The relationship between repolarization parameters and serum electrolyte levels in patients with J wave syndromes

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ABSTRACT. Background: Intravenous administration of magnesium (Mg²⁺) is effective for polymorphic ventricular tachycardia via homogenization of transmural ventricular repolarization. Mg²⁺ likely plays some role in the heterogeneity of repolarization in J wave syndromes. Objective: To investigate the relationship between the repolarization parameters and serum Mg²⁺, potassium (K⁺), and calcium (Ca²⁺) levels in J wave syndromes. Methods: Thirteen J-wave syndrome patients (Brugada and early repolarization [ER] syndromes), with documented episodes of ventricular fibrillation (VF), and 13 ER pattern (ERP) or Brugada type ECG patients were enrolled (25 males, mean age 48 ± 15 years). The 12-lead ECG-derived parameters including the QT, QT dispersion (QTd), Tpeak-Tend (Tp-e) interval, Tp-e dispersion (Tp-ed), Tp-e/QT ratio, and activation recovery interval (ARI) dispersion were calculated; the correlations between these parameters and electrolytes including Mg²⁺, K⁺, and Ca²⁺ were analyzed. Results: Although there was no association between serum K⁺ or Ca²⁺ and QTd, there was a strong negative correlation between serum Mg²⁺ and QTd in J wave syndrome patients with a history of VF (r = -0.715, p = 0.006). Also, there was a tendency for a negative correlation between serum Mg²⁺ and Tp-ed or ARI dispersion in J wave syndrome patients with a history of VF (r = -0.513, p = 0.072 and r = -0.53, p = 0.063, respectively). On the other hand, in 13 patients with a Brugada type ECG or ERP, no correlation was observed between serum Mg²⁺ and the QTd, Tp-ed or ARI dispersion. Conclusion: Serum Mg²⁺ may play an important role in the cardiac repolarization process in J wave syndromes.

Key words: J wave syndrome, repolarization, magnesium

Recently, great attention has been focused on early repolarization and the so-called “J wave syndrome” [1] because of the increased risk of ventricular fibrillation (VF) [2]. As for the causative genes for J wave syndromes, various genetic mutations related to sodium (Na⁺), Ca²⁺, and K⁺ channels have been reported [1]. Also, the triggering of VF is affected by factors such as the autonomic nervous system, hypokalemia, and certain drugs [1]. Indeed, hypokalemia is likely a key trigger of VF in J wave syndromes, as previously reported [3, 4]. However, the triggering mechanisms underlying J wave syndromes have not been fully elucidated. Furthermore, the relationships...
between the repolarization parameters and electrolytes other than K+, in patients with J wave syndromes have not been reported to date.

Mg2+ is the second most abundant intracellular cation and the fourth most abundant cation in the body [5]. Although only 1% of the Mg2+ in the body is in the plasma and interstitial fluid, Mg2+ is indispensable for normal physiological functions, including myocardial function; in fact, reduced Mg2+ levels could cause cardiovascular events [5]. Intravenous administration of Mg2+ has been shown to be effective for many types of arrhythmias including *torsade de pointes* (Tdp). One of the important anti-arrhythmic mechanisms of Mg2+ is thought to be a homogenizing, ventricular repolarization process [6]. Indeed, the heterogeneity of the ventricular repolarization across the ventricular wall is reported to be important for initiating and perpetuating polymorphic ventricular tachyarrhythmias. Intravenous Mg2+ has been shown to be effective in a prolonged QT interval model of polymorphic ventricular tachycardia via homogenization of transmural ventricular repolarization [7]. Because ventricular fibrillation in J wave syndromes is initiated by phase 2 re-entry leading to polymorphic ventricular tachyarrhythmias, there is a possibility that Mg2+ has some role or contributes to the heterogeneity of the repolarization in J wave syndromes. Hence, we sought to investigate the relationship between the repolarization parameters and serum Mg2+, K+, and Ca2+ levels in J wave syndromes.

### Methods

A total of 26 patients were enrolled (25 males, mean age 48 ± 15 years). Thirteen patients who met the diagnosis of J-wave syndrome (Brugada and ER syndromes) with documented episodes of VF, and thirteen early repolarization pattern (ERP) or Brugada type ECG patients were enrolled. A Brugada type ECG was defined in those patients who had a coved-type or saddle-back type ST-segment elevation in leads V1 to V3. An ERP was defined in those patients who had a J-wave elevation of >0.1 mV in at least two leads within the inferior and/or lateral chest leads.

### Serum electrolytes measurements

The serum Mg2+ and Ca2+ were measured by a colorimetric method. The serum K+ was measured by an ion selective electrode method. All measurements were performed with an auto-analyzer (LABOSPECT 008, Hitachi Inc. Tokyo, Japan).

### ECG recordings and analyses

In 13 patients who had the diagnosis of J-wave syndrome, with documented episodes of VF, a standard investigation protocol was performed in each patient including a 12-lead surface ECG, blood sampling, chest X-rays, 2-dimensional echocardiography, 24-hour Holter recordings, myocardial perfusion imaging, cardiac magnetic resonance imaging, cardiac catheterization when indicated, and an electrophysiology study when appropriate during their first admission. In the 13 ERP or Brugada type ECG patients, the standard investigation protocol was performed either in the outpatient clinic or during admission. The 12-lead ECG-derived parameters including the QT, QT dispersion (QTd), Tpeak-Tend (Tp-e) interval, Tp-e dispersion (Tp-ed), Tp-e /QT ratio, and activation recovery interval (ARI) dispersion were calculated using the QT observer Version 3.0 software (Nihon Kohden, Tokyo, Japan; figure 1).

The QT interval was defined as the interval from the onset of the QRS complex to the end of the T wave, which was the intersection of the isoelectric line and T wave. If a U wave existed, the end of the QT interval was taken to be the nadir between the T and U wave peaks. For QTd, the difference between the maximum and minimum intervals on the 12-lead ECG was calculated. ARI was defined as the time from the onset of the intrinsic deflection of the QRS (time of the minimum dV/dT) to the timing of the maximum upstroke velocity near the peak of the T wave (time of the maximum dV/dT). ARI was measured for each lead, and ARI dispersion was defined as the maximum ARI difference among the 12 leads. Tp-e, measured for each lead, was obtained by calculating the difference between the QT interval and QT peak interval, as measured from the beginning of
Figure 1. The method used for the measurement of the QT parameters is illustrated. A) Repolarization parameters. B) ARI dispersion.

QRS to the peak of the T-wave. Tp-e dispersion was defined as the difference between the maximum and minimum Tp-e intervals of the 12 leads.

The measurements were performed by two investigators blinded to the results of the other studies, and the average values were used. Then the correlations between these parameters and the electrolyte analyses, including Mg$^{2+}$, K$^+$, and Ca$^{2+}$, were determined. As for the patients whose electrolytes could be measured immediately after VF, those values were also evaluated. All participants gave their written informed consent. The local institutional ethics review board approved this study.

**Statistical analysis**

The values are presented as the mean ± standard deviation. Variable differences between the two groups were analyzed using Student’s t-test, and to test the association between two variables, the Pearson correlation method was used. All statistical analyses were performed on a personal computer with the Sigma Plot for Windows statistical package (version 11.2, Synstat Software, Inc., San Jose, CA, USA). A P-value <0.05 was considered significant.

**Results**

*Tables 1 and 2 show the serum K$^+$, Ca$^{2+}$, and Mg$^{2+}$ concentrations, and the QT maximum (max), QT minimum (min), QTd, Tp-e max, Tp-e min, Tp-ed, Tp-e/QT, and ARI dispersion in the J wave syndrome patients and Brugada type ECG or ERP patients, respectively. There was no significant difference between the two groups regarding serum K$^+$, Ca$^{2+}$, and Mg$^{2+}$ concentrations. Also, there was no significant difference between the two groups...*
Figure 1. (Continued).

regarding the QT max, QT min, QTd, Tp-e max, Tp-e min, Tp-ed, Tp-e/QT, and ARI dispersion. Although there was no association between serum K⁺ or Ca²⁺ and QTd (data not shown), there was a strong negative correlation between serum Mg and QTd in the J wave syndrome patients who had a history of VF ($r = -0.715$, $p = 0.006$, $n = 13$) (figure 2). Also, there was a tendency for a negative correlation between Mg²⁺ and Tp-ed or ARI dispersion in the J wave syndrome patients who had a history of VF ($r = -0.513$, $p = 0.072$ and $r = -0.53$, $p = 0.063$, respectively, $n = 13$: figures 3 and 4). On the other hand, in 13 patients with a Brugada type ECG or ERP, no correlation was observed between serum Mg²⁺ and QTd, Mg²⁺, and Tp-ed, and Mg²⁺ and ARI dispersion (figures 2-4). Furthermore, the serum K⁺ and Mg²⁺ exhibited relatively low values just after VF in the J wave syndrome patients who had a history of VF ($3.2 ± 0.4$ mEq/L and $1.97 ± 0.6$ mg/dL, respectively, $n = 7$).

Figure 5 shows a representative example from a 64-year-old male who was referred to our department for the treatment of Brugada syndrome. His QTd and ARId values were high, but the serum Mg²⁺ value was low.
Table 1. Serum electrolytes and QT parameters in J wave syndrome patients

<table>
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<th>K⁺</th>
<th>Ca²⁺</th>
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<th>QT minimum</th>
<th>QTd</th>
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Mean ± SD 2.1 ± 0.2 4.1 ± 0.3 9.4 ± 0.5 411 ± 39 353 ± 44 58 ± 20 106 ± 23 64 ± 13 42 ± 19 0.25 ± 0.04 138 ± 36

QTd: QT dispersion; Tp-e: Tpeak-Tend; Tp-ed: Tp-e dispersion; ARId: activation recovery interval dispersion; ms: millisecond; SD: standard deviation
### Table 2. Serum electrolytes and QT parameters in ER pattern or Brugada type ECG patients

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<th>K$^+$ (mmol/L)</th>
<th>Ca$^{2+}$ (mg/dL)</th>
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<th>QT minimum (ms)</th>
<th>QTd (ms)</th>
<th>Tp-e maximum (ms)</th>
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Mean ± SD 2.0 ± 0.1 4.1 ± 0.2 9.5 ± 0.3 400 ± 34 349 ± 26 51 ± 19 101 ± 14 59 ± 18 42 ± 12 0.25 ± 0.04 143 ± 53

QTd: QT dispersion; Tp-e: Tpeak-Tend; Tp-ed: Tp-e dispersion; ARId: activation recovery interval dispersion; ms: millisecond; SD: standard deviation
Discussion

In the present study, we demonstrated that the serum Mg\textsuperscript{2+} level may play an important role in the cardiac repolarization process in J wave syndromes. To the best of our knowledge, this is the first study to demonstrate that Mg\textsuperscript{2+} may be related to the arrhythmogenesis in J wave syndromes.

It has been suggested that the serum Mg\textsuperscript{2+} levels do not correlate with the tissue levels, and that a significant cardiac depletion exists even with normal concentrations in patients with heart disease. Indeed, Haigney \textit{et al.} demonstrated that the sublingual Mg\textsuperscript{2+} level, but not the serum Mg level, correlated inversely with the QT interval dispersion [8]. On the other hand, a recent meta-analysis by Qu \textit{et al.} suggested that a rise in the serum Mg\textsuperscript{2+} concentration was linearly associated with a reduction in the risk of total cardiovascular events [9], suggesting that a very slight change in the serum Mg\textsuperscript{2+} concentration has some role in controlling physiological function.

Although the serum Mg\textsuperscript{2+} concentrations in the present J wave syndrome patients were not very low compared to the physiological range, the significant correlation between the repolarization
parameters and Mg²⁺ concentration may indicate some pathophysiological role of Mg²⁺ in J wave syndromes. Mg²⁺ has been reported to exert its antiarrhythmic effect via modulation of cardiac ion channels.

Although there is little or no effect on the Na⁺ channels, it has been shown that the L-type Ca²⁺ current is inhibited by extracellular Mg²⁺ in a dose-dependent manner and the cardiac membrane stabilizing action of Mg²⁺ is mainly due to its modulation of the amplitude, and the activation/inactivation kinetics of the Ca²⁺ channels [5]. Furthermore, Mg²⁺ can also influence the inward and delayed-rectifier K⁺ channels, and Mg²⁺-induced rectification is influenced by extracellular K⁺ levels, which may also contribute to the membrane stabilizing effect [5].

Regarding the causative channels for J wave syndromes, in addition to decreases in the Na⁺ or L-type Ca²⁺ currents and increases in the outward K⁺ currents, the transient outward current (Ito) is thought to be an important current for the genesis of a gradient between the epicardium and endocardium [1]. The Ito current is also known to be modified by intracellular Mg²⁺ [10], and the ventricular action potential duration is reported to be shortened by a low intracellular Mg²⁺ level. Collectively, there is the possibility that those multichannel effects caused by Mg²⁺ may contribute to the membrane stabilizing action and

**Figure 3.** Correlation between the serum Mg²⁺ and Tp-ed in J wave syndrome patients who had a history of VF (A) and Brugada type ECG, or ERP patients (B).
homogenizing effects on repolarization in J wave syndromes.

Chinushi et al. reported that intravenous Mg\textsuperscript{2+} shortened the ARI slightly, and decreased the transmural dispersion of the repolarization in a prolonged QT interval canine model of polymorphic ventricular tachycardia [7]. In fact, from the clinical point of view, it has been shown that Mg\textsuperscript{2+} has little effect on the QT interval. In addition, from the cellular electrophysiological point of view, Mg\textsuperscript{2+} has little effect or only a mild effect on the shortening of the ventricular repolarization [11-13]. On the other hand, in guinea pig ventricular myocytes, dual effects of Mg\textsuperscript{2+} on the action potential duration have been reported, and its mechanism is explained by different degrees of inhibition of the Ca\textsuperscript{2+} current (I\textsubscript{Ca}) and K\textsuperscript{+} current (I\textsubscript{K}) [14]. In clinical practice, it is widely accepted that intravenous Mg\textsuperscript{2+} is effective for polymorphic ventricular tachycardia via homogenization of transmural ventricular repolarization [6]. Therefore, it is necessary to elucidate the mechanism of Mg\textsuperscript{2+} in the homogenization of repolarization.

Regarding the underlying mechanism of the homogenizing activity of ventricular repolarization, Haigney et al. presented important data and a discussion [8]. They demonstrated that the sublingual epithelial Mg\textsuperscript{2+} level correlated inversely with the QT interval dispersion in patients with
ventricular tachyarrhythmias and the change in Mg$^{2+}$ also correlated inversely with the change in the QT dispersion. They discussed the postulated mechanism underlying the action of Mg$^{2+}$ as a "regulator" of repolarization. Their speculation was based on previous reports as follows: "A reduction in cytosolic Mg$^{2+}$ results in a relative increase in the I$_{Ca}$, leading to a prolongation of the action potential duration in one beat, whereas the subsequent increase in cytosolic calcium may suppress the I$_{Ca}$ and enhance the calcium-sensitive repolarizing currents resulting in shortening of the action potential duration in the next beat. Additionally, delayed rectifier K$^+$ currents would also increase due to the low Mg$^{2+}$ situation, further hastening repolarization"[8]. In our study, although we did not demonstrate a correlation between the QT interval dispersion and a change in the Mg$^{2+}$ concentration, we found a correlation between the serum Mg$^{2+}$ levels and QTd, Tp-ed or ARI dispersion in J wave syndrome patients who had a history of VF, suggesting a similar homogenizing effect of Mg$^{2+}$ on the repolarization in J wave syndromes.

There is some controversy regarding the normal value of the QT interval. In a literature review, QT dispersion was reported to vary generally between 30 and 60 ms in normal subjects [15, 16]. The weighted mean $\pm$ SD value of the QT dispersion from studies in normal subjects is reported to be 33.4 $\pm$ 20.3 ms [15]. In our study comparing serum Mg$^{2+}$ concentration in J wave syndrome patients,
J wave syndrome and electrolytes

Mg 1.8 mg/dL
K 4.4 mmol/L
Ca 9.6 mg/dL

QT dispersion 81 ms
QT maximum 458 ms
QT minimum 377 ms
Tp-ed 44 ms
Tp-Te maximum 102 ms
Tp-Te minimum 58 ms

Figure 5. (Continued).

Table 3. Comparison of the serum Mg²⁺ concentration according to the QTd values in J wave syndrome patients

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<th>QTd &gt;50 ms (n = 8)</th>
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</tr>
<tr>
<td>Serum Mg²⁺ (mg/dL)</td>
<td>2.27</td>
<td>0.16</td>
</tr>
</tbody>
</table>

QTd: QT dispersion; ms: millisecond; SD: standard deviation
*t-test

there was a statistically significant difference in Mg²⁺ levels with a QTd cut-off value of 60 ms, as shown in table 3.

Verduyn et al. demonstrated that the suppression or prevention of Tdp by MgSO₄ is related to the diminution of the interventricular delta
action potential duration using an animal model of Tdp [17]. Since the interventricular dispersion of the repolarization may also be responsible for the pathogenesis in J wave syndromes [1], Mg²⁺ might have some beneficial effects for the prevention of polymorphic tachycardia in J wave syndromes.

It is widely accepted that the K⁺ level is critical for the development of ventricular tachyarrhythmias, including Tdp. Also, hypomagnesemia has been shown to exacerbate the proarrhythmogenic effect of hypokalemia, and Mg²⁺ has an additive effect, together with K⁺, on the prevention of various arrhythmias [1]. In our study, serum K⁺ and Mg²⁺ levels exhibited relatively low values just after VF in the J wave syndrome patients who had a history of VF. Therefore, it is also suggested that
serum Mg\(^{2+}\) and K\(^+\) levels might have some important, additive role in the repolarization process in J wave syndromes.

**Conclusion**

The serum Mg\(^{2+}\) level, which can cause homogenization of the ventricular repolarization, may play some important role in the cardiac repolarization process in J wave syndromes.

**Study limitations**

The results of this study should be interpreted in light of their limitations. First, the number of patients in the study group was small. Second, although the intracellular Mg\(^{2+}\) level is important for cardiac physiological activity including ionic channels, we could not evaluate the values in the present study. Third, because we did not use MgSO\(_4\) to treat polymorphic ventricular tachycardia in J wave syndrome patients, we could not evaluate the direct effect of Mg\(^{2+}\) on tachyarrhythmias in J wave syndromes.

**Disclosure**


**References**


