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Review Article



ATRIAL FIBRILLATION IN CRITICAL ILLNESS: A COMPREHENSIVE REVIEW OF DIAGNOSIS, HEMODYNAMIC IMPACT, AND MANAGEMENT STRATEGIES

AWAD AHA^{1*}, ABDELBAKY AM¹, ELMASRY WG¹, PERVAIZ R², SARWAR T²



¹Rashid Hospital, Dubai Health, Dubai, UAE ²Bahria International Hospital, Lahore, Pakistan *Corresponding author email address: ahmedhossam79@gmail.com



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ABSTRACT

Background: Atrial fibrillation (AF) is the most common sustained arrhythmia observed in critically ill patients and is linked to substantial morbidity and mortality. In the intensive care setting, AF frequently reflects systemic physiological stress and may exacerbate hemodynamic instability, predisposing patients to organ dysfunction and thromboembolic events. New-onset AF (NOAF) is of particular concern, as it often signifies worsening severity of illness in conditions such as sepsis, multi-organ failure, or major postoperative states. **Objective:** To review the clinical significance, epidemiology, diagnostic workup, associated complications, and evidence-based management strategies for atrial fibrillation in critically ill patients, with particular emphasis on hemodynamic stabilization, rate and rhythm control, and individualized anticoagulation. Study Design: Narrative review. Setting: Intensive care unit (ICU) and critical care populations. Duration of Study: last six years literature was searched. Methods: This narrative review synthesizes current evidence, contemporary guideline recommendations, and major studies addressing the epidemiology, pathophysiology, hemodynamic consequences, diagnostic evaluation, and therapeutic approaches for AF in the ICU. Literature from critical care, cardiology, and electrophysiology sources was examined to outline best practices and emerging perspectives. Results: Atrial fibrillation in the ICU is consistently associated with increased mortality, prolonged hospital and ICU length of stay, and a heightened risk of stroke and organ dysfunction. Diagnostic evaluation relies on electrocardiography, transthoracic echocardiography, and targeted laboratory testing to identify reversible precipitants, including sepsis, electrolyte abnormalities, and hypovolemia. Rate control is the preferred initial strategy in hemodynamically stable patients, commonly employing beta-blockers or calcium channel blockers. Rhythm control, most frequently using amiodarone, may be advantageous in patients with persistent hypotension or inadequate rate control. Anticoagulation provides substantial protection against thromboembolic events, although decisions must balance stroke risk with bleeding risk, renal function, and planned procedures. Use of standardized scores such as CHA₂DS₂-VASc and HAS-BLED supports individualized decision-making. Conclusion: Atrial fibrillation in critically ill patients serves as a marker of physiological deterioration and is an independent predictor of adverse outcomes. Optimal management requires prompt identification, correction of underlying triggers, evidencebased rate or rhythm control strategies, and carefully individualized anticoagulation. Further research is needed to develop ICU-specific AF management algorithms and to clarify the long-term prognostic implications of new-onset AF.

Keywords: Atrial Fibrillation, Critical Illness, ICU Management, Rate Control, Anticoagulation

Atrial fibrillation (AF) represents the most frequently encountered arrhythmia in critically ill patients, posing substantial diagnostic and therapeutic challenges. In intensive care units (ICUs), the condition often reflects a complex interplay between underlying systemic illness, hemodynamic derangements, and inflammatory or metabolic disturbances. The management of AF in this setting differs significantly from that in stable outpatient populations, as critically ill patients often require rapid stabilization, individualized therapeutic planning, and careful consideration of evolving physiologic status. This review provides a comprehensive, detailed narrative on the clinical significance, diagnostic evaluation, epidemiology, consequences, underlying mechanisms, and management strategies for AF in critically ill patients, with particular focus on rate and rhythm control and anticoagulation protocols.

Clinical Significance and Impact

AF carries important prognostic implications in critically ill patients. Its presence has consistently been associated with increased morbidity and mortality, largely because it often signals clinical deterioration or the presence of severe systemic disorders. Studies indicate that AF increases the risk of stroke by at least twofold and contributes substantially to overall mortality in ICU patients (1,2). New-onset AF (NOAF) in particular is considered a marker of acute physiological stress and is commonly observed in conditions such as septic shock,

with reported incidences reaching up to half of affected individuals, and in postoperative cardiac surgery patients, where rates range between 30 and 40 percent (3,4). Unlike chronic AF observed in the community, NOAF in the ICU is frequently transient but may still result in significant adverse outcomes due to the unstable nature of critically ill patients.

Diagnostic Evaluation

The diagnostic evaluation of AF in ICU settings requires prompt and precise assessment. The electrocardiogram (ECG) remains the fundamental tool for confirming the diagnosis, distinguishing AF from atrial flutter, and assessing the ventricular response rate. Continuous ECG monitoring is particularly useful in critically ill patients due to the high likelihood of intermittent or paroxysmal episodes. Echocardiography plays a crucial role in the evaluation, providing essential information on left atrial dimensions, ventricular systolic and diastolic function, and the presence of structural heart disease that may influence therapeutic decisions (5,6). Furthermore, it helps clinicians determine whether valvular abnormalities, ventricular dysfunction, or elevated filling pressures contribute to the onset or maintenance of AF. Laboratory investigations, including electrolyte, thyroid function, inflammatory markers, and arterial blood gas assessments, are often warranted given the frequent metabolic disturbances observed in ICU patients.

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Classification of Atrial Fibrillation

AF encountered in critically ill patients may be categorized into paroxysmal, persistent, or permanent forms, each with particular diagnostic and therapeutic implications. Paroxysmal AF is commonly observed in ICU patients as a brief, self-terminating arrhythmia triggered by underlying systemic stressors such as hypoxia, electrolyte abnormality, catecholamine excess, or systemic inflammation (1). Persistent and permanent AF, on the other hand, tends to be associated with longstanding structural cardiac abnormalities. However, in the ICU, even permanent AF may become clinically significant due to rapid ventricular rates or hemodynamic compromise, necessitating urgent management.

Epidemiology in the ICU

AF is the most common arrhythmia reported in ICU settings, with incidence rates ranging from 5 to 20 percent depending on the population studied and severity of illness (3,7). NOAF is more prevalent among older adults, patients with pre-existing cardiovascular disease, and those experiencing septic episodes or recovering from major surgeries. The inflammatory cascade, catecholamine surge, and autonomic imbalance observed in these patients have been identified as key contributors to arrhythmogenesis (8,9). Importantly, the onset of AF in the ICU is associated with prolonged mechanical ventilation, longer duration of ICU and hospital stay, and heightened long-term mortality.

Consequences of Atrial Fibrillation in Critical Illness

The consequences of AF in critically ill patients extend far beyond tachyarrhythmia. AF has been consistently linked with increased mortality, with studies reporting a 2 to 5-fold rise in death rates among ICU patients with AF compared to those without it (10,11). Hemodynamic instability is a prominent feature, particularly when rapid ventricular rates compromise ventricular filling and reduce cardiac output. Mean arterial pressure may fall below 65 mmHg, resulting in systemic hypoperfusion (12,4). In patients with pre-existing heart failure, AF frequently exacerbates left ventricular dysfunction and may precipitate pulmonary edema or cardiogenic shock (4,13). Moreover, the loss of atrial systole, particularly in patients with diastolic dysfunction, worsens hemodynamics.

Myocardial ischemia can develop secondary to impaired coronary perfusion and increased myocardial oxygen consumption due to rapid ventricular response (14). AF has also been associated with increased risks of acute kidney injury, hepatic congestion, and encephalopathy due to systemic hypoperfusion and microcirculatory disturbances

(15,3). Cardioembolic complications, including ischemic stroke, represent a significant threat, particularly in patients with prolonged or recurrent AF episodes, coexisting infection, or mechanical ventilation (16,17).

ATRIAL FIBRILLATION IN CRITICAL ILLNESS

PATHOPHYSIOLOGY

- · Systemic inflammation
- Catecholamine excess
- · Loss of atrial kick
- Tachycardia-induced ischemia

DIAGNOSTIC WORKUP

- ECG (confirmation, rate assessment)
- Echocardiography (LA size, LV function)
- · Labs: electrolytes, thyroid function, ABGs

MANAGEMENT STRATEGY

- Rate control: beta-blockers, calcium channel blockers
- Rhythm control; amiodarone
- Anticoagulation: DOACs or VKAs based on patient profle
- Correction of underlying causes (sepsis, hypoxia, electrolyte imbalance)

OUTCOMES & PREVENTION

- Early identification of new-onset atrial fibrillation improves outcomes
- Use risk scoring (CHA,DS,-VASc, HAS-BLED)
- Adequate anticoagulation reduces stroke risk

Figure 1: Graphical summary

Table 1: Hemodynamic Effects of AF in Critically Ill Patients

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Mechanism	Hemodynamic Effect	Clinical Consequence			
Loss of atrial contraction	Reduced ventricular filling	Low cardiac output, hypotension			
Rapid ventricular response	Shortened diastolic filling time	Myocardial ischemia, worsening heart failure			
Impaired coronary perfusion	Reduced myocardial oxygen delivery	Chest pain, arrhythmia worsening			
Ventricular rate variability	Beat-to-beat stroke volume changes	Hemodynamic instability			
Tachycardiomyopathy	Progressive LV dysfunction	Cardiogenic shock, pulmonary edema			

Underlying Mechanisms of Complications

Several pathophysiologic mechanisms explain the heightened risk of complications associated with AF in critically ill patients. The loss of atrial contraction reduces ventricular preload and stroke volume, particularly in patients who depend on the atrial kick for adequate cardiac output (18). High ventricular rates increase myocardial oxygen demand and shorten diastolic filling time, thereby impairing

coronary perfusion and potentially leading to myocardial ischemia (19). Another important mechanism is tachycardiomyopathy, characterized by ventricular dysfunction caused by prolonged episodes of rapid AF. This dysfunction may be reversible if sinus rhythm is restored in time (20). These factors underscore the importance of early recognition and control of AF in critically ill individuals.

Table 2: Predictors, Mechanisms, and Clinical Outcomes of AF in Critically Ill Patients

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Domain	Description		
Common Predictors	Advanced age, sepsis, electrolyte imbalance, major surgery, cardiovascular disease		
Pathophysiologic Mechanisms	Loss of atrial contraction, tachycardia-induced ischemia, inflammation, and catecholamine surge		
Major Clinical Outcomes	Hemodynamic instability, heart failure, acute kidney injury, encephalopathy, stroke, and increased mortality		

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Management of Hemodynamic Instability and Stability

Management strategies for AF in critically ill patients depend largely on the hemodynamic status and underlying causes. In hemodynamically unstable patients, immediate interventions are required. Hemodynamic instability often necessitates synchronized electrical cardioversion, especially when AF is the primary cause of hypotension or shock. Simultaneously, clinicians must correct reversible contributors such as sepsis, hypoxia, electrolyte imbalance, or ongoing bleeding.

In hemodynamically stable patients, rate control is generally preferred as the initial strategy. Beta-blockers and calcium channel blockers are commonly used to reduce the ventricular rate and improve hemodynamics (21). Beta-blockers tend to be more effective when sympathetic overactivity drives AF onset, whereas calcium channel blockers like diltiazem may be useful in patient's intolerant to beta-

blockade. Digoxin may be considered in patients with severe left ventricular dysfunction, though its utility is limited in high-adrenergic states common in critically ill patients.

Rhythm control strategies may be considered in selected stable patients, especially when AF contributes to ongoing hemodynamic compromise or when rapid restoration of sinus rhythm is desired. Amiodarone remains the preferred antiarrhythmic agent in ICU settings due to its effectiveness and favorable hemodynamic profile. Its intravenous formulation is particularly useful for achieving rapid chemical cardioversion (22). In contrast, agents such as flecainide or propafenone are generally avoided due to proarrhythmic risks. Decisions regarding rhythm control must be individualized, taking into account patient comorbidities, the duration of AF, and the likelihood of recurrence.

Table 3: Key Differences Between Rate Control and Rhythm Control in ICU Patients

Feature	Rate Control	Rhythm Control
Primary Goal	Control the ventricular response	Restore sinus rhythm
Common Agents	Beta blockers, calcium channel blockers	Amiodarone
Indications	Hemodynamically stable patients	Hemodynamic compromise or persistent symptoms
Advantages	Generally safer in unstable conditions	Rapid restoration of atrial contribution to cardiac output
Limitations	May not improve symptoms fully	Risk of drug toxicity or arrhythmia recurrence

Long-Term Management and Anticoagulation

Anticoagulation represents a vital aspect of AF management due to the risk of thromboembolism. In critically ill patients, however, anticoagulation decisions must be carefully individualized, balancing thrombotic risks against the substantial bleeding risks present in this population. Direct oral anticoagulants (DOACs) have gained widespread acceptance due to their predictable pharmacokinetics, fewer interactions, and lack of routine monitoring requirements. They are often preferred over traditional vitamin K antagonists in stable ICU patients (23, 24). Nevertheless, renal impairment, hepatic dysfunction, and drug interactions may limit their use.

The choice of anticoagulation must consider renal function, procedural requirements, bleeding risk, platelet counts, and hepatic metabolism (25, 24). Risk stratification tools such as the CHA2DS2-VASc and HAS-BLED scores provide useful guidance for individualized anticoagulation planning (26, 27). Evidence suggests that early initiation of anticoagulation in NOAF can significantly reduce the risk of thromboembolic events and improve outcomes (28). However, in patients with active bleeding, severe thrombocytopenia, or recent major surgery, anticoagulation may need to be temporarily withheld.

Table 4: Anticoagulation Considerations in ICU Patients with AF

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Factor	Consideration	Clinical Implication		
Renal function	DOACs require renal dose adjustment	Risk of drug accumulation		
Bleeding risk	Recent surgery, thrombocytopenia, coagulopathy	May require temporary withholding		
Drug interactions	Vasopressors, antibiotics, antifungals	May alter DOAC/VKA metabolism		
Need for procedures	Invasive lines, surgeries	Timing of anticoagulation interruption		
Stroke risk	CHA2DS2-VASc scoring	Guides the decision to initiate anticoagulation		

CONCLUSION

The management of AF in critically ill patients requires a nuanced, multidisciplinary approach grounded in an understanding of the underlying pathophysiology and the unique challenges inherent to ICU care. AF in this population is not merely an arrhythmia but a marker of systemic instability and an independent contributor to adverse outcomes. Prompt diagnostic evaluation, individualized rate or rhythm control, careful hemodynamic monitoring, and judicious anticoagulation are essential to reducing complications and improving prognosis. Future research should focus on developing standardized ICU-specific protocols for AF management and on exploring the long-term outcomes of NOAF.

DECLARATIONS

Data Availability Statement

All data generated or analysed during the study are included in the manuscript.

Ethics approval and consent to participate

Not Applicable

Consent for publication

Approved

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Not applicable

CONFLICT OF INTEREST

The authors declare no conflict of interest.

AUTHOR CONTRIBUTION

AHMED HOSAMELDIN AHMED AWAD:

Conceptualized the review, provided expert guidance, and critically revised the manuscript.

AHMED M. ABDELBAKY:

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Contributed to methodology development, interpreted evidence, and reviewed the manuscript.

WAEL GHALY ELMASRY:

Conducted the literature review, validated extracted data, and assisted in manuscript editing.

RIZWAN PERVAIZ:

Supported data verification and contributed to reviewing and refining the manuscript.

TAYYABA SARWAR:

Assisted in literature search, organized extracted data, and prepared tables and figures.

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