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Ventricular Arrhythmias in Electrolyte Imbalance

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SOUHRN

Cíl: Cílem studie bylo analyzovat patofyziologické mechanismy, elektrokardiografické projevy a terapeutické přístupy související s komorovými arytmiemi způsobenými elektrolytovou nerovnováhou, se zvláštním důrazem na poruchy draslíku, hořčíku a vápníku a jejich roli v arytmogenezi a klinickém hodnocení rizika.
Soubor a metodika: Systematický přehled literatury byl proveden v souladu s doporučeními PRISMA 2020. Vědecké publikace z let 2019–2025 byly vyhledávány v databázích PubMed/MEDLINE, Scopus, Web of Science a Google Scholar. Výzkumná otázka byla strukturována pomocí rámce PICO se zaměřením na dospělé pacienty s poruchami elektrolytů a rozvojem komorových arytmií. Z původně identifikovaných 1 847 záznamů byly po vícestupňovém screeningu odstraněny duplicity a nerelevantní studie. Padesát dva publikací splnilo kritéria pro zařazení a bylo analyzováno. Extrahovaná data zahrnovala koncentrace elektrolytů, elektrofyziologické mechanismy, elektrokardiografické ukazatele, terapeutické intervence a klinické výsledky. Vzhledem k heterogenitě studií byla místo metaanalýzy použita tematická syntéza.
Výsledky: Bylo zjištěno, že poruchy elektrolytů významně ovlivňují elektrofyziologii myokardu a zvyšují riziko komorových arytmií. Hypokalemie přispívá k arytmogenezi prostřednictvím dysfunkce draslíkových kanálů a prodloužení repolarizace, což podporuje vznik časných a opožděných následných depolarizací. Nerovnováha hořčíku a vápníku ovlivňuje L-typ vápníkových kanálů a ryanodinové receptory, čímž vytváří podmínky pro polymorfní komorovou tachykardii, včetně torsades de pointes. Nejvyšší arytmogenní riziko se vyskytuje při kombinovaném nedostatku draslíku a hořčíku (K^+ < 2,8 mmol/L a Mg^{2+} < 0,6 mmol/l). Kontinuální monitorování srdeční činnosti odhalilo klinicky významné arytmie přibližně u 30 % vysoce rizikových pacientů ve srovnání s 6 % při standardním sledování.
Závěr: Elektrolytová nerovnováha představuje významný reverzibilní faktor ve vývoji komorových arytmií. Včasná laboratorní diagnostika, elektrokardiografické monitorování a individualizované korekční protokoly, včetně suplementace draslíku a terapie hořčíkem, jsou zásadní pro snížení arytmického rizika a prevenci náhlé srdeční smrti.

ABSTRACT

Aim: The aim of the study was to analyse the pathophysiological mechanisms, electrocardiographic manifestations, and therapeutic approaches related to ventricular arrhythmias caused by electrolyte imbalance, with emphasis on disturbances of potassium, magnesium, and calcium and their role in arrhythmogenesis and clinical risk assessment.
Methods: A systematic literature review was conducted following PRISMA 2020 guidelines. Scientific publications from 2019–2025 were searched in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar. The research question was structured using the PICO framework focusing on adult patients with electrolyte disturbances and the development of ventricular arrhythmias. From 1,847 initially identified records, duplicates and irrelevant studies were excluded through multi-stage screening. Fifty-two publications met the inclusion criteria and were analysed. Data extraction included electrolyte concentrations, electrophysiological mechanisms, electrocardiographic indicators, therapeutic interventions, and clinical outcomes. Because of heterogeneity among the studies, thematic synthesis was applied instead of meta-analysis.
Results: Electrolyte disturbances were found to significantly influence myocardial electrophysiology and increase the risk of ventricular arrhythmias. Hypokalaemia contributes to arrhythmogenesis through dysfunction of potassium channels and prolongation of repolarisation, promoting early and delayed afterdepolarisations. Imbalances in magnesium and calcium affect L-type calcium channels and ryanodine receptors, creating conditions for polymorphic ventricular tachycardia, including torsades de pointes. The highest arrhythmogenic risk occurs in combined potassium and magnesium deficiency (K^+ <2.8 mmol/L and

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Mg^{2+} <0.6 mmol/L). Continuous cardiac monitoring detected clinically significant arrhythmias in approximately 30% of high-risk patients compared with 6% under standard observation.
Conclusions: Electrolyte imbalance is a major reversible factor in the development of ventricular arrhythmias. Early laboratory detection, electrocardiographic monitoring, and individualised correction protocols, including potassium supplementation and magnesium therapy, are essential for reducing arrhythmic risk and preventing sudden cardiac death.

Introduction

Electrolyte imbalance is a common cause of life-threatening ventricular arrhythmias, as changes in potassium, magnesium, and calcium levels directly affect myocardial electrical stability. Timely recognition and correction of electrolyte disturbances are critical for preventing sudden cardiac death, particularly in patients with concomitant cardiac pathology.

The increased incidence of ventricular arrhythmias in patients with electrolyte imbalance has been widely documented in clinical and observational studies. In particular, disturbances in potassium, magnesium, and calcium levels have been identified as key factors affecting myocardial electrical stability and predisposing patients to life-threatening arrhythmias. Studies conducted in patients with myocardial infarction and chronic kidney disease have demonstrated that electrolyte abnormalities significantly increase arrhythmic risk and contribute to adverse clinical outcomes.

At the same time, existing research has primarily focused on isolated electrolyte disturbances, especially hypokalaemia, while comparatively less attention has been given to the combined effects of multiple electrolyte imbalances. Clinical observations indicate that interactions between potassium, magnesium, and calcium disturbances may exert synergistic effects on arrhythmogenesis, yet these interactions remain insufficiently systematised in the literature.

Furthermore, several methodological limitations are evident across existing studies, including small sample sizes, lack of longitudinal follow-up, and limited integration of electrophysiological, clinical, and diagnostic data. These limitations restrict the generalisability of findings and hinder the development of a comprehensive understanding of arrhythmogenic mechanisms in electrolyte imbalance.

Despite detailed pathophysiological insights, the lack of quantitative correlations between the type of imbalance and arrhythmia frequency restricts clinical applicability.

The role of hypokalaemia as a cause of acquired long-QT syndrome was illustrated by Pintaningrum and Pramana[3] in a clinical case of a female patient with hypokalaemia and hyponatraemia who developed ventricular arrhythmia with a prolonged QTc interval (exceeding 638 ms) according to Heart Rhythm Society standards. Following electrolyte correction, electrocardiogram/electrocardiography (ECG) parameters improved and arrhythmogenic manifestations resolved. Yet, the single-case design precluded generalisation of population-level risks. Electrolyte profiles in patients with confirmed ventricular tachycardia or fibrillation were explored by Laslett et al.[4], comparing clinical and biochemical characteristics in VT/VF patients with heart failure. Hypokalaemia occurred significantly more often in the VT/VF group, with severe hypokalaemia (K <3.0 mmol/L) linked to gastrointestinal losses and higher diuretic doses. Hypomagnesaemia was less frequent but present in both cohorts. The study lacked follow-up data on how electrolyte correction affected arrhythmia recurrence.

Inflammatory cytokines and their role in atrial fibrillation in Coronavirus Disease 2019 (COVID-19) patients remain an emerging issue in cardiology. Tcholadze et al.[5] conducted a retrospective cohort study at Chapidze Hospital, Tbilisi, involving 100 inpatients aged 40–80 years with COVID-19. Significantly elevated interleukin-6 ($p = 0.024$), C-reactive protein ($p = 0.001$), and ferritin ($p < 0.001$) levels were found in those with atrial fibrillation, along with longer hospitalization and lower oxygen saturation. Limitations included small sample size and absence of long-term electrolyte monitoring. Exercise testing for detecting ventricular extrasystoles in athletes was used to differentiate benign from potentially dangerous arrhythmias. Chutkerashvili et al.[6] retrospectively analysed 855 athletes at Tbilisi State Medical University: 91 (10.6%) exhibited ventricular arrhythmias on resting and/or stress ECGs. The study demonstrated that exercise testing enhances screening potential by unmasking latent arrhythmogenic substrates via adrenergic stimulation. However, its retrospective nature and lack of electrolyte assessment during exertion limited conclusions.

The long-term impact of interleukin-6 on cardiovascular outcomes post-COVID-19 was examined by Tcholadze et al.[7] through a longitudinal study at Chapidze Heart Hospital, comparing in-hospital and six-month post-recovery data. Elevated IL-6 levels during acute infection correlated with increased cardiovascular risk, suggesting that cytokine-mediated systemic inflammation exacerbates cardiac complications. Limitations included short follow-up and insufficient data on electrolyte imbalance. The historical contribution of Georgian cardiologists to hypertension research was revisited by Pagava et al.[8], which introduced the "reversible" and "latent" forms. The study underscored that hypertension prevalence in Georgia remains a major public health issue, though its historical focus lacked experimental data and relevance to electrolyte balance.

Therefore, this study aims to provide a comprehensive and structured synthesis of current scientific evidence on ventricular arrhythmias associated with electrolyte imbalance, integrating pathophysiological mechanisms, clinical manifestations, and therapeutic strategies. The study seeks to systematize available data, identify key patterns of arrhythmogenesis, and highlight gaps that require further investigation.

Materials and methods

This study is a systematic literature review conducted in accordance with the PRISMA 2020 recommendations, covering the period from 2019 to 2025 to analyse current scientific evidence on ventricular arrhythmias in the context of electrolyte imbalance. While the formal systematic search was limited to publications from 2019 to 2025, several earlier studies were additionally included to provide essential theoretical and clinical background. These sources were not part of the systematic selection process but were incorporated selectively to support the interpretation of mechanisms and to ensure conceptual continuity within the analysis. The research question was formulated using the PICO framework: in publications investigating adult patients with electrolyte disturbances (hypo-/hyperkalaemia, hypomagnesaemia, hypo-/hypercalcaemia) (P – population), how do changes in electrolyte concentrations influence the occurrence and progression of ventricular arrhythmias (O – outcome) through pathophysiological mechanisms, electrocardiographic diagnostic criteria, and therapeutic strategies?

A systematic search was performed in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar during December 2024 – January 2025. The search strategy combined the following terms: ("ventricular arrhythmia*" OR "ventricular tachycardia" OR "torsades de pointes" OR "ventricular fibrillation") AND ("electrolyte imbalance" OR "hypokalaemia" OR "hyperkalaemia" OR "hypomagnesaemia" OR "hypocalcaemia" OR "potassium" OR "magnesium" OR "calcium") AND ("pathophysiology" OR "mechanism*" OR "diagnosis" OR "treatment" OR "management"). In PubMed, Medical Subject Headings (MeSH) terms were applied, including "Arrhythmias, Cardiac/etiology" and "Water-Electrolyte Imbalance/complications". In addition, manual searches of the reference lists of selected articles were undertaken to identify relevant sources that might have been missed by electronic screening, including earlier foundational studies necessary for contextual and theoretical clarification.

Inclusion criteria were: original studies, clinical case reports with detailed mechanistic descriptions, systematic reviews, and meta-analyses published in English in peer-reviewed journals that examined the relationship between disturbances in potassium, magnesium, calcium, or sodium concentrations and the development of ventricular arrhythmias; studies containing quantitative or qualitative data on electrophysiological mechanisms at the level of ion channels, electrocardiographic markers, or therapeutic approaches; studies in adult populations or with findings that could be extrapolated to adults. Exclusion criteria were: publications dealing exclusively with atrial arrhythmias; animal studies without translational relevance to clinical practice; studies with insufficient methodological description; duplicate publications; works in which electrolyte disturbances were only briefly mentioned; publications in languages other than English; and conference abstracts without full text.

The initial search identified 1,847 records across the four databases. After a multi-stage selection process, as depict-

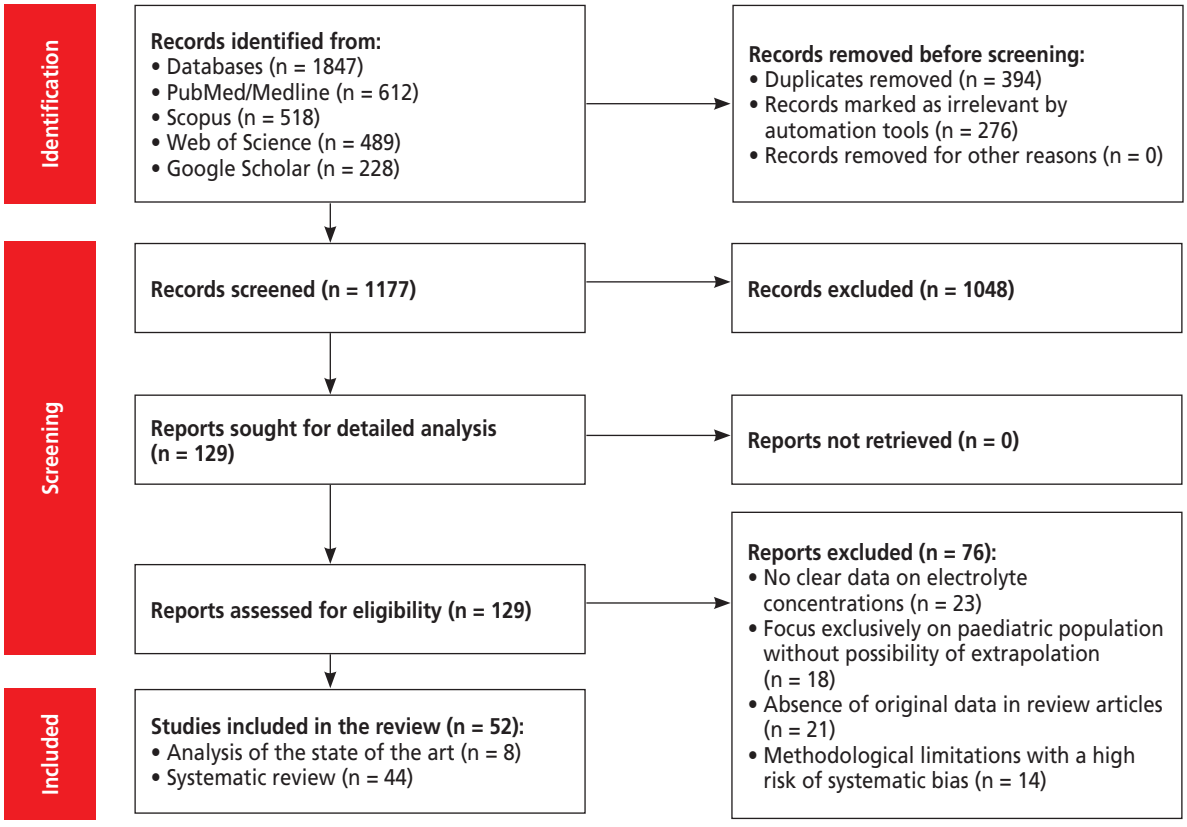

Fig. 1 – Flow diagram of source selection for the systematic review according to PRISMA 2020 methodology. Source: compiled by the author.

ed in **Figure 1**, 52 sources remained. Of the 52 studies included after screening, 44 were incorporated into the final systematic synthesis, while 8 were used selectively to provide contextual background and support the introductory overview of the topic. These 8 sources were not analysed as part of the formal systematic review but were included to enhance the conceptual framing of the study.

Study selection was performed independently by two reviewers using Mendeley Desktop 1.19.8 for reference management and documentation of the process; discrepancies were resolved by consensus or by consulting a third expert.

Data extraction was carried out independently by two reviewers using a standardised form that included: bibliographic details; study design, and population characteristics (sample size, age, sex, comorbidities); type and severity of electrolyte disturbance with specific concentrations in mmol/L; pathophysiological mechanisms (identified ion channels, changes in membrane potential, type of afterdepolarisation); clinical manifestations and diagnostic parameters (type of ventricular arrhythmia, ECG QT/QTc changes, wave morphology, monitoring methods); therapeutic interventions (route of administration, doses, infusion rates, concomitant therapy); and clinical outcomes (effectiveness of correction, recurrence rates, complications, mortality). For quantitative variables, means and standard deviations were extracted; for categorical variables, absolute frequencies and percentages. Disagreements in extracted data were resolved by re-examining the original publication until agreement was reached.

Methodological quality of included studies was assessed independently by two reviewers using validated tools appropriate to study design. The Newcastle-Ottawa Scale was used for observational studies, evaluating participant selection, group comparability, and outcome assessment (maximum 9 points). Case reports were assessed with the CAse REport guidelines (CARE), evaluating completeness of history, clinical presentation, diagnosis, and therapy. Systematic reviews were appraised using A MeaSurement Tool to Assess systematic Reviews (AMSTAR-2), which assesses 16 domains of methodological quality. Quality assessments informed the interpretation of evidence reliability, identification of potential sources of bias, and formulation of well-founded conclusions.

Due to substantial heterogeneity among the included studies, a meta-analysis was not undertaken; instead, thematic synthesis was applied. Extracted data were coded using a mixed inductive–deductive approach, assigning initial descriptive codes to discrete information fragments that were then grouped into higher-order descriptive themes. In the final stage, descriptive themes were integrated into three analytical categories: pathophysiological mechanisms by which electrolyte imbalance precipitates ventricular arrhythmias; clinical manifestations and electrocardiographic diagnosis in specific electrolyte disorders; and therapeutic strategies for correcting imbalance and pharmacological management of arrhythmias. Quantitative parameters were summarised in comparative tables of critical electrolyte concentrations, electrocardiographic indices, pharmacological dosages, and clinical outcomes, facilitating the identification of patterns and knowledge gaps.

Results and discussion

Pathophysiological mechanisms by which electrolyte imbalance precipitates ventricular arrhythmias

Between 2019 and 2025 it was established that hypokalaemia induced hyperpolarisation of the cardiomyocyte resting membrane potential through reduced IK1 current activity mediated by Kir2.1 channels. This decreased the stability of the membrane potential and increased cellular excitability. At the molecular level, such changes were attributable to mutations in the *KCNJ2* gene, which encodes Kir2.1. These mutations led to structural destabilisation of the channel, impairing its conductance and configuration in both the open and closed states.[9]

Electrophysiologically, hypokalaemia prolonged the action potential – particularly the repolarisation phase – thereby provoking early afterdepolarisations (EADs) through prolonged opening of L-type calcium channels. Excessive accumulation of calcium ions in the sarcoplasmic reticulum resulted in their spontaneous diastolic release via ryanodine receptors (RyR2), generating delayed afterdepolarisations (DADs) and promoting triggered activity.[10] In addition, hypokalaemia altered repolarisation gradients across layers of the ventricular myocardium, creating heterogeneity in action potential duration and strengthening substrates for re-entry. Bioengineered models based on human pluripotent stem cells showed that hypokalaemia reduced both longitudinal and transverse conduction velocities, shortened the effective refractory period from 242 to 165 ms, and increased calcium transient amplitude and action potential duration. These changes favoured re-entry by decreasing the wavelength of excitation and destabilising the depolarisation-repolarisation process.[11] Investigation of the functional role of the K2P1 channel demonstrated that, under hypokalaemic conditions, this channel altered ion selectivity and began to conduct inward Na^+ current, leading to spontaneous depolarisation of the resting potential, initiation of action potentials, and development of extrasystolic activity. In transgenic mice with cardiac expression of human K2P1, hypokalaemia more frequently led to ventricular extrasystoles, paroxysmal ventricular tachycardia, and ventricular fibrillation, confirming the arrhythmogenic role of this channel.[12] Thus, hypokalaemia initiated a complex set of disturbances encompassing resting potential hyperpolarisation, action potential prolongation, shortening of the refractory period, cellular calcium overload, and the creation of a heterogeneous electrophysiological substrate conducive to re-entry. Key contributors to these processes were changes in the function of the Kir2.1 and K2P1 potassium channels, alongside secondary alterations in calcium and sodium currents.

Calcium and magnesium imbalance modulated the electrophysiological properties of ventricular cardiomyocytes at the cellular level by affecting depolarisation-repolarisation dynamics and promoting polymorphic ventricular tachycardias of the torsades de pointes type through several interrelated mechanisms. Mutations in the ryanodine receptor (RyR2), the principal calcium-release channel of the sarcoplasmic reticulum, increased the likelihood of spontaneous diastolic calcium release, elicit-

ing DADs that act as triggers for arrhythmias. In a mouse model with the RyR2×R2474S mutation, Borile et al.[13] observed synchronous calcium release in adjacent cardiomyocytes, fostering electrical instability at the tissue level by overcoming intercellular electrotonic coupling. Another study showed that EADs can generate local regions with a resting potential above 0 mV and, given an appropriate tissue geometry with concave boundaries, can produce triggered activity capable of initiating recurrent re-entry tachycardia. Using computational modelling, Tsumoto et al.[14] found that chain-like emergence of EADs in clustered cardiomyocytes created directed electrical activity that facilitated torsades de pointes, particularly in the setting of a prolonged QT interval.

García-Cano et al.[15] reported in a clinical case that an *RYR2* gene mutation led to raised intracellular calcium concentration, resulting in delayed afterdepolarisations (DADs) and, consequently, triggered activity. This indicated dysfunction of sarcoplasmic reticulum calcium channels and involvement of the Na^+/Ca^{2+} exchanger in arrhythmia development. The observation suggested the possible presence of a latent catecholaminergic polymorphic ventricular tachycardia (CPVT) phenotype with a benign clinical course but a propensity to arrhythmogenesis during physical exertion. From the channelopathy perspective, Lerman et al.[16] emphasised the role of disordered calcium homeostasis – specifically, sarcoplasmic reticulum calcium overload and spontaneous Ca^{2+} release – which activated the Na^+/Ca^{2+} exchanger and produced DADs. At the same time, a reduced repolarisation reserve and increased late plateau inward current promoted the occurrence of EADs, which were directly implicated in the development of torsades de pointes within the framework of long-QT syndrome. Thus, calcium and magnesium imbalance affected the function of L-type calcium channels and RyR2 receptors, fostered spontaneous calcium release, EADs and DADs, and together created the conditions for triggered activity and the emergence of polymorphic ventricular tachycardia of the torsades de pointes type.

Hypernatraemia and hyponatraemia modulated the electrophysiological properties of ventricular cardiomyocytes by influencing the transmembrane potential, sodium-channel function and myocardial automaticity, notably by generating ectopic foci of excitation. Using two detailed mathematical models of cardiomyocytes, Verkerk and Wilders[17] showed in cellular electrophysiology simulations that hypernatraemia promoted hyperpolarisation of the resting membrane potential, increased the amplitude of calcium oscillations, raised the rate of depolarisation and augmented Na^+/K^+ pump current, while reducing the rapid and slow delayed rectifier potassium currents – ultimately prolonging the action potential. Importantly, these effects were marked only at extreme sodium levels, whereas changes were minor with moderate deviations. Timilsina et al.[18] described cases of transient atrial fibrillation arising during correction of chronic hypernatraemia in elderly patients without a prior history of arrhythmia. The authors hypothesised that sodium fluctuations during rehydration might provoke atrial stretch via preload changes, activating stretch-sensitive ion channels implicated in generating ectopic impulses. This pointed to a link between alterations in sodium balance and triggered activity, although the mechanism requires further confirmation.

Brown et al.[19] noted that even slight deviations in sodium concentration can modify neuromuscular transmission and lower the excitation threshold, while hyponatraemia is typically associated with generalised membrane depolarisation and a reduced transmembrane potential difference, promoting spontaneous activity – particularly under myocardial hypoxia or ischaemia. They emphasised that the electrophysiological consequences of disturbed sodium homeostasis occur in close interaction with other electrolyte abnormalities, especially hypokalaemia and hypocalcaemia, which together prolong repolarisation and increase the risk of late afterdepolarisations. Moreover, in a sepsis model, Castello et al.[20] showed that both hypernatraemia and moderate to severe hyponatraemia were associated with higher mortality at days 7 and 30. This indicated that sodium-balance disorders induce systemic changes, including effects on cardiac excitability and automaticity, particularly in critical illness. The authors highlighted that these disturbances may serve as markers of disease severity and predict the development of life-threatening arrhythmias. In summary, hypernatraemia and hyponatraemia alter the transmembrane potential of ventricular cardiomyocytes and sodium-channel activity, promote triggered activity and ectopic foci – especially in combined electrolyte disturbances such as hypokalaemia – and are therefore integrally linked to mechanisms of arrhythmogenesis.

Combined electrolyte disturbances exerted a synergistic arrhythmogenic effect at the levels of ion channels, gap junctions, and intercellular conductivity in the ventricular myocardium through several mechanisms that promoted fatal ventricular arrhythmias. A key role in disrupting the heart's electrical integrity was played by dysfunction of gap junctions formed by the protein Cx43. As shown by Yang et al.[21], after myocardial infarction Cx43 undergoes post-translational modifications and remodelling that impair its localisation and function, reduce intercellular electrical conductance, and facilitate ventricular tachyarrhythmias. These processes were potentiated by changes in Cx43 expression and phosphorylation that produced electrical disorganisation of the tissue. Ion channels – particularly calcium, sodium and potassium channels – also contributed to forming an arrhythmogenic substrate. Ma et al.[22] demonstrated that CaMKII activation disrupts ionic homeostasis by regulating sodium, potassium and calcium currents, thereby generating early and late afterdepolarisations. These electrical disturbances markedly increased the likelihood of initiating ventricular arrhythmias against a background of electrolyte imbalance.

Gap-junction dysfunction caused by fibrosis or inflammation likewise reduced electrical coupling and led to conduction anisotropy. Kléber and Jin[23] showed that reduced gap-junction conductance, combined with microscopic structural non-uniformity of the tissue, greatly heightens the risk of disorganised impulse propagation – particularly where electrolyte disturbances exacerbate functional disintegration between cardiomyocytes. A particularly critical pattern for fatal arrhythmias was the combination of hypokalaemia and hypocalcaemia, which prolonged action-potential duration and impaired

Table 1 – Comparative characteristics of electrophysiological changes in different types of electrolyte disturbance

Type of electrolyte imbalance	Effect on resting potential (mV)	Effect on QT/QTc duration	Specific ion channels	Primary mechanism of arrhythmogenesis	Typical ventricular arrhythmias	Critical concentrations (mmol/L)	Risk of torsades de pointes
Hypokalaemia	Hyperpolarisation (–90 to –100 mV)	QT prolongation by 50–120 ms per 1 mmol/L ↓ K^+	IKr, IKs, IK1, Kir2.1	Delayed repolarisation, EADs, triggered activity	PVCs, non-sustained ventricular tachycardia (NSVT), TdP, VF	<2.5 (moderate), <2.0 (high)	High (QTc >500 ms)
Hyperkalaemia	Depolarisation (–70 to –55 mV)	QT shortening, QRS >130 ms	INa, IK1, Kir2.1	Reduced excitability, slowed conduction, re-entry	Broad-complex VT, sine-wave pattern, VF, asystole	>6.5 (moderate), >7.5 (high), >8.5 (critical)	Low
Hypomagnesaemia	Mild hyperpolarisation (≈–85 mV)	QT prolongation by 40–90 ms	TRPM6/7, IKr, ICa-L	IKr block, Ca^{2+}-dependent EADs/DADs, increased triggered activity	TdP, polymorphic VT, PVCs, VF	<0.7 (moderate), <0.5 (high risk)	Very high
Hypocalcaemia	Mild depolarisation (≈–75 mV)	QT prolongation by 40–100 ms (ST segment)	ICa-L, RyR2	Prolonged action-potential plateau, EADs	Monomorphic VT, TdP (less common), PVCs	<1.8 (total Ca^{2+}), <0.9 (ionised)	Moderate
Hypercalcaemia	Mild hyperpolarisation	QT shortening <360 ms	ICa-L, RyR2, Ca^{2+} pumps	Shortened phase 2, ↑ automaticity, lower depolarisation threshold	PVCs, VT (with cardiac glycosides)	>2.8 (total), >1.4 (ionised)	Low
Hyponatraemia	Depolarisation (–75 to –65 mV)	Variable QT	INa (SCN5A), Na^+/K^+-ATPase	Reduced phase – 0 upstroke, conduction disturbance	Broad PVCs, NSVT, VT	<125 (severe), <115 (critical)	Low–moderate
K^+ < 2.8 + Mg^{2+} < 0.6	Marked hyperpolarisation (<–100 mV)	QT >550–600 ms	IKr, IK1, TRPM6/7	Profound repolarisation delay, multiple EADs, synergistic phase-3 prolongation	TdP, refractory polymorphic VT, VF	K^+ <2.8 + Mg^{2+} <0.6	Critically high

Source: compiled by the author based on Pintaningrum and Pramana[3], Brown et al.[25], Liu et al.[26]

repolarisation against a background of reduced sodium–potassium exchange and intracellular calcium overload. As demonstrated by Padget et al.[24], in acute viral infection – where Cx43 is modified and ion channels are disrupted – there was reduced conductance, prolonged action potentials and electrical instability even before any structural cardiomyopathy appeared. This confirmed that, even in the absence of significant myocardial remodelling, the combination of ion-channel changes and impaired intercellular communication creates a powerful arrhythmogenic substrate. Hence, the synergistic effect of combined electrolyte disturbances in the ventricular myocardium is realised through concurrent dysfunction of ion channels, Cx43-dependent gap junctions and tissue organisation, establishing the conditions for initiation and maintenance of ventricular arrhythmias, with particularly dangerous patterns involving hypokalaemia, hypocalcaemia and heightened CaMKII activity.

Studies have confirmed that different types of electrolyte disturbance exert specific effects on myocardial electrophysiology by altering resting potential parameters, the duration of repolarisation and ionic currents, thereby creating conditions for ventricular arrhythmias of varying severity. **Table 1** presents an updated comparative profile of these changes according to recent evidence.

Analysis of **Table 1** revealed a clear trend towards increased arrhythmogenic risk where repolarisation processes are impaired, particularly with combined potassium and magnesium deficiency. Determinants in such states include not only absolute concentration decreases but also their interaction with blockade of potassium and calcium currents, which promotes early afterdepolarisations. These changes generally occur without substantial structural myocardial remodelling but markedly destabilise electrophysiological integrity. Notably, even small shifts in membrane potential can reduce excitability or slow conduction – preconditions for re-entry mechanisms – seen particularly in hyperkalaemia and hyponatraemia. Despite a lower risk of torsades de pointes in these settings, their potential lethality is high due to asystole or ventricular fibrillation. Overall, the greatest clinical danger is posed by combined ionic deficits that create a polymodal arrhythmogenic substrate – characterised by delayed repolarisation, early and late afterdepolarisations, conduction disturbances, and pronounced abnormalities of automaticity.

Clinical manifestations and electrocardiographic diagnosis of ventricular arrhythmias in specific electrolyte disorders

Khan et al.[28] showed that hypokalaemia leads to sequential, quantitatively measurable ECG changes, from T-wave flattening at potassium <3.5 mmol/L and the appearance of U-waves at <3.0 mmol/L to marked QTc prolongation >500 ms when potassium falls below 2.5 mmol/L.

The authors identified a clear correlation between decreasing potassium and increasing T-peak to U-end distance, reflecting ventricular repolarisation heterogeneity and an elevated risk of polymorphic ventricular tachycardia. Tsai et al.[27] described the dynamics of ECG changes in severe hyperkalaemia (K^+ >7.5 mmol/L), including progressive P-wave disappearance, QRS widening beyond 160 ms, and the development of a sine-wave pattern preceding ventricular fibrillation.

These changes were accompanied by tall, symmetrical T-waves in the anterior precordial leads from K^+ >6.0 mmol/L. The authors stressed that the transition from a normal ECG to a sine-wave pattern occurs rapidly and demands immediate intervention, as the presence of this pattern signals a high risk of sudden asystole.

The review by Teymouri et al.[29] confirmed that hypokalaemia manifests with variability in T- and U-wave amplitude and morphology, with a tendency for T- and U-wave fusion in patients with concomitant QT prolongation. In hyperkalaemia, the principal early marker is a tall, symmetrical T-wave, whereas a falling R-wave amplitude and progressive QRS widening are markers of progression to instability. The authors proposed an "electrocardiographic spectrum" concept, within which the trajectory of changes enables risk stratification. Based on patients with renal failure, Varga et al.[30] showed that ECG changes in hyperkalaemia do not always correlate with absolute potassium levels, particularly in chronically adapted patients. Nevertheless, diffuse tall, symmetrical T-waves, reduced or absent P-waves, QRS >140 ms, and a sine-wave pattern had high specificity for identifying patients at immediate risk of life-threatening arrhythmias. They also noted that interpretation must account for clinical context, the rate of electrolyte change, and any pre-existing conduction system disease. Thus, the nature and sequence of ECG changes in hypo- and hyperkalaemia correlate both with potassium concentration and its dynamics, allowing the ECG to serve as a tool not only for diagnosis but also for risk stratification of fatal ventricular arrhythmias.

Clinical manifestations and ECG markers of ventricular arrhythmias due to hypomagnesaemia and hypocalcaemia are closely tied to repolarisation changes, increased QT dispersion, T-wave alternans, and the emergence of early or late afterdepolarisations, creating a favourable substrate for arrhythmogenic events. Yang et al.[31] showed that in isolated hypomagnesaemia, QTc, T-peak to T-end interval (Tpe) interval, and the Tpe/QT ratio increased significantly, indicating heightened repolarisation dispersion, a key factor in arrhythmia formation. QRS duration and PR interval remained unchanged, excluding intraventricular conduction delay as the cause of these changes. Kulkarni et al.[32] investigated T-wave alternans (TWA) as a reliable marker of electrical instability. They reported that spatially discordant repolarisation alternans – that is, asynchronous changes in action-potential duration in adjacent myocardial regions – creates conditions for the re-entry mechanism underlying ventricular tachycardia and fibrillation. This phenomenon is associated with increased repolarisation dispersion and is a salient element of arrhythmogenic pathophysiology in electrolyte deficiency.

In a clinical study of patients with autosomal-dominant hypocalcaemia, Hartley et al.[33] found that sustained reductions in serum calcium and magnesium prolonged QTc via slowed repolarisation. Following correction with Encaleret, QTc normalised, indicating a direct dependence of repolarisation changes on calcium and magnesium levels, particularly through effects on L-type calcium chan-

nels and calcium-dependent inactivation. In addition, Reynard et al.[34] characterised ECG parameters reflecting both repolarisation (QTc, QT dispersion, Tpeak-Tend, TWA) and conduction (fragmented QRS, QRS dispersion). They noted that it is the combination of conduction and repolarisation abnormalities – captured, for example, by the electrophysiological balance index (QT/QRSd) – that heightens the risk of ventricular arrhythmias. These parameters not only identify individuals at increased risk of sudden cardiac death but also assist differential diagnosis of QT prolongation, including distinguishing electrolyte shifts from drug-induced or congenital long-QT syndromes. Therefore, hypomagnesaemia and hypocalcaemia are associated with pronounced alterations in ventricular repolarisation – manifesting as QTc prolongation, increased Tpe and Tpe/QT, T-wave alternans, and early afterdepolarisations – that create an arrhythmogenic substrate. Differential diagnosis of QT prolongation should be based on electrolyte status, medication history, assessment of TWA, and interval analysis considering heart rate and ECG wave morphology.

Between 2019 and 2025, multiple studies enabled quantitative and qualitative assessment of the diagnostic performance of cardiomonitoring methods – including Holter monitoring, telemetry, implantable devices, and electrophysiological study – for detecting ventricular arrhythmias, particularly those driven by electrolyte disturbances, and for monitoring the efficacy of their correction. In the randomised multicentre Telemedical cardiac risk assessment by implantable cardiac monitors in post-myocardial infarction patients (SMART-MI-DZHK9) trial, implantable cardiac monitors in patients with residual autonomic dysfunction after myocardial infarction detected prognostically significant ventricular arrhythmias in 30% of cases – substantially higher than the 6% seen with standard follow-up. Recorded events included ventricular fibrillation, ventricular tachycardia, and heart block, indicating high diagnostic sensitivity of these devices to subclinical rhythm disturbances potentially linked to impaired electrolyte homeostasis.[35]

In a clinical comparison of mobile telemetry and prolonged continuous ECG monitoring, the latter demonstrated a significantly higher detection rate of arrhythmias, including ventricular tachycardia (identified in 13 patients versus 7 with telemetry), indicating the superiority of continuous human signal analysis over algorithmic systems. In particular, continuous monitoring detected far more arrhythmic episodes overall – 61 versus 19 – which could be critical when evaluating the effectiveness of treatment for electrolyte disorders.[36] In a paediatric cohort of patients with hypertrophic cardiomyopathy, 72-hour Holter monitoring with telemetry revealed significant associations between sleep disturbances and an increased frequency of premature ventricular contractions and episodes of ventricular tachycardia. Although this study was conducted in a paediatric population, its inclusion is justified by the fact that the fundamental electrophysiological mechanisms underlying arrhythmogenesis–such as autonomic imbalance, ion-channel dysfunction, and electrolyte-related triggers–are consistent across age groups. Therefore, these findings are considered conceptually relevant for understanding arrhythmia mechanisms in adults, while acknowledging the limitations of direct clinical extrapolation. However, the extrapolation of paediatric data to adult populations should be interpreted with caution and considered as supportive rather than primary evidence.[37]

In an emergency department study, 24-hour Holter monitoring identified diagnostically significant arrhythmias in more than a quarter of patients presenting with syncope. Although results lacked clear diagnostic value in some cases, even non-specific changes predicted 30-day rehospitalisation, indicating latent risk and the potential role of electrolyte disturbances as arrhythmic triggers.[38–40] Thus, rhythm-monitoring methods – especially prolonged continuous ECG, implantable monitors, and telemetric systems – demonstrated high diagnostic capability for detecting ventricular arrhythmias associated with electrolyte imbalance and enabled assessment of therapeutic efficacy to reduce arrhythmic burden. **Table 2** summarises data from primary clinical and electrocardiographic studies on electrolyte disturbances and associated ventricular arrhythmias. It integrates reported ECG features, arrhythmia types, monitoring approaches, and clinically relevant diagnostic patterns described in the reviewed literature.

The analysis shows a clear correlation between the severity of electrolyte disturbance and the risk of life-threatening ventricular arrhythmias. The greatest danger is posed by combined potassium-magnesium disorders, severe hypokalaemia and hyperkalaemia, which are associated with unstable tachycardia phenotypes, electrical storms, and a need for continuous monitoring.[41–42] ECG changes progress from minimal to critical as electrolyte deficit worsens, reflecting a gradual rise in myocardial electrophysiological instability. It is also evident that key elements of risk stratification include not only classical ECG parameters (QTc, T–U waves, QRS width) but also contemporary methods such as T-wave alternans, beat-to-beat QT analysis, and telemetry, underscoring the growing role of dynamic, highly sensitive monitoring in identifying patients prone to fatal arrhythmias. The importance of metabolic and cardiac biomarkers (e.g., lactate, pH, troponins) is likewise increasing, especially in combined or severe disorders, indicating the need for a multidisciplinary monitoring approach that extends beyond ECG alone.[43,44] Overall, **Table 2** demonstrates that arrhythmic risk is determined by the intersection of four factors: depth of electrolyte imbalance, severity of ECG findings, arrhythmia type, and monitoring capabilities. It is the comprehensive assessment of these parameters that enables optimisation of patient management.

Notably, **Table 2** is intended as a structured analytical summary of patterns reported across the reviewed studies rather than as a formal diagnostic guideline. **Table 2** reflects synthesised evidence from relevant primary sources and should be interpreted as a comparative clinical framework rather than a normative protocol.

Therapeutic strategies for correcting electrolyte imbalance and pharmacological management of ventricular arrhythmias

Current clinical protocols for correcting hypokalaemia in patients with acute ventricular arrhythmias adopt an individualised approach that considers the degree of po-

Table 2 – Diagnostic criteria for ventricular arrhythmias in electrolyte imbalanc

Electrolyte disturbance	Concentration range	Specific ECG changes	Types of ventricular arrhythmia	Diagnostic monitoring methods	Risk class	Additional biomarkers	Recommended ECG monitoring frequency
Mild hypokalaemia	3.0–3.5 mmol/L	T-wave flattening, appearance of U-wave, ST up to 1 mm	Isolated PVCs (<30/hour), couplets	12-lead ECG, baseline monitoring	Low	Aldosterone, renin, creatinine, brain natriuretic peptide (BNP)	Every 24 h until normalisation
Moderate hypokalaemia	2.5–3.0 mmol/L	U >1 mm, QTc 470–500 ms, ST ↓ >1 mm, T inversion	Frequent PVCs, NSVT, bigeminy, trigeminy	24–48 h Holter, telemetry, TWA	Intermediate	Magnesium, troponin I (TnI), creatine kinase MB fraction (CK-MB), lactate, pH	Every 6–12 h + telemetry
Severe hypokalaemia	<2.5 mmol/L	QTc >500 ms, T-U fusion, QRS >100 ms	Polymorphic VT, TdP, VF, sustained VT	Continuous telemetry, intensive care unit (ICU) monitoring, defibrillation	High/Critical	Mg^{2+}, pH, lactate, troponin, digoxin	Continuous + ECG every 2–4 h
Moderate hyperkalaemia	5.5–6.5 mmol/L	T >10 mm, QT <360 ms, PR >200 ms	PVCs, idioventricular rhythm	12-lead ECG, baseline monitoring	Intermediate	Creatinine, urea, pH, glucose, CK	Every 4–6 h + after each intervention
Severe hyperkalaemia	>6.5 mmol/L	QRS >120 ms, sine-wave pattern, absent P	Broad-complex VT, VF, asystole, atrioventricular (AV) blocks	Continuous telemetry, ICU monitoring	High / critical	pH <7.2, creatinine, Ca^{2+}, glucose, insulin	ECG every 1–2 h
Hypomagnesaemia	<0.7 mmol/L	QTc 480–550 ms, T-U alternans, QT dispersion >80 ms	TdP (up to 80%), PVCs, polymorphic VT	Holter with QT-dispersion, TWA monitoring	High	K^+ (<3.0), calcium, albumin, phosphate	Every 4–6 h + 24-h Holter
Hypocalcaemia	<1.8 mmol/L (total) or <0.9 (ionised)	QTc prolongation 460–520 ms (via ST), narrow T	Monomorphic VT, rare TdP, PVCs	ECG + QT analysis, telemetry	Intermediate	Parathyroid hormone (PTH), vitamin D, Mg^{2+}, phosphate	Every 8–12 h
K^+ <2.8 + Mg^{2+} <0.6	K^+ <3.0 + Mg^{2+}<0.7	QTc >520 ms, marked T-U alternans	TdP, electrical storm (>3 VT/VF/24 h)	Beat-to-beat QT analysis, implantable cardioverter--defibrillator (ICD) monitoring, telemetry	Critical	pH, lactate, troponin, BNP, digoxin	Hourly ECG + continuous monitoring

Source: compiled by the author based on Khan et al.[28], Teymouri et al.[29], Yang et al.[31], Hartley et al.[33], Reynard et al.[34], Bauer et al.[35], Willcox et al.[36], and Lopes et al.[38].

tassium deficit, arrhythmic risk, concomitant electrolyte abnormalities, and patient status. Intravenous potassium is preferred in severe hypokalaemia (level <2.5 mmol/L) or when electrocardiographic changes are present, whereas oral administration is appropriate for mild or moderate deficits where intestinal absorption is intact. Amirnovin et al.[45] showed that a tiered intravenous potassium regimen in post-cardiac surgery patients achieved effective potassium elevation with minimal risk of hyperkalaemia, supporting structured protocols with controlled infusion rates and regular monitoring. Magnesium correction is integral when hypomagnesaemia drives renal potassium losses. As noted by Gaynor and Cornejo,[46] attempts to raise potassium without concurrent magnesium repletion may fail because magnesium deficiency promotes kaliuresis. The authors emphasised that intravenous magnesium – in the context of ventricular arrhythmias – raises the threshold for ventricular fibrillation, which is clinically significant in patients at high risk of life-threatening arrhythmias.

Monitoring recommendations included mandatory electrolyte checks with each potassium administration, at least daily for intravenous therapy, as described by Mustafa and Befkadu.[47] Protocols also mandated ECG surveillance when potassium infusion rates exceed 10 mmol/h because higher rates are associated with iatrogenic hyperkalaemia. The study showed that only 16% of patients with moderate or severe hypokalaemia received intravenous potassium and that concurrent monitoring was infrequently used, highlighting a need to improve protocol adherence. A specific clinical context was examined by Beaulieu and Kurczewski,[48] who studied electrolyte changes in neurosurgical patients undergoing therapeutic hypothermia. Despite a daily median potassium dose of 45 mmol, hypokalaemia persisted in 30% of cases, indicating reduced efficacy of standard regimens under specific physiological conditions. Magnesium levels, by contrast, remained stable with routine correction, underscoring the need for personalised electrolyte dosing in complex patients with secondary disturbances.[49] Thus, contemporary guidance for treating hypokalaemia in acute ventricular arrhythmias rests on the degree of deficit, route of administration, obligatory assessment of magnesium status, regular electrolyte and ECG monitoring, and avoidance of rapid unmonitored infusions. Individualised protocols support effective, safe therapy while preventing iatrogenic hyperkalaemia and potentially fatal arrhythmias.[50,51]

Intravenous magnesium sulphate remains a widely accepted first-line treatment in the emergency management of torsades de pointes (TdP) and other polymorphic ventricular tachycardias associated with QT prolongation. Antiarrhythmic mechanisms include reducing transmembrane calcium influx via L-type channels, stabilising myocardial cell membrane potential, and attenuating early afterdepolarisations, which is pivotal in preventing TdP.[52–54] Clinically, magnesium also shortens cardiomyocyte action-potential duration, thereby mitigating electrical instability. Matsuura et al.[55] reported a case of drug-induced QT prolongation with TdP unresponsive to a single 6 g magnesium dose; arrhythmia termination was achieved only after reaching an ionised magnesium level of 1.31 mmol/L via continuous infusion at 1 g/h, underscoring the importance of dynamic monitoring of ionised, rather than total, magnesium. In a clinical study by Narang and Ozcan,[56] intravenous magnesium sulphate combined with isoprenaline stabilised rhythm and abolished arrhythmia in patients with defibrillation-refractory TdP arising from combined electrolyte abnormalities, including hypomagnesaemia. Although dosing specifics were not given, high-dose use was justified in severe arrhythmogenic instability.

In a randomised controlled trial evaluating magnesium for ventricular arrhythmias in dogs, Schoeller et al.[57] showed that 0.1 mmol/kg (0.2 mEq/kg) magnesium sulphate significantly reduced ventricular ectopics and heart rate, though full conversion to sinus rhythm occurred in only two cases, suggesting a dose-dependent response and the need for human trials to confirm clinical utility. The antiarrhythmic mechanism of magnesium via calcium-channel blockade and membrane stabilisation is conserved among mammals, supporting cautious extrapolation of dose-response data to humans. In loperamide-induced TdP, Iqbal et al.[58] showed that intravenous magnesium 2 g effectively controlled ventricular tachycardias resistant to other treatments even when baseline electrolytes were normal, underscoring magnesium's versatility as baseline therapy in drug-induced QT-prolongation scenarios. Accordingly, magnesium sulphate is effective for terminating TdP regardless of the aetiology of QT prolongation – drug-induced, electrolyte-mediated, or idiopathic.[59] A bolus of 1–2 g with possible transition to continuous infusion (e.g., 1 g/h) tailored to clinical response and ionised magnesium level is considered optimal. Randomised trials and clinical cases support magnesium as first-line therapy in the emergency treatment of TdP, with emphasis on electrolyte monitoring and individualised dosing.

In patients with recurrent ventricular arrhythmias amid chronic or refractory electrolyte disturbances, interventional treatments – such as temporary pacing and ICD implantation – were justified based on clinical indications of rhythm instability unresponsive to medication.[60] In Tixier et al.,[61] temporary overdrive pacing was used to treat electrical storm in patients with ventricular fibrillation resistant to amiodarone, beta-blockers, and lidocaine. Temporary pacing reduced ventricular arrhythmia burden, particularly after recent ischaemic episodes, and served as a bridge to catheter ablation or optimisation of medical therapy, providing rapid electrophysiological stabilisation without major complications in most cases. Mitkowski[62] noted that ICD implantation for secondary prevention should proceed once reversible causes of arrhythmia have been excluded. In patients with chronic kidney disease (CKD), malabsorption, or endocrinopathies – where electrolyte imbalances are prolonged or recurrent – ICD placement was considered when the risk of recurrent life-threatening ventricular arrhythmias was high, accompanied by careful evaluation of the potential to restore electrolyte balance, particularly in reversible hypokalaemia or hypomagnesaemia.[63,64]

In a retrospective study, Meter and Borovac[65] described a case of refractory electrical storm after acute myocardial infarction in a patient with concomitant CKD. Temporary

ventricular overdrive pacing terminated arrhythmia unresponsive to drug therapy and served as a bridge to ICD implantation, confirming the utility of temporary pacing as bridging therapy in patients with severe electrolyte disturbances and high arrhythmic mortality risk. Analysis of Italy's national registry by Proclemer et al.[66] showed that ICD implantation for primary prevention predominated among heart-failure patients, with a substantial proportion also implanted for secondary prevention after cardiac arrest. In patients with comorbidities complicating electrolyte balance – including endocrine disorders and malabsorption syndromes – long-term management strategies combined ICD therapy, medication, electrolyte correction, and regular rhythm monitoring.

Correction of hypokalaemia and hypomagnesaemia in patients with ventricular arrhythmias requires a personalised strategy that accounts for the severity of imbalance, comorbidities, and risk of fatal complications. The choice between intravenous and oral administration, the necessity of concurrent magnesium correction, and continuous monitoring of treatment effectiveness enable stabilisation while avoiding iatrogenic hyperkalaemia. Interventional strategies such as temporary pacing and ICD implantation are justified in refractory or recurrent arrhythmias driven by chronic electrolyte disturbances and are complemented by long-term rhythm and metabolic control.

Conclusions

A systematic review of 52 scientific publications from 2019–2025 allowed for the synthesis of current evidence and the formulation of a conceptual framework describing ventricular arrhythmias in electrolyte imbalance. The study provides a structured summary of reported electrolyte thresholds and their potential association with arrhythmogenic risk.

Hypokalaemia is considered to contribute to a cascade of electrophysiological changes via Kir2.1 and K2P1 potassium-channel dysfunction, causing resting potential hyperpolarisation, action-potential prolongation, and critical shortening of the refractory period from 242 to 165 ms. Calcium and magnesium imbalance appears to modulate L-type calcium-channel and RyR2 receptor function, creating conditions for torsades de pointes through spontaneous diastolic calcium release. Combined potassium <2.8 mmol/L and magnesium <0.6 mmol/L may produce a synergistic arrhythmogenic effect via joint dysfunction of ion channels, Connexin-43-dependent gap junctions, and intercellular conductance, creating a critically high risk of fatal arrhythmias.

Systematisation of electrocardiographic markers revealed a clear correlation between imbalance severity and progression of ECG changes, enabling ECG-based risk stratification of sudden cardiac death. Hypokalaemia shows sequential changes from T-wave flattening below 3.5 mmol/L to QTc >500 ms below 2.5 mmol/L, while hyperkalaemia presents with tall, symmetrical T-waves above 6.0 mmol/L progressing to a sine-wave pattern. Prolonged cardiomonitoring detected prognostically significant ventricular arrhythmias in 30% of patients at risk of electrolyte imbalance compared with 6% under standard follow-up. Therapeutic protocols require an individualised approach with intravenous potassium at concentrations below 2.5 mmol/L, mandatory concurrent magnesium correction, and ECG monitoring when infusion rates exceed 10 mmol/h. Magnesium sulphate in a 1–2 g bolus remains first-line in emergency management of torsades de pointes, with monitoring of the ionised fraction.

Clinical implementation entails stratified monitoring protocols, comprehensive diagnostic panels including metabolic biomarkers, and personalised correction algorithms that account for comorbidities. A limitation of this review is substantial heterogeneity among included studies, precluding meta-analysis and quantitative estimation of therapeutic effect size. Future research should focus on addressing the key gaps identified in the current body of evidence. In particular, there is a need for prospective clinical studies evaluating the impact of combined electrolyte disturbances on the development and progression of ventricular arrhythmias, as most available data focus on isolated imbalances.

In addition, the literature demonstrates considerable variability in reported electrocardiographic thresholds, especially regarding QTc prolongation and its association with specific electrolyte levels, highlighting the need for standardisation of diagnostic criteria. Further investigation is also required to assess the long-term outcomes and recurrence rates of ventricular arrhythmias following correction of electrolyte imbalance, as current data are largely limited to acute clinical observations.

Finally, comparative studies evaluating the effectiveness of different cardiac monitoring strategies, including continuous ECG monitoring, telemetry, and implantable devices, are needed to determine optimal approaches for risk stratification and clinical management.

Conflict of interest
The author declares none.

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Ethical statement and consent to participate
All procedures performed in the study were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments. Prior to the study, all patients signed a voluntary informed consent for participation in the study.

Consent for publication
All patients signed a voluntary informed consent to the processing of their personal data for the study.

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