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The Role of Magnesium Supplementation in the Treatment of Permanent Atrial Fibrillation: A Literature Review

Oliwia Barbara Gutowska

ORCID <https://orcid.org/0009-0000-7378-7383>

E-mail: oliwia.gutowska.01@gmail.com

Medical University of Gdańsk, Gdańsk, Poland

Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk

Weronika Nowak

ORCID <https://orcid.org/0009-0009-4325-4602>

E-mail: weronikanowak@gumed.edu.pl

Medical University of Gdańsk, Gdańsk, Poland

Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk

Wiktoria Magdalena Kostuch

ORCID <https://orcid.org/0009-0003-8084-0551>

E-mail: wiktoriakostuch@gumed.edu.pl

Medical University of Gdańsk, Gdańsk, Poland

Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk

Łukasz Kowalewski

ORCID <https://orcid.org/0009-0008-4005-2610>

E-mail: lek.lukasz.kowalewski@gmail.com

University Clinical Centre, Gdańsk, Poland

Dębinki 7, 80-952 Gdańsk

Maria Aleksandra Styczyńska

ORCID <https://orcid.org/0009-0005-3583-1271>

E-mail: m.styczynska00@gmail.com

University Clinical Centre, Gdańsk, Poland

Dębinki 7, 80-952 Gdańsk

Amir Naziri

ORCID <https://orcid.org/0009-0009-4465-1685>

E-mail: amirnaziri@o2.pl

University Clinical Centre, Gdańsk, Poland

Dębinki 7, 80-952 Gdańsk

Julianna Rozalia Chyl

ORCID <https://orcid.org/0009-0003-1115-1226>

E-mail: julianna.chyl@gmail.com

Medical University of Gdańsk, Gdańsk, Poland

Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk

Agata Zimon

ORCID <https://orcid.org/0009-0000-6163-841X>

E-mail: agatazimon1@gmail.com

Medical University of Gdańsk, Gdańsk, Poland

Marii Skłodowskiej-Curie 3a, 80-210 Gdańsk

Martyna Zielnik

ORCID <https://orcid.org/0009-0007-9787-0625>

E-mail: martynazie@icloud.com

The John Paul II Western Hospital in Grodzisk Mazowiecki, Grodzisk Mazowiecki, Poland

Daleka 11, 05-825 Grodzisk Mazowiecki

Katarzyna Stróżna

ORCID <https://orcid.org/0009-0007-7614-1108>

E-mail: katarzyna.strozna1@gmail.com

Medical Center of Postgraduate Education, Professor Witold Orłowski Independent Public

Clinical Hospital, Warszawa, Poland

Czeriakowska 231, 00-416 Warszawa

Corresponding Author

Oliwia Barbara Gutowska, e-mail: oliwia.gutowska.01@gmail.com

Abstract

Introduction and objective: Atrial fibrillation (AF) is one of the most common sustained arrhythmias in the population. Growing interest in the impact of electrolyte disturbances on the pathomechanism of AF creates opportunities to evaluate the effectiveness of their supplementation in a therapeutic context.

Aim: To present the current state of knowledge regarding the role of magnesium in the prevention and treatment of AF, particularly in patients with permanent AF.

Materials and Methods: A narrative literature review was conducted using available databases, searching for publications published between 2005 and 2025. After applying the inclusion criteria, selected publications were analyzed, including randomized trials, cohort studies, and review articles.

Results: Magnesium plays an important role in the functioning of the cardiovascular system. It contributes to the electrical stability of cardiomyocytes and influences proper conduction within the cardiac conduction system. Most available publications focus on its role in patients undergoing cardiac surgery, while there is a lack of clear evidence confirming the effectiveness of incorporating magnesium into the standard treatment of permanent AF.

Conclusions: Despite its potential antiarrhythmic properties, magnesium is not included in recommendations for the chronic treatment of AF, and its use is mainly limited to correcting possible deficiencies. The limited availability of high-quality methodological studies indicates the need for further research to determine the role of magnesium in the treatment of permanent AF.

Keywords: atrial fibrillation, magnesium, hypomagnesemia, arrhythmia

Introduction

Clinical significance of atrial fibrillation

Atrial fibrillation (AF) is one of the most common sustained cardiac arrhythmias in the adult population, making it a significant public health concern worldwide. Epidemiological studies conducted in recent years indicate that the number of patients affected by AF continues to increase, accompanied by growing awareness among physicians of various specialties regarding its treatment and prevention [1]. This condition is associated with an increased risk of serious thromboembolic complications, such as ischemic stroke, myocardial infarction, and pulmonary embolism, making it an important health problem across different age groups [2]. The prevalence of this arrhythmia increases with age and with the presence of comorbidities such as hypertension and heart failure, placing patients with AF at a higher-than-population risk of thromboembolic events. Furthermore, AF significantly contributes to a deterioration in quality of life and increased mortality [1]. Ongoing research conducted over many years has improved the understanding of the pathomechanism underlying AF and its clinical implications, while optimization of treatment strategies remains one of the key challenges in contemporary cardiology.

The Role of Electrolytes in the Pathomechanism of Atrial Fibrillation

The pathogenesis of AF is complex and involves both structural and electrical alterations occurring within the atrial myocardium. As a consequence of these changes, atrial remodelling and enlargement develop, contributing to the chaotic activation of cardiac muscle cells and their ineffective contraction [3]. Electrolytes are essential for maintaining the action potential of cardiomyocytes and ensuring proper electrical conduction. Disturbances in water-electrolyte balance, such as changes in potassium, sodium, or calcium concentrations, may significantly affect the resting membrane potential of cardiomyocytes, thereby impairing their normal depolarisation [3]. Another important macronutrient whose abnormal concentration may promote both the development and persistence of arrhythmias is magnesium. It is the second most abundant intracellular cation and acts as a cofactor in numerous enzymatic reactions. Furthermore, it plays a crucial role in regulating the activity of membrane ion channels, making it a potential target for maintaining normal cardiac rhythm [4].

Magnesium, as a potential preventive and therapeutic factor in cardiac arrhythmias, has been the subject of numerous studies over recent years. Research findings suggest that magnesium may exert antiarrhythmic effects through its role in stabilising the membrane potential, modulating calcium ion influx into cardiomyocytes, and influencing the function of the cardiac conduction system [5]. Its role has been particularly well documented in the prevention of AF among patients undergoing cardiac surgery and in the management of AF episodes in hospital settings [6–9]. Despite these observations, the role of magnesium supplementation as an adjunctive treatment in patients with permanent AF remains unclear, and the available evidence is inconsistent [5]. Moreover, current guidelines for the management of permanent AF do not recommend the routine administration of magnesium as a therapeutic strategy, indicating that the available scientific evidence remains insufficient [10]. The discrepancy between research findings and clinical recommendations suggests that the role of magnesium supplementation in the treatment of patients with permanent AF requires further investigation to provide a more comprehensive understanding of the issue.

Aim of the Study and Review Methods

This article constitutes a narrative literature review aimed at analysing current scientific evidence regarding the role of magnesium supplementation in the treatment of permanent atrial fibrillation. The choice of this review format was determined by the limited number of available studies addressing the topic of interest, as well as by substantial methodological differences

and the heterogeneity of the patient populations described. The literature review was conducted using publications available in the online databases PubMed and Scopus. In addition, current recommendations of the European Society of Cardiology (ESC) and supplementary resources such as Google Scholar and ResearchGate were consulted.

The literature search strategy included publications published between 2005 and 2025. The identification and selection of relevant studies were based on the following Polish and English keywords related to magnesium supplementation (“magnesium”, “magnesium supplementation”, “electrolytes”, “magnesium deficiency”) combined with terms referring to atrial fibrillation (“atrial fibrillation”, “permanent atrial fibrillation”, “arrhythmia”, “postoperative atrial fibrillation”) using the Boolean operator AND.

Due to the limited number of studies investigating magnesium supplementation in permanent atrial fibrillation, the present review was not restricted solely to randomised studies. The analysis included original prospective and retrospective studies as well as cohort studies. To provide the theoretical background for the topic under investigation, publications outside the predefined time frame, including review articles, were also cited. The main limitations of the present review include the potential subjectivity associated with the selection and interpretation of the available literature, the high methodological heterogeneity of the analysed studies, and the insufficient amount of data addressing the effectiveness of magnesium supplementation in patients with permanent atrial fibrillation.

Current State of Knowledge

Definition and Classification

AF is considered one of the most common arrhythmias belonging to the group of supraventricular tachycardias. Its characteristic feature is the uncoordinated activation of the atrial myocardium, which is often accompanied by an irregular and rapid ventricular rhythm [11]. Abnormalities in electrical conduction within the atrial conduction system lead to chaotic activation of cardiomyocytes, resulting in reduced haemodynamic efficiency [3]. On the electrocardiogram, fibrillatory (f) waves can be observed, replacing the regular P waves, along with a variable ventricular response.

Various classification systems for AF have been described in the literature, based on the nature of the arrhythmia, its potential triggering factor, or the duration of the episode. The classification according to AF duration includes paroxysmal, persistent, long-standing persistent, and permanent AF [10]. Paroxysmal AF is diagnosed when an episode of arrhythmia

terminates spontaneously or following medical intervention within less than seven days. Persistent AF refers to an arrhythmia lasting longer than seven days that does not resolve spontaneously, whereas permanent AF is defined as a condition in which attempts to restore sinus rhythm have been abandoned and treatment is focused on rate control and the prevention of thromboembolic complications [10]. AF is a progressive disease and may evolve from paroxysmal forms to permanent AF over time. This phenomenon, supported by clinical observations, is associated, among other factors, with the ageing process and the coexistence of comorbidities such as hypertension and diabetes mellitus, which contribute to the development of structural changes within the atrial myocardium [12,13].

Pathophysiology of Atrial Fibrillation

The high prevalence of AF in the population has contributed to a better understanding of its underlying mechanisms. The pathophysiology of AF is complex and multifactorial, requiring a multidisciplinary approach to achieve optimal therapeutic outcomes. A key role is attributed to structural changes occurring within the atrial myocardium as well as within the atrial conduction system, collectively referred to as atrial remodelling [11,14]. The principal changes observed during the remodelling process include atrial hypertrophy and fibrosis. At the cellular level, shortening of the cardiomyocyte action potential and refractory period can be observed, promoting the formation of re-entry circuits and the development of arrhythmias [3,11].

Triggering factors also play an important role in the development of AF. These include ectopic foci generating electrical impulses, most commonly located within the pulmonary veins. Oxidative stress, inflammation, disturbances in calcium homeostasis, and alterations in autonomic nervous system activity are believed to contribute to both the initiation and maintenance of AF [12,15].

Risk Factors and Comorbidities

Advanced age, comorbid conditions—including hypertension, heart failure, and obesity—as well as lifestyle-related factors such as tobacco smoking and excessive alcohol consumption are considered the most important risk factors for the development of AF [1,16]. Electrolyte disturbances are also recognized in the literature as additional factors predisposing individuals to AF. Hypomagnesemia, which frequently coexists with hypokalemia, has been shown to reduce the activity of the sodium–potassium ATPase, thereby increasing cardiomyocyte excitability and impairing conduction within the cardiac conduction system. Consequently, it

may serve both as a triggering factor for the onset of arrhythmia and as a contributor to its progression toward a permanent form [15,17].

Treatment Strategies for Atrial Fibrillation

The 2024 ESC Guidelines [10] emphasize the importance of providing patients with AF comprehensive multidisciplinary care aimed not only at treating the arrhythmia itself but also at preventing risk factors, managing potential complications, and continuously evaluating treatment effectiveness. The main pillars of therapy include symptom reduction through rhythm and rate control strategies, as well as thromboembolic prevention. Depending on the type of AF, treatment may involve antiarrhythmic drugs, interventional procedures, rate-controlling medications, and anticoagulants. For patients diagnosed with permanent AF, the guidelines recommend the use of beta-blockers, digoxin, and calcium channel antagonists, as indicated, to maintain a resting heart rate below 110 beats per minute, in addition to anticoagulant therapy. Magnesium supplementation, however, is not included in standard AF treatment recommendations due to the limited amount of available scientific evidence supporting its potential effectiveness [16].

Magnesium Homeostasis and Its Impact on the Cardiovascular System

Magnesium is the second most abundant intracellular cation and is essential for the regulation of numerous biological processes. It acts as a cofactor in a wide range of enzymatic reactions and contributes to the maintenance of intracellular homeostasis [18]. The adult human body contains approximately 25 g of magnesium, with its major stores located in bone tissue, skeletal muscle, and soft tissues. The regulation of magnesium concentration depends on its absorption within the gastrointestinal tract as well as its excretion and reabsorption in the renal tubules [19]. Disturbances in any of these processes may affect calcium homeostasis and result in either magnesium excess or deficiency, both of which may have important clinical implications.

Due to its pleiotropic effects on the cardiovascular system, magnesium has been the subject of numerous observational and experimental studies. It plays a crucial role in regulating cardiomyocyte contractility, electrical conduction within the myocardium, and vascular tone [20]. Magnesium also exhibits antithrombotic and antioxidant properties, making it a potentially important therapeutic factor supporting cardiovascular function [20,21].

Causes and Clinical Consequences of Hypomagnesemia

Hypomagnesemia is considered a relatively common electrolyte disorder both in the general population and among hospitalized patients [22]. The most common causes of magnesium deficiency include inadequate dietary intake, impaired gastrointestinal absorption, kidney disease, and the use of medications that increase magnesium loss or reduce its absorption, such as diuretics and proton pump inhibitors [19]. Clinically, hypomagnesemia may manifest through neuromuscular and cardiovascular disturbances. Magnesium deficiency can predispose individuals to arrhythmias by impairing the function of the sodium–potassium pump, thereby increasing cardiomyocyte excitability and prolonging the refractory period of cardiac cells [18,22]. Furthermore, hypomagnesemia frequently coexists with other electrolyte abnormalities, particularly hypokalaemia, which may further enhance its arrhythmogenic potential [15].

Understanding the role of magnesium in maintaining normal cardiac conduction highlights the correction of hypomagnesemia as a potential therapeutic target for preserving the electrical stability of the heart. However, further clinical studies are required to confirm the effectiveness of long-term magnesium supplementation and its impact on clinical outcomes [15,16].

Role of Magnesium in the Prevention of Atrial Fibrillation

As an easily available agent with no clinically significant adverse effects, magnesium may represent an important target in the prevention of AF. Its physiological effects on calcium, potassium, and sodium channels enable the regulation of ion transport across the cell membrane, thereby contributing to the restoration of cardiomyocyte electrical stability [4,16]. In addition, magnesium protects myocardial tissue against excessive inflammatory responses through the regulation of free radical generation, reducing the risk of myocardial cell damage and conduction disturbances [20].

Clinical evidence regarding the use of magnesium supplementation as a preventive strategy for AF has been most extensively documented in patients undergoing cardiac surgery and in critically ill patients treated in intensive care units. Postoperative atrial fibrillation (POAF) is one of the most common complications of the early postoperative period and is associated with an increased risk of thromboembolic events, prolonged hospitalization, higher treatment costs, and increased mortality [9,23]. The pathophysiology of POAF is complex and involves interactions between multiple factors, including oxidative stress affecting cardiomyocytes,

autonomic nervous system dysregulation, and electrolyte disturbances, particularly abnormalities in potassium and magnesium levels [4].

Table 1 summarizes selected studies evaluating the effectiveness of magnesium administration as a preventive measure against POAF, however, their findings remain inconsistent. Several randomized controlled trials and meta-analyses suggest that prophylactic magnesium administration may significantly reduce the incidence of POAF. Researchers attribute this effect to the potential role of magnesium in improving the electrical stability of the myocardium and its antiarrhythmic properties related to the modulation of ion channel function [7,23]. Similar results were reported by Shiga et al. [24], whose meta-analysis demonstrated a reduced incidence of POAF in patients undergoing cardiac surgery who received prophylactic magnesium supplementation. Likewise, Fanning et al. [25] observed a lower incidence of POAF in patients undergoing coronary artery bypass grafting (CABG) who received prophylactic magnesium sulphate before surgery. However, not all studies have confirmed the effectiveness of this intervention. Lancaster et al. [26] found no protective effect of magnesium supplementation and, in fact, reported a higher incidence of POAF among patients who received magnesium supplementation. These discrepancies may result from differences in magnesium dosage, timing of administration, route of administration (oral versus intravenous) and the substantial heterogeneity of the study populations. Furthermore, the cited studies differ with respect to endpoint selection and lack standardization in methods used to assess magnesium concentrations. Owing to these methodological limitations, a definitive evaluation of the effectiveness of magnesium in the prevention of POAF remains challenging.

It is also important to consider the highly specific characteristics of the studied populations. Patients undergoing cardiac surgery constitute a particularly heterogeneous group with numerous comorbidities, which further limits the generalizability of the findings to the general population and to patients with chronic forms of AF. Based on the available evidence, magnesium administration may be considered a component of AF prevention in selected clinical settings. Nevertheless, its effectiveness appears to depend on multiple factors, including the route of administration and the presence of concomitant electrolyte disturbances. At the same time, the amount of available evidence concerning patients with chronic AF remains insufficient to draw definitive conclusions, highlighting the need for further research aimed at establishing optimal administration protocols and identifying patient groups most likely to benefit from this intervention.

Table 1. Selected studies evaluating the use of magnesium in the prevention of atrial fibrillation

Study	Study design	Study population	Intervention	Endpoint	Outcome
Gu et al. [7]	Meta-analysis	Patients after CABG (n=1028)	Mg vs placebo	POAF	↓ incidence of AF
Yarnell et al. [8]	Randomized controlled trial	Critically ill ICU patients (n=4198)	Mg	AF within 24 h	↓ incidence of AF
Shiga et al. [24]	Meta-analysis	Patients undergoing cardiac surgery (n=2069)	Mg	POAF	↓ incidence of AF
Fanning et al. [25]	Randomized controlled trial	Patients after CABG (n=99)	Mg	POAF	↓ incidence of AF
Lancaster et al. [26]	Randomized controlled trial	Patients after CABG (n=2041)	Mg + K	POAF	↑ risk of AF

Abbreviations: n – study population; Mg – magnesium; K – potassium; POAF – postoperative atrial fibrillation; CABG – coronary artery bypass grafting; ICU – intensive care unit

Role of Magnesium in the Treatment of Atrial Fibrillation

Hypomagnesemia is considered an important risk factor for the development of arrhythmias. One cohort study demonstrated an increased incidence of AF among individuals without pre-existing cardiovascular disease who exhibited reduced serum magnesium concentrations [15]. Given the high prevalence of this electrolyte disturbance in the general population, the hypothesis has emerged that magnesium supplementation may play a potential role in the treatment of AF.

In the management of cardiac arrhythmias, magnesium is administered primarily via the intravenous route, where it may serve as an adjunct to standard antiarrhythmic therapy, although the benefits of its administration remain a subject of debate [23]. Clinical evidence suggests that intravenous magnesium administration may reduce the occurrence of supraventricular arrhythmias and improve cardiac electrical stability by decreasing cardiomyocyte excitability [6,22]. Magnesium supplementation is also particularly important in patients with hypomagnesemia, contributing to the regulation of cardiomyocyte action potentials and the restoration of myocardial electrical balance. Furthermore, magnesium may influence ventricular rate by slowing impulse conduction through the cardiac conduction system, an effect that may be beneficial in AF. Available studies also suggest a potential synergistic effect between magnesium and antiarrhythmic agents such as digoxin and beta-

blockers, particularly in the management of acute forms of AF [27]. Nevertheless, it should be emphasized that magnesium remains an adjunctive treatment and should not be considered a substitute for standard antiarrhythmic therapy, as its effect on the conversion of tachyarrhythmias to normal sinus rhythm remains uncertain.

Due to the limited number of studies evaluating oral magnesium supplementation as a treatment strategy for AF, its effectiveness in this form remains unclear. Most available publications focus on the relationship between serum magnesium concentrations and the risk of developing new-onset arrhythmias rather than directly assessing the impact of oral magnesium supplementation on disease progression. Lower serum magnesium levels have been associated with an increased risk of arrhythmia in various study populations [27,28]. However, the limited availability of randomized controlled trials prevents a definitive assessment of the effectiveness of this intervention in patients with permanent AF. Consequently, long-term magnesium supplementation is currently not recommended for this patient population [10].

Significance of Magnesium Supplementation in Patients with Permanent Atrial Fibrillation

As discussed in the previous sections, the classification of AF distinguishes several forms of the arrhythmia that differ in duration and therapeutic approach. A particularly important form is permanent AF, which is characterized by persistent structural changes within the atrial myocardium and the cardiac conduction system, as well as by a modified treatment strategy focused on rate control and the prevention of AF-related complications [10]. In this form of AF, restoration of sinus rhythm is no longer considered a therapeutic goal, which may indicate a potential role for supportive interventions such as magnesium supplementation.

Most studies have focused on the role of magnesium supplementation in reducing the risk of arrhythmia following cardiac surgery [9,23], whereas the number of randomized clinical trials investigating magnesium administration as part of the treatment strategy for permanent AF remains insufficient. Available studies indicate an association between lower serum magnesium concentrations and an increased risk of arrhythmia development, however, they do not provide direct evidence supporting a beneficial effect of magnesium supplementation in patients with permanent AF [15,16]. A definitive assessment of the effectiveness of this therapeutic approach is further complicated by the substantial heterogeneity of study populations and the lack of standardized magnesium administration protocols.

From a physiological perspective, magnesium plays numerous important roles in the human body through its influence on normal cardiovascular function. Its effects on ion channel activity

contribute both to the stabilization of cardiomyocyte cell membranes and the restoration of action potentials, as well as to the regulation of conduction within the cardiac conduction system [4]. Furthermore, magnesium may exert synergistic effects when administered together with antiarrhythmic drugs, potentially enhancing treatment efficacy. These properties suggest that magnesium supplementation may offer potential benefits when incorporated into the treatment strategy for patients with permanent AF, however, there is currently a lack of experimental studies confirming this hypothesis.

It is also important to emphasize that, according to current clinical guidelines, long-term magnesium supplementation is not recommended in this group of patients [10]. Current recommendations highlight the importance of promptly identifying and treating reversible causes of AF. Therefore, correction of electrolyte disturbances, such as hypomagnesemia and the frequently accompanying hypokalaemia, remains an important component of clinical management. However, within the framework of standard therapy for permanent AF, magnesium is not considered an independent treatment modality. Consequently, the role of magnesium supplementation may be limited to correcting serum magnesium deficiency and serving as a potential adjunct to standard AF therapy in selected clinical situations.

Given the existing research gap regarding long-term magnesium supplementation in patients with permanent AF, there is currently insufficient evidence to support its routine use in all patients.

Conclusion

Atrial fibrillation is one of the most common sustained cardiac arrhythmias in the general population. It not only impairs quality of life but also significantly increases the risk of thromboembolic complications, heart failure, and mortality, making it a major clinical challenge. Continuous advances in medicine have enabled the search for novel therapeutic approaches, however, effective causal treatment options for permanent AF remain limited.

This literature review aimed to evaluate the role of magnesium in the pathogenesis, prevention and treatment of AF. Available clinical evidence indicates that magnesium is an essential electrolyte involved in the regulation of normal cardiovascular function. It contributes to the electrical stability of the myocardium through its direct effects on ion channels. Furthermore, its pleiotropic properties include antioxidant activity, which may help reduce the remodelling processes that play a significant role in the pathophysiology of permanent AF.

The strongest clinical evidence supporting magnesium supplementation in AF prevention comes from studies involving patients undergoing cardiac surgery. In many of the cited studies,

magnesium administration was associated with a reduced incidence of postoperative AF. Nevertheless, substantial methodological differences, including variations in magnesium assessment methods, routes of administration, dosing regimens, and the considerable heterogeneity of study populations, limit the ability to draw definitive conclusions from the available evidence. In the treatment of AF, magnesium is used primarily as an adjunctive therapy, particularly in the management of acute arrhythmic episodes and in the correction of electrolyte disturbances.

It should also be emphasized that a clear gap exists in the available literature regarding long-term magnesium supplementation in patients with permanent AF. According to current clinical guidelines, magnesium is not recommended as part of the standard treatment of permanent AF, and its role should be limited to supportive therapy in selected clinical situations. This recommendation reflects the lack of adequately powered randomized controlled trials capable of providing conclusive evidence regarding the effectiveness of this intervention.

In conclusion, magnesium is an essential electrolyte for the proper functioning of the myocardium. Although it exhibits potential antiarrhythmic properties, its clinical significance in the treatment of permanent atrial fibrillation remains uncertain. The evidence reviewed in this article highlights the need for further high-quality clinical studies to identify patient populations that may benefit from this intervention. Only such studies will enable a reliable assessment of the effectiveness of long-term magnesium supplementation in patients with permanent AF and support the development of clear recommendations regarding its role in the management of this arrhythmia.

Disclosure

AI.

The authors utilized artificial intelligence tools for language optimization and support in preparing and refining the English-language version of the manuscript and Abstract. AI was also used to format the bibliography in accordance with the journal's requirements and the Vancouver style. Following the use of these tools, the authors conducted a thorough substantive verification of the generated content, including the accuracy of citations, bibliographic information, and consistency of the presented data with the original source publications. The authors assume full responsibility for the entire content of the manuscript and confirm that artificial intelligence was not used to generate scientific conclusions or as a substitute for the authors' scientific judgment. AI did not serve as a co-author of this work.

Author Contributions

Conceptualization: Oliwia Barbara Gutowska, Weronika Nowak, Łukasz Kowalewski

Methodology: Oliwia Barbara Gutowska, Weronika Nowak, Wiktoria Magdalena Kostuch, Łukasz Kowalewski

Investigation: Oliwia Barbara Gutowska, Weronika Nowak, Wiktoria Magdalena Kostuch, Maria Aleksandra Styczyńska, Amir Naziri, Julianna Rozalia Chyl, Agata Zimon, Martyna Zielnik, Katarzyna Stróżna

Data curation: Weronika Nowak, Wiktoria Magdalena Kostuch, Maria Aleksandra Styczyńska, Agata Zimon, Martyna Zielnik, Katarzyna Stróżna

Writing – original draft preparation: Oliwia Barbara Gutowska, Weronika Nowak, Wiktoria Magdalena Kostuch, Julianna Rozalia Chyl, Agata Zimon, Maria Aleksandra Styczyńska, Martyna Zielnik, Katarzyna Stróżna

Writing – review and editing: Oliwia Barbara Gutowska, Weronika Nowak, Wiktoria Magdalena Kostuch, Łukasz Kowalewski, Maria Aleksandra Styczyńska, Amir Naziri, Julianna Rozalia Chyl, Agata Zimon, Martyna Zielnik, Katarzyna Stróżna

Supervision: Łukasz Kowalewski, Maria Aleksandra Styczyńska

Project administration: Oliwia Barbara Gutowska, Weronika Nowak

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