World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(REVIEW ARTICLE)

ATRIAL FIBRILLATION IN THE MIDDLE EAST: THE EPIDEMIOLOGICAL SHIFT FROM RHEUMATIC TO METABOLIC AF

Anmar Mandourah *

Medical emergency services department, College of al-Qunfudah health sciences, Umm Al-Qura University.
* Corresponding Author

ORCID Details
Anmar Mandourah: https://orcid.org/0009-0002-0061-5542

World Journal of Biology Pharmacy and Health Sciences, 2026, 28(02), 108–115

Publication history: Received on 27 September 2026; revised on 05 November 2026; accepted on 07 November 2026

Article DOI: https://doi.org/10.30574/wjbphs.2026.28.2.0543

Copyright © 2026 Author(s) retain the copyright of this article. This article is published under the terms of the Creative Commons Attribution License 4.0

ABSTRACT

Background: There is a significant epidemiological transition in atrial fibrillation (AF) in the Middle East. Historically, rheumatic heart disease (RHD) has provided a significant AF substrate by damaging the mitral valve, overloading atrial pressures, and causing fibrosis. Recent regional statistics reveal a declining trend in RHD, with obesity, diabetes, hypertension, and metabolic dysfunction-related steatotic liver disease (MASLD) on the rise. These disorders produce an alternative atrial substrate which frequently occurs in the absence of a serious valve disease.

Methods: The systematic review of the literature was performed with PubMed as the main database and Google Scholar as the auxiliary search and citation management tool. The literature published after 2021 and up to September 2026 was in scope. Search terms: atrial fibrillation, Middle East, MENA, Saudi Arabia, Gulf, rheumatic heart disease, obesity, diabetes, metabolic syndrome, MASLD, fatty liver, and atrial remodeling. The number of publications that were retained was eleven since they covered the regional disease trends, Saudi metabolic risk, AF mechanisms, Gulf non-valvular AF, or management variation between rheumatic and metabolic AF.

Findings: RHD incidence across the Middle East and North Africa declined by 63.4% between 1990 and 2019.[1] During the same period, high body mass index exposure increased by 76.0%, while high fasting plasma glucose exposure increased by 92.4%.[2] AF and atrial flutter incident cases increased from about 49,900 in 1990 to 145,164 in 2021, while prevalent cases rose from about 474,000 to 1.42 million.[3] Saudi studies show substantial metabolic syndrome and MASLD in early and middle adulthood.[5,6] However, direct longitudinal Saudi evidence **linking these exposures** to AF onset during the fourth and fifth decades remains limited.

Conclusions: The Middle East seems to be shifting to a less rheumatic AF model with an increasing role of metabolic non-valvular AF. RHD has clinical implications due to the presence of rheumatic cohorts that need special anticoagulation and valve evaluation. Future Saudi research is needed to compare rheumatic, metabolic, and mixed AF phenotypes with age, status of the valves, metabolic load, atrial morphology, and clinical outcome.

Keywords: Atrial fibrillation; Rheumatic heart disease; Metabolic syndrome; Obesity; Diabetes; MASLD; Saudi Arabia; Middle East

1 INTRODUCTION

Atrial fibrillation reflects the cardiovascular and systemic disorders present within each population. In the Middle East, RHD historically provided an important AF substrate through mitral damage, atrial enlargement, fibrosis, and conduction slowing.

That model is becoming less dominant among younger populations. Regional RHD incidence has fallen sharply, while major metabolic exposures have moved in the opposite direction.[1,2] AF burden has also increased rather than declined.[3] These opposing trends suggest that the disease process feeding AF is changing. However, this transition is rarely a total epidemiological replacement; rather, it introduces a complex "double-hit" state where subclinical rheumatic mechanical damage and systemic metabolic dysfunction co-exist in transitioning demographics. [1,2,3]

Saudi Arabia provides an important setting for studying this transition. Obesity, abdominal adiposity, abnormal glucose metabolism, fatty liver disease, sleep apnea, and hypertension are common before old age.[5,6] Such conditions may subject atrial tissue to decades of metabolic, inflammatory, and hemodynamic strains prior to clinical AF diagnosis.

It is not a total replacement of rheumatic AF. Rather, overlaps cohorts persist: older patients with known valve damage and newer patients more exposed to obesity, diabetes, hypertension, and other metabolic disease.

This review assesses evidence in favor of such a transition. It also discusses the biological disparities between rheumatic and metabolic AF, management implications of mixed cohorts, and gaps in the evidence that need to be bridged before a unique Saudi middle-aged metabolic AF phenotype is established. Crucially, it critically evaluates the epidemiological methodologies used to model these regional shifts and highlights the clinical paradoxes faced by physicians operating under contemporary guidelines.[1,2,3,11]

2 METHODS

This study was designed as a structured narrative review. The narrative design was chosen due to the fact that the research question posits an epidemiological and mechanistic transition, not a single treatment effect that can be analyzed using pooled statistics. The evidence comprises analyses of the Global Burden of Disease, observational cohorts, randomized trials, mechanistic synthesis, and clinical guidance.

The primary database was PubMed, and Google Scholar was utilized as an additional source of search and reference management. The records of the publisher and DOI verified the publication and access. The search was conducted between January 2021 and September 2026.

Search combinations included “atrial fibrillation AND Middle East,” “atrial fibrillation AND Saudi Arabia,” “atrial fibrillation AND Gulf,” “rheumatic heart disease AND MENA,” “obesity AND atrial fibrillation,” “metabolic syndrome AND Saudi Arabia,” “MASLD AND Saudi Arabia,” and “fatty liver AND atrial fibrillation.” Additional searches addressed epicardial fat, atrial remodeling, anticoagulation, and risk-factor management.

Studies were included when they contributed to at least one of five evidence domains. These domains were RHD trends, contemporary regional AF burden, Saudi metabolic disease, biological mechanisms of metabolic AF, and clinical management differences between rheumatic and non-valvular AF. Priority was given to regional population studies, Saudi primary research, large cohort analyses, systematic reviews, and studies with clear clinical relevance.

One randomized trial was included because it directly defines anticoagulation in rheumatic AF.[4] One major guideline was included because it integrates metabolic risk management into contemporary AF care.[11] Case reports, non-peer-reviewed summaries, and studies older than five years were excluded. Eleven publications formed the final evidence set. While narrative in synthesis, the data extraction rigorously evaluated study design limitations, specifically focusing on the algorithmic modeling assumptions of Global Burden of Disease (GBD) datasets versus real-world prospective registries. [1-3,10]

Table 1: Evidence Included in the Structured Narrative Review

Study	Design/Setting	Main Contribution	Methodological/Analytical Considerations
Safiri et al, 2023[1]	MENA; GBD analysis	Defines declining RHD burden	Uses DisMod-MR 2.1 Bayesian modeling; subject to data-smoothing and regional coding variations.
Malekpour et al, 2023[2]	MENA; GBD analysis	Defines rising metabolic exposure	Evaluates Summary Exposure Values (SEVs); modeled estimates rather than direct cohort tracking.
Ghayada et al, 2026[3]	NAME; GBD analysis	Defines increasing AF/AFL burden	Reports age-standardized rates (ASIR/ASPR); highlights male-female outcome disparities.
Connolly et al, 2022[4]	Multinational randomized trial	Defines anticoagulation in rheumatic AF	Definitive RCT trial comparing Rivaroxaban vs VKA in echocardiographically proven RHD.
Eldakhakhny et al, 2023[5]	Saudi cross-sectional study	Shows early metabolic syndrome	Primary care cross-sectional sampling; establishes onset of central adiposity in early 30s.
Alqahtani et al, 2026[6]	Saudi multicenter cohort	Defines contemporary MASLD burden	Vibration-controlled transient elastography (VCTE) validated prospective cohort.
Younossi et al, 2024[7]	MENA systematic review	Places MASLD in regional context	Comprehensive meta-analysis highlighting high T2DM-MASLD overlap.
Jaiswal et al, 2023[8]	Meta-analysis	Links fatty liver disease with AF	Large pooled observational cohort (>18M patients); demonstrates HR 1.42 correlation.
Gawałko et al, 2023[9]	Mechanistic review	Explains adiposity-related atrial remodeling	Translational molecular review detailing epicardial fat secretome and NLRP3 inflammasome pathways.
El Kadri et al, 2025[10]	UAE prospective registry	Describes Gulf non-valvular AF	Real-world observational cohort showing NOAC preference and younger NVAF presentation.
Van Gelder et al, 2024[11]	ESC guideline	Defines modern risk-factor management	Introduces AF-CARE framework, elevating comorbidity management to Class I recommendations.

2.1 Declining Rheumatic Disease Without Immediate Disappearance of Rheumatic AF

The reduction in RHD is one of the clearest changes in Middle Eastern cardiovascular epidemiology. Safiri et al. reported that regional annual RHD incidence fell by 63.4% between 1990 and 2019.[1] The age-standardized death rate also declined by 63.4%, while the disability-adjusted life-year rate fell by 61.3%.

These transformations suggest a reduction in the number of younger patients who are getting into the classic rheumatic pathway. But prevalence did not fall in parallel. In 2019, the age-standardized prevalence was 388.9 cases per 100,000 people, which is a little higher than it was in 1990; hence the inability to regard RHD as a problem of the past only.[1] This paradox—falling incidence alongside persistent prevalence—underscores the prolonged survival of historical RHD cohorts due to improved healthcare access, surgical valve interventions, and secondary penicillin prophylaxis, rather than a failure of primary prevention. [1]

The valve damage may last decades following acute rheumatic fever. Mitral stenosis, mitral regurgitation, pulmonary hypertension, and atrial enlargement can be survived by patients who were exposed at earlier ages. These organizational developments remain in favor of AF as new cases of RHD are becoming less frequent.

The clinical difference is still significant. The article by Connolly et al. examined 4,531 patients who suffered AF related to RHD in the INVICTUS trial.[4] The mean age was 50.5 years. Vitamin K antagonist therapy produced better cardiovascular outcomes than rivaroxaban without increasing major bleeding.

This result has direct relevance to transitional Middle Eastern cohorts. A middle-aged patient with rheumatic mitral stenosis cannot automatically follow the same anticoagulation pathway as a patient with obesity-related non-valvular

AF. The mechanism of AF still influences treatment after the rhythm diagnosis is established. Furthermore, it complicates regional clinical decision-making: in transitional MENA populations where subclinical rheumatic valve damage may co-exist with rapid metabolic weight gain, misclassifying valvular AF as non-valvular AF risks severe thromboembolic failure under direct oral anticoagulant (DOAC) regimens. [1,4,11]

2.2 Rising Metabolic Risk and AF Burden

The decline of RHD has occurred during rapid growth in metabolic risk. Malekpour et al. evaluated metabolic exposures across North Africa and the Middle East from 1990 to 2019.[2] Age-standardized exposure to high fasting plasma glucose increased by 92.4%, while exposure to high BMI increased by 76.0%.

The burden attributable to these risks also moved unfavorably. Age-standardized mortality linked to high fasting plasma glucose increased by 21.4%, while its disability-adjusted life-year rate increased by 27.0%.[2] These trends show that metabolic disease is not only becoming more common; it is also producing substantial clinical harm.

AF burden rose during the same wider period. Ghayada et al. found that all-age AF and atrial flutter incident cases increased by 190.9% from 1990 to 2021.[3] Prevalent cases increased by 198.8%, and mortality increased by more than 230%. Age-standardized incidence and prevalence also showed upward regional trends, especially among men.[3] Interestingly, while men exhibited higher age-standardized incidence and prevalence rates, Ghayada et al. revealed that women bore a disproportionately higher burden of AF-related mortality (ASMR EAPC 1.12 vs 0.25 in males) and disability-adjusted life years, a gender disparity projected to persist through 2031. [3]

Population growth and aging explain part of these increases, but not the entire pattern. The rise in age-standardized measures indicates an additional epidemiological component. These trends support a transition in the disease environment surrounding AF. They do not prove that metabolic disease caused every additional case because Global Burden of Disease analyses are modeled estimates. Still, the simultaneous fall in RHD incidence and rise in metabolic exposure provides a strong population-level signal.

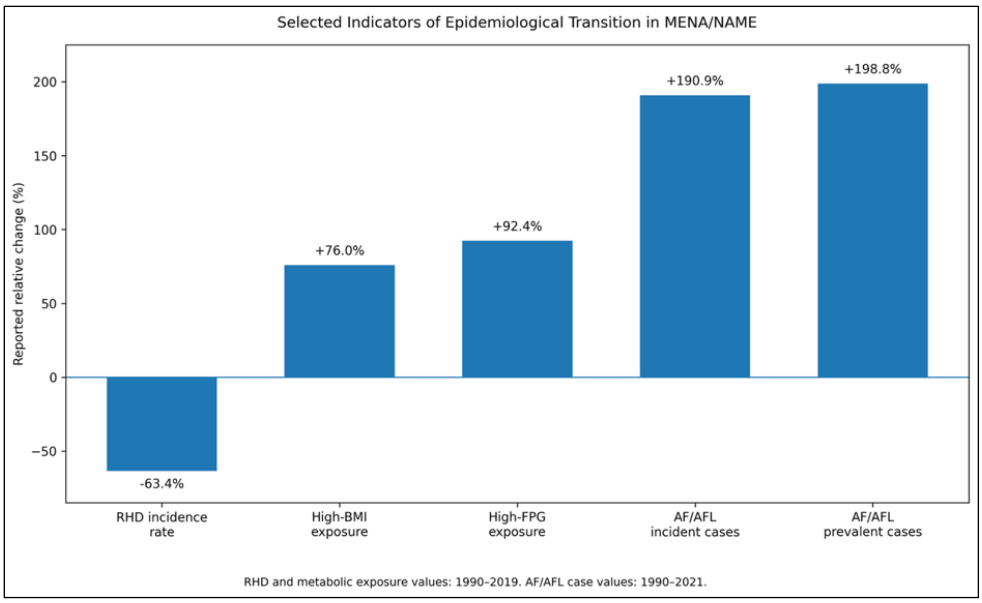

Figure 1: Selected indicators of epidemiological transition in the Middle East and North Africa. RHD annual incidence declined by 63.4% from 1990 to 2019.[1] High-BMI and high-FPG summary exposure values increased by 76.0% and 92.4%, respectively.[2] AF/AFL incident and prevalent case counts increased by 190.9% and 198.8% from 1990 to 2021.[3] The plotted measures are different epidemiological metrics and are shown together only to demonstrate the direction and scale of change.

2.3 Saudi Arabia and Early Metabolic Substrate Formation

Saudi evidence suggests that metabolic exposure begins well before AF is usually diagnosed. Eldakhakhny et al. studied 1,339 non-diabetic adults recruited from public health centers in Jeddah.[5] Metabolic syndrome was already present in 16% of this selected population. Age was the strongest predictor, with risk increasing around the early thirties.

Above this age, increased waist circumference became a dominant abnormality in both sexes.[5] A person who develops central obesity and hypertension in early adulthood may accumulate prolonged atrial pressure loading and

inflammatory exposure before AF appears on an electrocardiogram. This early-onset metabolic trajectory creates a prolonged subclinical 'remodeling window' spanning 15 to 25 years. During this period, chronic low-grade systemic inflammation, hyperinsulinemia, and subtle volume overload continuously compromise atrial structural and electrical integrity long before the patient presents with clinically overt arrhythmia. [2,5,8,9]

Saudi MASLD data reinforce this concept. Alqahtani et al. studied 2,979 adults across seven primary care centers.[6] Mean age was 41.9 years. MASLD was present in 37.3% of participants, with an age- and sex-adjusted prevalence of 34.2%.

Diabetes, increased waist circumference, and high obstructive sleep apnea risk were independently associated with MASLD.[6] These factors overlap closely with established AF risk pathways. The liver findings therefore act as a marker of broader metabolic injury rather than an isolated hepatic problem.

Regional evidence shows a similar pattern. Younossi et al. estimated MENA MASLD prevalence at 39.43% in the general population.[7] Among people with type 2 diabetes, prevalence reached 68.71%. Estimated prevalence also increased across successive study periods, supporting continued expansion of the metabolic substrate. Modelled projections from this meta-analysis indicate over 141.5 million individuals live with MASLD across the MENA region, driven heavily by rapid urbanization, physical inactivity, and dietary transitions, establishing the Gulf region as a global epicenter for metabolic cardiovascular risk. [2, 7]

2.4 Fatty Liver Disease and AF

Fatty liver disease may help identify patients with advanced metabolic risk. Jaiswal et al. pooled 12 cohort studies involving more than 18 million participants.[8] NAFLD, the terminology used in the included studies, was associated with a 42% greater risk of AF. It was also linked with greater risks of heart failure and stroke.

The correlation does not support the idea that hepatic fat is the cause of AF. Upstream drivers are also similar in both conditions, such as visceral obesity, insulin resistance, hypertension, oxidative stress, and chronic inflammation. Fatty liver could thus play a role as an indicator of metabolic severity as well as a potential source of systemic inflammatory signaling. From a mechanistic perspective, the 'liver-heart axis' operates via two distinct pathways: (1) systemic endocrine dysfunction, where the steatotic and inflamed liver secretes pro-inflammatory cytokines (IL-6, TNF-α, C-reactive protein) and pro-fibrotic hepatokines while downregulating cardioprotective adiponectin; and (2) localized epicardial interaction, where systemic dyslipidemia drives ectopic lipid deposition directly into epicardial adipose tissue (EAT) surrounding the atria. This dual hit accelerates fibro-fatty myocardial infiltration and electrical heterogeneity. [8,9]

Such a difference is relevant to Saudi research. MASLD may be included in AF cohorts as a phenotypic indicator as opposed to a comorbidity. A patient who has central obesity, diabetes, MASLD, and sleep apnea probably has a different atrial risk profile compared to that of a patient with the same BMI with minimal metabolic organ damage.

2.5 From Valve-Driven AF to Metabolic Atrial Cardiomyopathy

Rheumatic and metabolic AF are different at the atrial biology level. Rheumatic AF typically starts with a distinguishable mechanical lesion. Mitral stenosis increases left atrial pressure, and regurgitation produces chronic volume loading. Both processes widen the atrium and enhance fibrosis, conduction heterogeneity, and re-entry.

Metabolic AF is more diffuse. No one lesion describes the process. Evidence of the interplay of obesity, hypertension, insulin resistance, sleep apnea, inflammation, and ectopic fat occurs over time.

According to Gawalko et al., adipose tissue is an inflammatory and endocrine organ.[9] Of particular interest is the epicardial adipose tissue, which is directly adjacent to atrial myocardium. It is capable of releasing inflammatory cytokines, adipokines, profibrotic mediators, and reactive oxygen species to the adjacent tissue. Crucially, epicardial adipose tissue (EAT) exhibits a unique, location-specific transcriptomic signature; peri-atrial EAT specifically overexpresses genes governing oxidative phosphorylation, neutrophil infiltration, and calcium signaling, creating a highly pro-inflammatory microenvironment that directly interfaces with the underlying atrial endocardium. [9]

These signals stimulate atrial fibrosis and change the work of cardiomyocytes. Fibrosis slows down the conduction of electricity and enhances heterogeneity of conduction. Autonomic regulation and calcium processing can be disrupted by metabolic disease as well, raising the likelihood of triggered activity and arrhythmia.[9] At the cellular level, metabolic stress accelerates the activation of the NLRP3 inflammasome within atrial cardiomyocytes. Active NLRP3 triggers intracellular caspase-1 cleavage and IL-1β maturation, which directly disrupts sarcoplasmic reticulum calcium handling by increasing diastolic Ryanodine Receptor type-2 (RyR2) leakiness. This calcium instability promotes spontaneous delayed afterdepolarizations (DADs) and early afterdepolarizations (EADs) that trigger AF, while

simultaneously upregulating ultra-rapid delayed-rectifier potassium currents (I_{Kur}) to shorten the effective refractory period (ERP) and sustain re-entrant waves. [9]

Obesity will cause hemodynamic stress due to increased circulating volume and increased filling pressures. Hypertension increases afterload and atrial pressure. Sleep apnea contributes to intermittent hypoxia, sympathetic arousal, and intermittent alterations in intrathoracic pressure. Diabetes adds oxidative stress, glycation damage, microvascular dysfunction, and additional fibrosis.

This model is the reason why rhythm treatment may not be enough. Cardioversion and ablation treat electrical disease, but have no effect on uncontrolled hypertension, obesity, diabetes, sleep apnea, or insulin resistance unless risk factors are also treated. Furthermore, basic research demonstrates that obesity-induced structural and electrophysiological remodeling exhibits striking plasticity; animal models show that a 30% weight reduction can partially reverse EAT infiltration, reduce interstitial fibrosis, and normalize shortened ERPs and conduction velocities, underscoring the necessity of upstream metabolic intervention. [9]

2.6 Contemporary Gulf Non-Valvular AF

The FLOW-AF registry is helpful in providing evidence that non-valvular AF is a proven Gulf clinical issue. El Kadri et al. conducted a prospective study of 198 patients who were recently diagnosed with non-valvular AF in 6 centers in the United Arab Emirates.[10] The mean age was 63.44 years. This average age at presentation is notably nearly a decade younger than typical Western AF registry demographics (where mean age typically exceeds 70 to 75 years), reinforcing that Middle Eastern populations accumulate significant metabolic risk and develop clinical NVAF significantly earlier in life. [10]

More than half received non-vitamin K antagonist oral anticoagulants. One-year all-cause mortality was 6.7%, while reported bleeding occurred in 2.8%.[10] The study is limited and incapable of establishing the incidence of the region, but it captures a current population of Gulf NVAF that can be treated by using the modern anticoagulation pathways. Notably, baseline cardiovascular comorbidity was high in this UAE cohort: 70% had hypertension, 47% had diabetes mellitus, 45% had hypercholesterolemia, and 43.8% were obese. The 47% diabetes prevalence is more than double the ~20% rate observed in Western NVAF registries, highlighting a distinct, highly metabolic regional disease profile. Furthermore, healthcare resource utilization data from the FLOW-AF registry revealed an average annual total management cost of USD $1,209.4 per patient, with antithrombotic pharmacotherapy constituting the primary direct cost driver. [10]

Excessive age restricts exaggeration. Present Gulf data do not indicate AF already focuses on 40-50 age groups. It rather demonstrates that significant AF-producing metabolic risks are accrued earlier.

2.7 Clinical Meaning of Transitional Cohorts

A phenotype-based approach is necessitated by the coexistence of rheumatic and metabolic AF. The underlying substrate cannot be determined by age. Echocardiography is also necessary in case of uncertainty regarding the valve status since moderate or severe rheumatic mitral stenosis alters the choice of anticoagulants and overall management. The 2024 ESC Guidelines explicitly reinforce this by issuing a Class I (Level C) recommendation for transthoracic echocardiography in all patients with newly diagnosed AF to evaluate structural heart disease, valve status, and chamber dimensions to guide treatment pathways. [11]

INVICTUS favors the further use of vitamin K antagonist in AF related to RHD.[4] The 2024 European Society of Cardiology guideline also distinguishes most non-valvular AF patients from those with mechanical valves or moderate-to-severe mitral stenosis.[11]

The metabolic pathway requires a different assessment. Weight alone is insufficient. Waist circumference, blood pressure, glucose control, lipid status, sleep apnea, physical activity, and smoking should be evaluated. MASLD may also deserve attention in Saudi patients because of its high national prevalence.[6]

The 2024 ESC guideline places comorbidity and risk-factor management at the beginning of the AF-CARE framework.[11] This represents a major shift in AF treatment. Management is no longer limited to stroke prevention, rate control, rhythm control, and ablation. Under the AF-CARE principles—(C) Comorbidity and risk factor management, (A) Avoid stroke and thromboembolism, (R) Reduce symptoms by rate and rhythm control, and (E) Evaluation and dynamic reassessment—upstream cardiometabolic management is elevated to a foundational Class I recommendation. For example, the guideline now explicitly recommends targeted blood pressure control (target 120–129/70–79 mmHg), SGLT2 inhibitors for heart failure regardless of LVEF (Class I, Level A), effective glycemic control in diabetes (Class I, Level C), and structured weight loss targeting ≥ 10% body weight reduction in obese individuals (Class I, Level B). [11]

For overweight and obese patients, sustained weight reduction can lower AF symptoms and burden.[11] Hemodynamic stress is prevented by blood pressure control. Treatment of diabetes could inhibit the persistence of metabolic damage, and treatment of sleep apnea could decrease nocturnal autonomic and pressure imbalance.

This practice is particularly applicable to Saudi transitional cohorts. An effective rhythm process can be of short-term help when there is still some underlying metabolic substrate to develop. Long-term care should thus consider both the electrical disease as well as the conditions that cause atrial remodeling.

2.8 Research Gaps and a Saudi Research Agenda

The biggest evidence gap is that there is no large study that directly relates age, metabolic phenotype, valve status, and incident AF in Saudi Arabia. The literature determines individual elements of the transition. Local statistics indicate a downward trend in RHD, an upward trend in metabolic risk, and an upward trend in the burden of AF.[1–3] Saudi studies show early metabolic syndrome and high MASLD prevalence.[5,6]

Nevertheless, these data sets cannot be used to establish that the same individuals develop a metabolic disease in early years and develop AF in their forties or fifties. The main hypothesis is biologically robust yet not thoroughly tested.

Newly diagnosed patients should then be classified by age and underlying substrate in a national prospective AF registry. Suggested age groups include 30-39, 40-49, 50-59, 60-69, and 70 years or older. Valve disease, left atrial volume, ventricular function, and other structural results should be documented using standardized echocardiography.

Metabolic phenotyping needs to comprise BMI, waist circumference, blood pressure, HbA1c, fasting glucose, and lipids. Screening for sleep apnea must be included, and MASLD could be assessed by validated imaging or noninvasive liver measurements. Patients were then categorized as rheumatic, metabolic, mixed, and other AF phenotypes.

Follow-up is to investigate recurrence, stroke, heart failure, hospitalization, ablation response, anticoagulant complications, and mortality. Such a design would directly test the presence of metabolic AF coming up earlier and whether the results are different than those of rheumatic AF.

The estimation of regional burden also needs to be validated with the national clinical registries. The modeled data are useful in long term trends but rely on the information available as a source. Further full coverage of the Saudi registry would enhance estimates of AF age of onset, phenotype, sex variation, and outcome.

3 DISCUSSION

The reviewed evidence supports an epidemiological transition, but the phrase “rheumatic to metabolic AF” requires careful interpretation. RHD incidence is falling, while metabolic exposure and AF burden are rising.[1–3] Saudi metabolic disease is already substantial in adults near the age range proposed for a transitional cohort.[5,6]

The biological link is also coherent. Adiposity promotes atrial inflammation, fibrosis, autonomic change, mechanical loading, and electrical instability.[9] Fatty liver disease is associated with increased AF risk and may identify patients with advanced metabolic dysfunction.[8]

Yet the evidence remains incomplete. Global burden studies rely on modeled data. Cross-sectional Saudi metabolic studies cannot establish future AF incidence. The UAE registry confirms contemporary NVAF but is relatively small.[10] No large Saudi longitudinal cohort currently demonstrates that AF incidence is rising specifically among adults aged 40 to 50.

The remaining question is how strongly Saudi metabolic burden is changing AF age, phenotype, response to therapy, and outcomes.

4 CONCLUSION

AF in the Middle East seems to be shifting from a predominantly rheumatic pattern to a mixed disease model. The incidence of RHD has been significantly reduced, but the established rheumatic valve disease is still of clinical significance. Meanwhile, the metabolic substrate of non-valvular AF is growing due to the presence of obesity, abnormality of glucose metabolism, hypertension, MASLD, and other related disorders.

Saudi Arabia demonstrates strong indications of metabolic exposure at a relatively young age. These risks may foster inflammation, fibrosis, hemodynamic strain, autonomic dysfunction, and electrical remodeling in the atria. But there is no evidence so far that shows a rapid increase in AF in Saudi adults aged 40 to 50.

The second study phase must examine the direct relationship between metabolic phenotype and AF development, valve condition, atrial anatomy, and patient outcome. The national registry based on phenotype would be able to determine the existence of a specific younger metabolic AF cohort and would inform earlier screening, prevention, and treatment.

REFERENCES

[1] Safiri S, Mousavi SE, Noori M, Nejadghaderi SA, Sullman MJM, Singh K, et al. The burden of rheumatic heart disease in the Middle East and North Africa region, 1990-2019. Arch Cardiovasc Dis. 2023;116(12):542-554. https://doi.org/10.1016/j.acvd.2023.09.005

[2] Malekpour MR, Abbasi-Kangevari M, Ghamari SH, Khanali J, Heidari-Foroozan M, Saeedi Moghaddam S, et al. The burden of metabolic risk factors in North Africa and the Middle East, 1990-2019: findings from the Global Burden of Disease Study. EClinicalMedicine. 2023;60:102022. https://doi.org/10.1016/j.eclinm.2023.102022

[3] Ghayada I, Hammad B, Shahin M, Abdel Kamel S, Al-Huneidy R, Almazaraa Y, et al. Epidemiologic shift and future projections of atrial fibrillation and flutter in North Africa and the Middle East region: a comprehensive Global Burden of Disease analysis. Heart Rhythm O2. Published online June 12, 2026. https://doi.org/10.1016/j.hroo.2026.06.002

[4] Connolly SJ, Karthikeyan G, Ntsekhe M, Haileamlak A, El Sayed A, El Ghamrawy A, et al. Rivaroxaban in rheumatic heart disease-associated atrial fibrillation. N Engl J Med. 2022;387(11):978-988. https://doi.org/10.1056/NEJMoa2209051

[5] Eldakhakhny B, Enani S, Jambi H, Ajabnoor G, Al-Ahmadi J, Al-Raddadi R, et al. Prevalence and factors associated with metabolic syndrome among non-diabetic Saudi adults: a cross-sectional study. Biomedicines. 2023;11(12):3242. https://doi.org/10.3390/biomedicines11123242

[6] Alqahtani SA, AlMuhaidib S, Sanai FM, Abaalkhail F, Alotaibi HF, Khoja B, et al. Vibration-controlled transient elastography-based prevalence of metabolic dysfunction-associated steatotic liver disease in Saudi Arabia: a prospective cohort study. Clin Gastroenterol Hepatol. Published online July 15, 2026. https://doi.org/10.1016/j.cgh.2026.06.037

[7] Younossi ZM, Golabi P, Paik J, Owrangi S, Yilmaz Y, El-Kassas M, et al. Prevalence of metabolic dysfunction-associated steatotic liver disease in the Middle East and North Africa. Liver Int. 2024;44(4):1061-1070. https://doi.org/10.1111/liv.15852

[8] Jaiswal V, Ang SP, Huang H, Momi NK, Hameed M, Naz S, et al. Association between nonalcoholic fatty liver disease and atrial fibrillation and other clinical outcomes: a meta-analysis. J Investig Med. 2023;71(6):591-602. https://doi.org/10.1177/10815589231164777

[9] Gawałko M, Saljic A, Li N, Abu-Taha I, Jespersen T, Linz D, et al. Adiposity-associated atrial fibrillation: molecular determinants, mechanisms, and clinical significance. Cardiovasc Res. 2023;119(3):614-630. https://doi.org/10.1093/cvr/cvac093

[10] El Kadri M, AlMuti K, Agrawal A, Bazargani N, Gamaleldin MFS, Ahmed HM, et al. The Atrial Fibrillation Registry (The FLOW-AF Registry): insights from the United Arab Emirates—patient characteristics, treatment, and one-year outcomes. J Cardiovasc Electrophysiol. 2025;36(4):813-823. https://doi.org/10.1111/jce.16598

[11] Van Gelder IC, Rienstra M, Bunting KV, Casado-Arroyo R, Caso V, Crijns HJGM, et al. 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2024;45(36):3314-3414. https://doi.org/10.1093/eurheartj/ehae176