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MANAGEMENT OF ATRIAL FIBRILLATION IN PATIENTS ON CHRONIC HEMODIALYSIS: A LITERATURE REVIEW

Jaouad Nguadi, Youssef Lahmouz, Hanae El Ghiati, Jihane Fagouri, Hicham Bouzelmat, Ali Chaib

Rythmology Department – Mohammed V Military Training Hospital.

***Corresponding Author: Jaouad Nguadi**

Rythmology Department – Mohammed V Military Training Hospital.

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ABSTRACT

Atrial fibrillation (AF) is highly prevalent in patients on chronic hemodialysis (HD), with an estimated prevalence of 10–27%, posing a major challenge due to elevated risks of ischemic stroke and major bleeding. This review examines epidemiology, pathophysiology, risk assessment, and therapeutic strategies, aligned with the latest societal guidelines (ACC/AHA/ACCP/HRS 2023, ESC 2024). Oral anticoagulation (OAC) remains controversial due to an unfavorable risk-benefit profile, with warfarin preferred in end-stage renal disease (ESRD), while direct oral anticoagulants (DOACs) are restricted. Rate control is prioritized, with rhythm control emerging for symptomatic patients. A multidisciplinary, individualized approach is essential to optimize outcomes.

KEYWORDS: Atrial fibrillation; Chronic hemodialysis; Anticoagulation; Guidelines; End-stage renal disease.

INTRODUCTION

Atrial fibrillation (AF) is the most common arrhythmia in patients with chronic kidney disease (CKD), particularly in ESRD on hemodialysis (HD). This population faces compounded thromboembolic risk from AF and heightened bleeding risk from uremia, platelet dysfunction, anemia, and cardiovascular comorbidities. Despite advances in nephrology and cardiology, AF management in HD patients remains complex, with OAC underutilization due to uncertainty regarding efficacy and safety. This literature review, structured in accordance with standards of publications in the *Journal of the American College of Cardiology* (JACC), the European Society of Cardiology (ESC), American College of Cardiology (ACC), and American Heart Association (AHA) guidelines, synthesizes recent data (2023–2025) on prevalence, pathophysiology, and therapeutic strategies, incorporating the latest recommendations.

Epidemiology

The prevalence of AF in chronic HD patients is significantly higher than in the general population, ranging from 10 to 27% across cohort studies, with an annual incidence of 5–7%. A recent meta-analysis including over 2 million dialysis patients reported a pooled prevalence of 14% in HD cohorts, increasing with age (>80 years: up to 32%) and dialysis vintage.[3] These patients exhibit a 5- to 10-fold increased risk of ischemic stroke compared to the general population, with AF-attributable cardiovascular mortality rising by 20–30%.[9] OAC utilization increased from 20.5% in 2007 to 34.1% in 2020 among incident AF cases on HD, reflecting gradual adoption but persistent under-treatment, especially in high-bleeding-risk subgroups.[2]

Pathophysiology

The AF–CKD relationship is bidirectional: AF accelerates CKD progression via hypotension, renal stasis, and renin-angiotensin-aldosterone system (RAAS) activation, while CKD promotes AF through chronic volume overload, electrolyte imbalances (hypokalemia, hypomagnesemia), systemic inflammation, myocardial fibrosis, and endothelial dysfunction.[4] In HD patients, intradialytic hemodynamic fluctuations, repeated heparin exposure, and rapid volume shifts increase arrhythmic burden, with paroxysmal or subclinical AF predominating—detected in up to 40% of asymptomatic cases via implantable monitors.[8] These mechanisms drive hypercoagulability (uremic prothrombotic state, left atrial appendage stasis) alongside major bleeding

vulnerability (uremic platelet dysfunction, anemia, frequent antiplatelet use).

Risk Assessment for Thrombosis and Bleeding

Thromboembolic risk is assessed using the **CHA_2DS_2-VASc** score, modified by adding one point for eGFR <30 mL/min/1.73 m² or HD (annual stroke risk >10% without OAC in scores ≥6).[12] The **ATRIA** score, incorporating proteinuria and severe CKD, demonstrates superior calibration and discrimination (c-statistic 0.74 vs. 0.68 for CHA_2DS_2-VASc) in dialysis patients.[13] For bleeding risk, the **HAS-BLED** score has limited sensitivity and overestimates risk in HD; an individualized approach is favored, accounting for prior bleeding, anemia (Hb <10 g/dL), peritoneal vs. HD, and concomitant antiplatelets. High AF burden (>5.5 h/day on continuous monitoring) independently amplifies thromboembolic risk, supporting implantable monitoring for risk stratification.[16]

Therapeutic Strategies

Oral Anticoagulation

OAC is the cornerstone of stroke prevention, but net clinical benefit is uncertain in HD due to a 1.5- to 2-fold increase in major bleeding versus the general population.[13] **Warfarin** (target INR 2.0–3.0) remains first-line, reducing ischemic stroke by 20–30% in observational studies but increasing vascular calcification and intracranial hemorrhage (HR 1.8).[14] **DOACs** (apixaban 2.5 mg BID, rivaroxaban 15 mg daily for eGFR 15–30 mL/min) show superior safety over warfarin in observational data (20% reduction in major bleeding), but no dedicated randomized controlled trials (RCTs) exist in HD.[19] **Dabigatran** and **edoxaban** are contraindicated in ESRD (eGFR <15 mL/min). A 2024 meta-analysis of 18 observational studies (n=72,000) found neutral mortality benefit but significant stroke reduction with DOACs vs. no OAC (RR 0.68; 95% CI 0.52–0.89).[15] A "no OAC" strategy is advocated in very high bleeding risk, with **left atrial appendage occlusion (LAAO)** emerging as an alternative (ongoing **LAA-KIDNEY** trial, results expected 2027).[20]

Rate Control

Rate control (resting HR <110 bpm, <120 bpm during activity) is first-line (Class I, LOE A), using **beta-blockers** (metoprolol succinate 25–100 mg/day) or **non-dihydropyridine calcium channel blockers** (diltiazem 120–360 mg/day)—both effective, well-tolerated, and partially dialyzable.[17] **Digoxin** is adjunctive but high-risk (renal accumulation, cardiac toxicity, increased mortality in cohorts). A 2025 registry study (n=12,000) linked early rate-control therapy to reduced stroke (HR 0.80) and all-cause mortality (HR 0.75).[16]

Rhythm Control

Reserved for symptomatic patients refractory to rate control (Class IIa, LOE B), **amiodarone** is preferred (non-renal clearance, 100–200 mg/day) or **catheter ablation**offers 60–70% efficacy at 1 year, but with elevated periprocedural risk in HD (8% major complications vs. 3% in general population).[18] Sodium channel blockers (flecainide, propafenone) are contraindicated in structural heart disease or ischemia.

Societal Guideline Recommendations

The **2023 ACC/AHA/ACCP/HRS guidelines** (Class IIb, LOE B-NR) recommend warfarin as reasonable OAC in ESRD/HD, with reduced-dose apixaban (2.5 mg BID) considered if warfarin is contraindicated or INR unstable, but not routine due to limited data.[10] The **2024 ESC guidelines** (Class IIa, LOE C-EO) endorse warfarin for eGFR <15 mL/min or HD, with DOACs for eGFR 15–50 mL/min, emphasizing shared decision-making via the **AF-CARE** framework (Comorbidities, Avoid stroke, Reduce symptoms, Early and continued care, Access to treatment).[11] Both prioritize rate control, multidisciplinary management (cardiology–nephrology), and dynamic bleeding risk reassessment.

Society	OAC in HD/ESRD	Rate/Rhythm Control	Class/LOE
ACC/AHA 2023	Warfarin (IIb); Apixaban reduced (IIb)	Beta-blockers/CCBs (I); Amiodarone/Ablation (IIa)	B-NR / C-LD
ESC 2024	Warfarin (IIa); DOAC if eGFR >15 (IIb)	Beta-blockers (I); Ablation (IIa)	C-EO / B-R

Challenges and Future Directions

Key challenges include under-treatment (only 25% of eligible patients receive OAC), drug interactions (heparin, antiplatelets), lack of prospective RCTs in HD, and inter-individual bleeding risk variability. Future directions include **factor XI inhibitors** (abelacimab, asundexian—phase III trials ongoing), **percutaneous LAAO** (60% stroke reduction in preliminary registries), and **implantable monitoring** to quantify AF burden and guide OAC initiation. Artificial intelligence models using intradialytic ECG for event prediction are under exploration.

CONCLUSION

AF management in chronic HD requires a highly individualized approach, balancing thromboembolic and bleeding risks, with warfarin as the reference OAC and rate control as the priority. Recent guidelines (ACC/AHA 2023, ESC 2024) advocate caution, but ongoing trials (LAA-KIDNEY, FXI inhibitors) promise improved options. Interdisciplinary collaboration (cardiologist, nephrologist, hematologist) and dynamic reassessment are imperative to optimize quality of life and survival in this very high-risk population.

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