EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Coden USA: EJBPBO
Volume: 12
Issue: 12
Year: 2025

MANAGEMENT OF ATRIAL FIBRILLATION IN PATIENTS WITH CIRRHOSIS: 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.

DOI: https://doi.org/10.5281/zenodo.17746167

How to cite this Article: *Jaouad Nguadi, Youssef Lahmouz, Hanae El Ghiati, Jihane Fagouri, Hicham Bouzelmat, Ali Chaib. (2025). MANAGEMENT OF ATRIAL FIBRILLATION IN PATIENTS WITH CIRRHOSIS: A LITERATURE REVIEW. European Journal of Biomedical and Pharmaceutical Sciences, 12(12), 63–65.

Article Received on 25/10/2025 **Article Revised on 14/11/2025** **Article Published on 01/12/2025**

ABSTRACT

Atrial fibrillation (AF) is increasingly prevalent in patients with liver cirrhosis, with estimates ranging from 8–25%, driven by hemodynamic, inflammatory, and coagulopathic alterations. This population faces heightened risks of thromboembolism and catastrophic bleeding due to portal hypertension, varices, and thrombocytopenia. This review synthesizes epidemiology, pathophysiology, risk stratification, and management strategies, aligned with the latest societal guidelines (ACC/AHA/ACCP/HRS 2023, ESC 2024). Oral anticoagulation (OAC) is challenging owing to unpredictable pharmacokinetics and bleeding propensity; vitamin K antagonists (VKAs) and reduced-dose direct oral anticoagulants (DOACs) are conditionally endorsed. Rate control is preferred, with rhythm control reserved for symptomatic cases. Multidisciplinary decision-making integrating hepatology and cardiology is critical.

KEYWORDS: Atrial fibrillation; Liver cirrhosis; Anticoagulation; Guidelines; Portal hypertension.

INTRODUCTION

Atrial fibrillation (AF) is the most common sustained arrhythmia in patients with advanced liver disease, particularly cirrhosis. The interplay of systemic inflammation, autonomic dysfunction, and altered hemostasis amplifies both thromboembolic and hemorrhagic risks. Despite growing recognition, management remains poorly codified, with OAC under-prescribed due to fears of variceal bleeding and drug metabolism variability. This literature review, structured per standards of the Journal of the American College of Cardiology (JACC), European Society of Cardiology (ESC), American College of Cardiology (ACC), and American Heart Association (AHA), consolidates data from 2023–2025 on prevalence, mechanisms, and therapeutic approaches, incorporating current guideline recommendations.

Epidemiology

AF prevalence in cirrhosis ranges from 8% in compensated disease to 25% in decompensated cohorts, exceeding age-matched general populations by 3–5 fold. A 2024 meta-analysis of 1.8 million cirrhotic patients reported pooled prevalence of 16%, rising with Child-Pugh class (Class C: 28%) and alcohol etiology.[3] Annual incidence approximates 4–6%. AF confers a 2.5-fold increase in mortality, predominantly from thromboembolism (stroke HR 3.1) and variceal hemorrhage (HR 4.2) in anticoagulated patients.[9] OAC prescription rose from 15% in 2015 to 28% in 2023 among incident AF cases, yet remains suboptimal in high-MELD subgroups.[2]

Pathophysiology

The AF–cirrhosis relationship is bidirectional: cirrhosis promotes AF via hyperdynamic circulation, left atrial enlargement, QT prolongation, and neurohormonal activation (RAAS, sympathetic surge), while AF exacerbates hepatic congestion and decompensation.[4] Systemic inflammation (IL-6, TNF-α), endotoxinemia, and oxidative stress drive myocardial remodeling. Coagulopathy is complex—hypo-coagulable (reduced factors II, VII, IX, X; thrombocytopenia) yet hypercoagulable (elevated von Willebrand factor, factor VIII; endothelial dysfunction). Portal hypertension predisposes to variceal bleeding, while splanchnic

vasodilation alters drug clearance. Subclinical AF is detected in 35% via loop recorders, correlating with ascites and encephalopathy.[8]

Risk Assessment for Thrombosis and Bleeding Thromboembolic risk is evaluated using CHA_2DS_2-VASc, with cirrhosis adding 1–2 points (annual stroke risk >8% at scores ≥5).[12] The GARFIELD-AF model, incorporating liver dysfunction, outperforms CHA_2DS_2-VASc in cirrhosis (c-statistic 0.76 vs. 0.69).[13] Bleeding risk exceeds general populations 3-fold; HAS-BLED overestimates, failing to capture variceal or portal gastropathy contributions. Modified scores integrating Child-Pugh ≥B, platelet count $<50\times10^9$/L, and prior variceal bleed are advocated. High AF burden (>6 h/day) independently predicts stroke (HR 2.8), supporting prolonged monitoring.[16]

Therapeutic Strategies

Oral Anticoagulation

OAC is indicated for CHA_2DS_2-VASc ≥2 (men) or ≥3 (women), but net benefit is marginal in advanced cirrhosis due to 2–3-fold bleeding escalation.[13] VKAs (target INR 2.0–2.5) reduce stroke by 25–35% in observational data but increase major bleeding (HR 2.1), particularly variceal (HR 3.5).[14] DOACs demonstrate favorable pharmacokinetics in Child-Pugh A–B (apixaban 2.5–5 mg BID; rivaroxaban 15–20 mg daily), with 30% lower bleeding versus VKAs in meta-analyses, though contraindicated in Child-Pugh C.[19] Edoxaban and dabigatran are avoided due to hepatic metabolism. A 2025 registry (n=58,000) reported DOACs associated with neutral mortality but superior stroke prevention (RR 0.62; 95% CI 0.48–0.79) versus no OAC.[15] Non-OAC strategies include endoscopic variceal banding prophylaxis and left atrial appendage occlusion (LAAO) in select cases (ongoing CIRRHOSIS-AF trial, results 2028).[20]

Rate Control

Rate control (resting HR <110 bpm) is first-line (Class I, LOE A), using beta-blockers (carvedilol 6.25–25 mg/day; propranolol 20–80 mg/day)—preferred for dual portal pressure reduction—or non-dihydropyridine calcium channel blockers (diltiazem 120–240 mg/day).[17] Digoxin is cautioned (hepatic clearance variability). A 2024 cohort (n=9,500) linked beta-blocker initiation to reduced decompensation (HR 0.72) and mortality (HR 0.68).[16]

Rhythm Control Indicated for symptomatic failure of rate control (Class IIa, LOE B), amiodarone (100–200 mg/day) is favored (minimal hepatic metabolism). Catheter ablation yields 55–65% 1-year success but carries 10% complication rate (pericardial effusion, hepatic decompensation).[18] Electrical cardioversion is effective acutely but recurrence exceeds 70% at 6 months. Class Ic agents are contraindicated in structural disease.

Societal Guideline Recommendations The 2023 ACC/AHA/ACCP/HRS guidelines (Class IIb, LOE B-NR) endorse VKAs or reduced-dose DOACs in Child-Pugh A–B, with LAAO consideration in high-bleeding-risk. The 2024 ESC guidelines (Class IIa, LOE C-EO) recommend DOACs over VKAs in Child-Pugh A–B, VKAs in Child-Pugh C if INR controllable, emphasizing hepatology input and variceal screening. Both prioritize rate control and multidisciplinary care.

Society	OAC in Cirrhosis	Rate/Rhythm Control	Class/LOE
ACC/AHA 2023	VKAs/DOACs reduced (IIb)	Beta-blockers (I); Amiodarone/Ablation (IIa)	B-NR / C-LD
ESC 2024	DOACs Child A–B (IIa); VKAs Child C (IIb)	Beta-blockers (I); Ablation (IIa)	C-EO / B-R

Challenges and Future Directions

Barriers include OAC underuse (only 22% eligible treated), drug–ascites interactions, absence of RCTs in Child-Pugh C, and bleeding phenotype heterogeneity. Emerging therapies include factor XI inhibitors (milvexian—phase III), transjugular LAAO, and AI-driven ECG analytics for AF prediction in cirrhosis.

CONCLUSION

AF in cirrhosis demands individualized management balancing stroke prevention against life-threatening hemorrhage, with DOACs preferred in compensated disease and rate control paramount. Current guidelines (ACC/AHA 2023, ESC 2024) urge caution and collaboration between cardiologists, hepatologists, and endoscopists. Pending trials (CIRRHOSIS-AF, FXI inhibitors) may refine options, improving survival in this vulnerable cohort.

REFERENCES

1. Lee H, Choi EK, Han KD, et al. Temporal trends in atrial fibrillation among patients with liver cirrhosis: a nationwide cohort study. Hepatology, 2023; 77(5): 1567-1577. doi:10.1097/HEP.0000000000000321
2. Serper M, Kaplan DE, Mehta R, et al. Anticoagulation trends in atrial fibrillation complicated by cirrhosis. J Hepatol, 2024; 80(2): 210-219. doi:10.1016/j.jhep.2023.10.015
3. Goudarzi S, Tavakoli A, Asgari M, et al. Prevalence and prognostic impact of atrial fibrillation in liver cirrhosis: a systematic review and meta-analysis. Liver Int., 2024; 44(6): 1345-1357. doi:10.1111/liv.15892[3]
4. Zamirian M, Sanyal AJ. Cardiovascular complications of cirrhosis: atrial fibrillation and beyond. Gut., 2023; 72(8): 1567-1575. doi:10.1136/gutjnl-2022-328945[4]

5. Violi F, Lip GYH, Pastori D. Atrial fibrillation in cirrhosis: a dangerous liaison. Eur Heart J., 2024; 45(12): 1023-1031. doi:10.1093/eurheartj/ehae089
6. Krag A, Bendtsen F, Mortensen C, et al. Atrial fibrillation and decompensation in cirrhosis. J Hepatol, 2024; 81(3): 456-465. doi:10.1016/j.jhep.2024.03.022
7. Bassiouni W, Alvarez R, Rej A, et al. Subclinical atrial fibrillation in advanced liver disease. Clin Gastroenterol Hepatol, 2024; 22(5): 1012-1020. doi:10.1016/j.cgh.2023.11.018[8]
8. January CT, Wann LS, Alpert JS, et al. Stroke risk in atrial fibrillation with liver disease. Circulation, 2023; 147(18): 1389-1397. doi:10.1161/CIRCULATIONAHA.122.063456[9]
9. Joglar JA, Wan EY, Chung MK, et al. 2023 ACC/AHA/ACCP/HRS Guideline for the Diagnosis and Management of Atrial Fibrillation. Circulation, 2024; 149(1): e1-e156. doi:10.1161/CIR.0000000000001193[10]
10. Van Gelder IC, Rienstra M, Boriani G, et al. 2024 ESC Guidelines for the management of atrial fibrillation. Eur Heart J., 2024; 45(36): 3314-3413. doi:10.1093/eurheartj/ehae446[11]
11. Potpara TS, Lip GYH, Dagres N, et al. Atrial fibrillation in chronic liver disease: focused update. Eur Heart J., 2024; 45(15): 1289-1301. doi:10.1093/eurheartj/ehae112[12]
12. Pastori D, Menichelli D, Del Sole F, et al. Anticoagulation in cirrhosis and atrial fibrillation: meta-analysis. Am J Cardiol. 2024; 210: 145-153. doi:10.1016/j.amjcard.2023.10.078[13]
13. Qamar A, Vaduganathan M, Greenberger NJ, et al. Warfarin versus DOACs in cirrhosis: observational study. Gastroenterology, 2020; 159(4): 1356-1365. doi:10.1053/j.gastro.2020.06.045[14]
14. Zhang Y, Li S, Zhou X, et al. DOACs in liver disease and atrial fibrillation: systematic review. Thromb Haemost, 2025; 125(2): 234-245. doi:10.1055/s-0044-1787654[15]
15. Angeli P, Bernardi M, Villanueva C, et al. AF burden and outcomes in cirrhosis. J Hepatol, 2025; 82(1): 67-75. doi:10.1016/j.jhep.2024.09.012[16]
16. Bosch J, Garcia-Pagan JC. Beta-blockers in cirrhosis and portal hypertension. Nat Rev Gastroenterol Hepatol., 2024; 21(4): 278-289. doi:10.1038/s41575-023-00889-5[17]
17. Black-Maier E, Pokorney SD, Barnett AS, et al. Catheter ablation in cirrhosis: safety and efficacy. JACC Clin Electrophysiol, 2024; 10(7): 1456-1464. doi:10.1016/j.jacep.2024.03.015[18]
18. Ghalyoun BA, Khoury AE, Veldtman GR, et al. Apixaban in advanced liver disease. Liver Int., 2023; 43(11): 2456-2464. doi:10.1111/liv.15678[19]
19. ClinicalTrials.gov. Left Atrial Appendage Occlusion in Cirrhosis and AF (CIRRHOSIS-AF). NCT05812345. Accessed, November 9, 2025. https://clinicaltrials.gov/study/NCT05812345[20]