis. In the parent cohort, patient information was extracted from the hospital information system and cross-checked against source records; when data were incomplete in the electronic record, paper medical files were reviewed to complete key variables where possible.

For the present analysis, we focused specifically on the role of menopausal status as a potential modifier of risk. Among the original cohort, a subset of 322 female patients had clear documentation of menopausal status in their medical records and were included in this secondary analysis. All participants had provided informed consent for the anonymized use of their data in research. No additional data collection was performed for this analysis. Missing BMI values were handled using multiple imputations based on age and diabetes status.

Variables and Outcomes

Menopausal status was categorized as premenopausal or postmenopausal. Menopausal status was determined based on clinical documentation in medical records, including: (1) cessation of menstrual periods for at least 12 months, (2) history of bilateral oophorectomy.[9] Hormonal assays (FSH, estradiol) were not routinely available in this retrospective cohort. Surgical menopause (bilateral oophorectomy) was included in the postmenopausal group. Hormone replacement therapy (HRT) use was not recorded in the medical records; however, HRT is extremely uncommon in this population due to limited prescription practices in Iran during the study period. This limitation is acknowledged. Other covariates included age, body mass index (BMI), hypertension (HTN), and diabetes mellitus (DM), as previously defined. Variables were selected for inclusion based on clinical relevance and prior literature, regardless of univariate p-values, to avoid selection bias.

The study outcome was catheter dysfunction due to thrombosis, with catheter-related infection considered a competing event. Thrombosis was defined as impaired catheter function requiring replacement, based on flow

parameters and clinical judgment. Infection was defined by a positive blood culture from the catheter line or peripheral sample, combined with clinical signs such as fever or chills.

Statistical Analysis

Descriptive statistics were used to summarize patient characteristics by menopausal status. Continuous variables were compared using independent *t*-tests or U Mann Whitney tests, as appropriate. Categorical variables were compared using Chi-square tests.

Time-to-event analysis was performed using Fine and Gray competing risk regression models, treating thrombosis and infection as mutually exclusive failure outcomes. Sub-distribution hazard ratios (SHRs) with 95% confidence intervals (CIs) were estimated separately for each outcome. Variables were selected for inclusion based on clinical relevance or univariate association ($p < 0.2$). To assess potential collinearity between age and menopausal status, variance inflation factors (VIFs) were calculated. In addition, sensitivity analyses were performed by excluding age from the competing-risk regression models. To visually present the results, adjusted cumulative incidence function (CIF) curves were generated for thrombosis and infection by menopausal status, accounting for the competing event. Kaplan–Meier curves with Cox-based predicted survival were additionally produced as supportive visualizations. All statistical analyses were conducted in Stata version 16.1 (StataCorp LLC, College Station, TX).

Results

Out of 322 patients included in the subgroup analysis based on menopausal status, 227 (70.5%) were postmenopausal and 95 (29.5%) were premenopausal. Postmenopausal patients were significantly older (mean age: 62.9 ± 8.0 vs 38.6 ± 10.8 years, $p < 0.001$) and had higher BMI (29.6 ± 5.9 vs 27.2 ± 6.0 kg/m^2, $p = 0.001$). Median duration of catheter function was slightly lower in postmenopausal patients (234 vs 262 days), although the difference was not statistically significant ($p = 0.209$). Catheter thrombosis occurred significantly more frequently in postmenopausal patients (68.3% vs 37.9%, $p < 0.001$), while infection-related dysfunction was more common among premenopausal patients (38.9% vs 18.5%, $p < 0.001$). Baseline characteristics were shown in Table 1.

When stratified by thrombosis status, those who experienced thrombosis were older (58.3 ± 12.6 vs 52.1 ± 15.7 years, $p < 0.001$), had higher BMI (29.6 ± 5.9 vs 27.9 ± 6.1 kg/m^2, $p = 0.013$), and had shorter median follow-up (233 vs 301 days, $p = 0.007$). Diabetes and hypertension did not show significant differences. Among patients who developed catheter infection, younger age was observed (50.7 ± 16.2 vs 57.4 ± 13.2 years, $p = 0.001$), while BMI showed a non-significant trend toward lower values (27.8 vs 29.3 kg/m^2, $p = 0.065$). Duration of follow-up was similar between infected and non-infected groups ($p = 0.48$) (Table 2).

Table 1 Baseline Characteristics of Patients by Menopausal Status

	Menopause (n=227)	**No Menopause (n=95)**	**p-value**
Age (years)	62.9 ± 8.0	38.6 ± 10.8	< 0.001
BMI (kg/m^2)	27.2 ± 6.0	29.6 ± 5.9	0.001
Follow-up (days)	262 (317)	234 (316)	0.209*
DM	55 (24.2)	14 (17.7)	0.057
HTN	65 (28.3)	30 (31.58)	0.597
Catheter Infection	42 (18.5)	37 (38.9)	<0.001
Catheter Thrombosis	155 (68.3)	36 (37.9)	<0.001

Notes: Data are mean ± SD for normally distributed parameters or n (%); p value and statistically significant p values (p<0.05) are in bold. *The rank-sum test is used to compare the Median of two continuous distributions.

Abbreviations: BMI, body mass index; DM, diabetes mellitus; HTN, hypertension.

Table 2 Comparison of Demographic and Clinical Characteristics by Type of Outcomes

Variable	Thrombosis		p-value	Infection		p-value
	Yes (n=191)	No (n=131)		Yes (n=79)	No (n=243)	
Age	58.26 ± 12.58	52.08 ± 15.73	< 0.001	50.65 ± 16.24	57.40 ± 13.16	0.001
BMI	29.59 ± 5.87	27.89 ± 6.09	0.013	27.81 ± 5.71	29.25 ± 6.07	0.065
Follow-up (days)	233 (274)	301 (420)	0.007	276 (357)	243 (303)	0.48
DM	58 (30.37)	37 (28.2)	0.68	18 (22.7)	51 (20.9)	0.73
HTN	39 (20.42)	30 (22.9)	0.59	24 (30.4)	71 (29.2)	0.84

Notes: Data are mean ± SD for normally distributed parameters or n (%); p value and statistically significant p values (p<0.05) are in bold. The rank-sum test is used to compare the Median of two continuous distributions.
Abbreviations: BMI, body mass index; DM, diabetes mellitus; HTN, hypertension.

Table 3 Competing Risk Regression Model for Catheter Thrombosis (Thrombosis as Failure Event, Infection as Competing Risk)

Variable	SHR	P>z	95% CI
Age	1.00	0.764	[0.97, 1.02]
Menopause	2.54	0.006	[1.30, 4.94]
BMI	1.04	0.017	[1.01, 1.08]
HTN	1.62	0.029	[1.05, 2.50]
DM	0.68	0.112	[0.43, 1.09]

Note: Sub-distribution hazard ratios (SHRs) and 95% confidence intervals for thrombosis-associated variables.
Abbreviations: BMI, body mass index; HTN, hypertension; DM, diabetes mellitus; SHR, sub-distribution hazard ratio; CI, confidence interval.

Survival Analysis and Competing Risk Models

Competing risk regression models were used to assess the association between patient characteristics and catheter dysfunction outcomes, with thrombosis and infection treated as mutually exclusive failure events. In the model for thrombosis, postmenopausal status (SHR = 2.54, 95% CI: 1.30–4.94, $p = 0.006$), higher BMI (SHR = 1.04 per unit, 95% CI: 1.01–1.08, $p = 0.017$), and hypertension (SHR = 1.62, 95% CI: 1.05–2.50, $p = 0.029$) were independently associated with increased risk. Age and diabetes mellitus were not significant predictors (Table 3).

For infection-related dysfunction, menopause was not significantly associated (SHR = 0.52, 95% CI: 0.21–1.26, $p = 0.146$), though diabetes showed a borderline association with increased risk (SHR = 1.81, 95% CI: 0.95–3.44, $p = 0.071$). Other variables, including age, BMI, and hypertension, were not significant (Table 4).

The adjusted CIF curve for thrombosis showed a consistently higher cumulative incidence among postmenopausal women compared with premenopausal women throughout follow-up (Figure 1). In contrast, the adjusted CIF curve for infection showed a higher cumulative incidence among premenopausal women, while postmenopausal women had a lower incidence over time (Figure 2). Moreover, Kaplan–Meier curves with Cox-based predicted survival (not accounting for the competing risk of the alternate event) showed lower thrombosis-free survival among postmenopausal women and lower infection-free survival among premenopausal women, in line with the competing-risk findings.

Table 4 Competing Risk Regression Model for Catheter Infection (Infection as Failure Event, Thrombosis as Competing Risk)

Variable	SHR	P>z	95% CI
Age	0.98	0.235	[0.95, 1.01]
Menopause	0.52	0.146	[0.21, 1.26]
BMI	0.96	0.122	[0.91, 1.01]
HTN	0.81	0.463	[0.45, 1.43]
DM	1.81	0.071	[0.95, 3.44]

Note: Sub-distribution hazard ratios (SHRs) and 95% confidence intervals for infection-associated variables.

Abbreviations: BMI, body mass index; DM, diabetes mellitus; HTN, hypertension; SHR, sub-distribution hazard ratio; CI, confidence interval.

Sensitivity Analysis: Assessment of the Age and Menopause Collinearity

Variance inflation factor (VIF) analysis did not indicate problematic multicollinearity among the included covariates, with the highest VIF observed being 2.6, suggesting acceptable levels of collinearity (Table S1; Figure S1).

In sensitivity analyses excluding age from the model, competing risk regression with thrombosis as the primary outcome and infection as the competing event showed that menopause remained significantly associated with a higher subdistribution hazard of thrombosis. The direction and magnitude of associations were consistent with the main analysis (Table S2; Figure S2).

When infection was considered as the primary outcome and thrombosis as the competing event, menopause was associated with a significantly lower subdistribution hazard of infection. Given the absence of problematic multicollinearity based on VIF assessment, the age-adjusted model was retained as the primary analysis, and the age-excluded model was considered as a sensitivity analysis (Table S3).

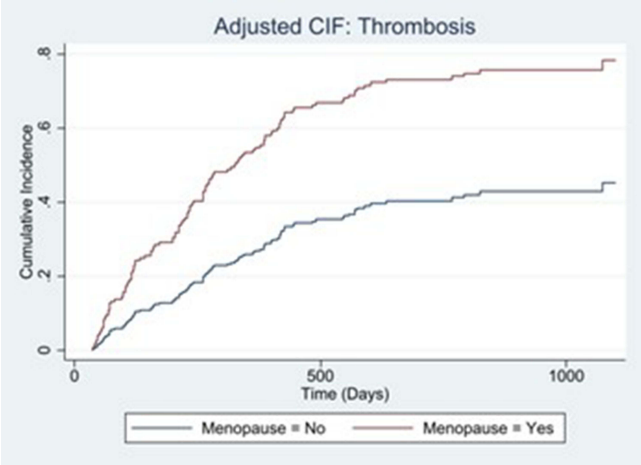

Figure 1 Cumulative incidence curves for catheter thrombosis by menopausal status, with catheter-related infection treated as the competing event.

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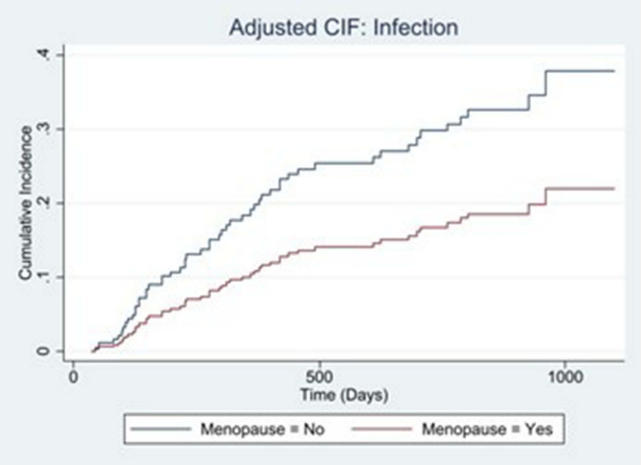

Figure 2 Cumulative incidence curves for catheter-related infection by menopausal status, with catheter thrombosis treated as the competing event.

Discussion

This study showed that catheter thrombosis in menopause women was detected significantly more than non-menopause women. Menopause is characterized by a decline in estrogen levels, which influences coagulation and fibrinolytic systems. Estrogen has both protective and prothrombotic effects, depending on its form and route of administration. Natural estrogen (17β-estradiol) has vasodilatory and anti-inflammatory effects, whereas synthetic estrogens (eg, in hormone therapy) may increase thrombotic risk.[10] The loss of endogenous estrogen after menopause has been correlated with endothelial dysfunction, increased vascular inflammation, and a prothrombotic state. Studies suggest that menopause is associated with elevated levels of fibrinogen, factor VII, and plasminogen activator inhibitor-1 (PAI-1), contributing to a hypercoagulable state.[11]

On the other hand, chronic kidney disease (CKD) and end-stage renal disease (ESRD) are prevalent in older women, necessitating long-term vascular access. However, menopause and uremia synergistically increase thrombosis risk potentially related to hypercoagulability from estrogen deficiency and chronic inflammation. Studies report that women on hemodialysis have a higher incidence of catheter-related thrombosis than men.[12] Menopausal status has been studied in relation to various cardiovascular and vascular health risks, but its role as an independent risk factor for vascular access failure specifically is not well-established in literature. However, menopause is associated with changes in vascular health, such as reduced estrogen levels, which can lead to endothelial dysfunction, increased inflammation, and altered coagulation—all of which could potentially influence vascular access outcomes.

While we propose biological mechanisms involving estrogen decline, it is important to emphasize that this study did not measure hormonal levels, coagulation factors, or inflammatory markers. The proposed mechanisms are inferred from broader literature and require direct investigation in future studies.

In this study, older age and higher BMI were significantly associated with catheter thrombosis, but when the results were adjusted for these two factors, the role of menopause on thrombosis remained significant. Beyond hormonal changes, menopause is often accompanied by additional thrombotic events risk factors. Advancing age is an independent risk factor for thrombosis.[13] Adiposity increases proinflammatory cytokines and thrombogenic factors.[14] Considering competition risk of thrombosis by infection, menopause (SHR 2.5) and BMI (1.04) and hypertension (1.6) were the strong predictors for thrombosis in catheters. While age and diabetes were not significant predictors.

In this study, initially there was a significant rate of infection in non-menopause women; However, the effect did not remain significant after adjustment for age. Other studies have shown that estrogen plays a crucial role in modulating immune responses. Its decline during menopause decreased anti-inflammatory cytokines (eg, IL-10) and increased pro-inflammatory markers (eg, IL-6, TNF-α), impairing infection defense,[15] decreased T-cell proliferation and impaired neutrophil function,[16] increases vascular permeability and bacterial adhesion.[17] Also, estrogen deficiency reduces phagocytic activity, weakening pathogen clearance.[16] Menopause-associated immune dysfunction increases susceptibility

to infections, particularly in the genitourinary and respiratory systems. While hormone replacement therapy may offer protective benefits.

However, there is a lack of data about the risk of vascular access infection in postmenopausal women. Considering competition risk of infection by thrombosis, none of the factors were predictors for infection in catheters. While diabetes had a trend to be a significant predictor of infection in catheter (SHR 1.8). Diabetes is an independent factor associated with a higher risk of catheter-related bloodstream infections (CRBSI) in hemodialysis patients.[18,19]

There are some suggestions about catheter dysfunction prevention. The guideline of KDOQI suggests low-dose aspirin and low-concentration citrate and TPA and also allows clinician discretion for locking solutions.[1] These hypothesis-generating findings suggest that menopausal status may identify a higher-risk subgroup for catheter thrombosis. However, randomized trials are needed before any clinical practice changes can be recommended.

Limitation

This study has some limitations. While adjusted for age and BMI, unmeasured confounders (eg, anticoagulant/antiplatelet use, catheter lock solutions, hormone replacement therapy, genetic thrombophilia, inflammatory markers, or CKD stage) may influence thrombosis risk. The study's retrospective and observational nature preclude causal conclusions. Menopause may correlate with other age-related factors (eg, vascular calcification) rather than directly causing thrombosis. However, the effect of menopause was adjusted for age to omit this confounding effect. Despite age adjustment, the strong correlation between menopause and age raises the possibility that residual confounding from age-related vascular changes may partially explain the observed association. Future studies should include age-matched premenopausal women (eg, those with premature ovarian failure or surgical menopause) to better disentangle these effects. Additionally, the study lacked data on hormone replacement therapy (HRT) use, which could potentially influence thrombotic risk in postmenopausal women. Although HRT is uncommon in the dialysis population, the absence of this information limits our ability to fully distinguish between the effects of endogenous hormonal decline and exogenous estrogen exposure. Finally, competing events such as catheter removal related to infection vs thrombosis may distort risk estimates. In this regard, the results of this study are evaluated according to competition risk of infection for thrombosis.

Conclusion

This study suggests an association of the relationship between menopause and CVC thrombosis risk in hemodialysis patients. It may be considered for clinicians to consider preventing strategies of CVC thrombosis and closer monitoring in menopause end stage renal disease patients in clinical practice. However, given the observational design, strong correlation between age and menopausal status, and lack of hormonal measurements, these findings should be considered hypothesis-generating. Prospective studies with hormonal profiling and age-matched controls are needed to confirm whether menopause independently contributes to thrombosis risk and to identify potential targets for intervention. Also, interventional studies are needed before recommending routine thromboprophylaxis in this population.

Data Sharing Statement

The data used in this study are not publicly accessible as they belong to a larger institutional database managed by Iran University of Medical Sciences. However, the corresponding author can provide access to the datasets upon justified request.

Ethics Approval and Consent to Participate

The study was approved by the Iran University of medical sciences ethical committee numbered: IR.IUMS.REC.1394.8611215654. All patients signed informed consent at the beginning of admission and allowed their clinical data to be used for research purposes. All methods were carried out following relevant guidelines and regulations or the declaration of Helsinki.

Consent for Publication

All patients signed informed consent at the beginning of admission and allowed their clinical data to be published anonymously.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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