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Ventricular Repolarization Disturbances after High Dose Intravenous Methylprednisolone Therapy in Children

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Abstract:

Background: Corticosteroid therapy is widely used in pediatrics in many inflammatory and autoimmune disorders.Reports have documented various cardiovascular disorders and arrhythmias occurring during corticosteroid pulse therapy. This study aimed to assess the impact of high-dose intravenous methylprednisolone on indices of ventricular repolarization in children at Benha University Hospitals. **Methods:** This observational study included 50 children (30 males, 20 females; mean age 7.1 ± 4.5 years) who received pulse steroid therapy,dose of pulse steroid therapy 10-30mg/kg for 3-5days with max dose 1gram/d.Each underwent full clinical evaluation, laboratory investigations, and ECG monitoring 4 hours before and 12 hours after methylprednisolone infusion. **Results:** The most common indications for therapy were nephrotic syndrome (16%), ADEM (10%), GBS (8%), and autoimmune hemolytic anemia (8%). ECG parameters including heart rate (HR), corrected QT interval (cQT), Tpeak-Tend interval (Tp-e), and Tp-e/QTc ratio significantly increased post-therapy, while PR interval significantly decreased ($p < 0.05$). No significant changes were noted in QRS or JTc. Adverse effects were reported in 46% of patients: fatigue & weakness (18%), headache (22%), irritability (6%), blurred vision (4%), sleep disturbance (10%), GIT irritation (14%), hotness (6%), and palpitation (10%). All patients survived, with no arrhythmias,86% responding to steroid therapy alone, while 14% required IVIG. The average hospital stay was 14.2 ± 3.9 days. **Conclusion:** High-dose intravenous methylprednisolone significantly alters ventricular repolarization markers, indicating a potential risk of arrhythmia in pediatric patients. ECG monitoring is essential during therapy. Identifying at-risk patients early and ensuring careful follow-up can minimize adverse cardiac events.

Keywords: Ventricular repolarization; methylprednisolone; high dose; ECG; children

Introduction

Acute exacerbations of multiple sclerosis, asthma, arthritis, exacerbations of long-term asthma, and other conditions, are managed and treated with methylprednisolone, an FDA-approved medication. The immunosuppressive and anti-inflammatory properties of the systemic corticosteroid methylprednisolone make it an effective treatment for a wide range of medical conditions, including those affecting the endocrine system, the immune system, the blood, and the lungs [(1)].

High-dose (pulse) steroid therapy has seen a more widespread adoption for the treatment of a variety of inflammatory and autoimmune disorders since its initial application in 1969 and 1973 to address acute rejection following renal transplantation. Methylprednisolone is the agent most commonly used in the context of pulse steroid therapy and the usual maximum dose is 1 gram given intravenously for ten to twenty minutes [(2)].

Side effects associated with long-term oral corticosteroid therapy are well known and includes glucose metabolism disturbances, hypertension, osteoporosis, increased risk of thromboembolism, obesity and muscle wasting. There are fewer adverse effects associated with intravenous pulse steroid medication than with long-term oral therapy [(3)].

Pulse steroid therapy's cardiac adverse effects are inadequately documented. Case reports provide the bulk of the little evidence, which supports the presence of acute myocardial infarction, ventricular arrhythmias, abrupt heart failure, and unexpected death [(4)].

In clinical practice, the electrocardiogram (ECG) is frequently employed to diagnose cardiac arrhythmias, conduction disturbances, electrolyte and metabolic disorders, drug effects, myocardial ischemia, and structural alterations of the myocardium. Trans membrane action potentials (APs) of atrial and ventricular myocytes are represented by the ECG waveforms [(5)].

Depolarization and repolarization are the two fundamental processes that comprise ECG recording during a typical cardiac cycle. While the QRS complex signifies ventricular depolarization and activity, the QT interval is the time between the start of the QRS complex and the end of the T wave (ventricular repolarization) [(6)].

Before this, one way to measure QT dispersion was by looking at the distribution of ventricular recovery latencies, which is the difference between the maximum and minimum QT intervals. When looking for a general abnormality in repolarizations, QT dispersion is one crude and unreliable method to use. Paradoxical ventricular tachycardia/ventricular fibrillation (VT/VF) and sudden cardiac death are symptoms of QT dispersion [(7)].

The objective of this investigation is to monitor the impact of high-dose intravenous methylprednisolone therapy on indices of ventricular repolarization in children at Benha University Hospitals.

Patients and methods

This observational study was conducted on 50 children who were given pulse steroid therapy at Benha University Hospitals, during the period from February 2024 to December 2024.

Inclusions criteria:

- Children below 16 years with sever inflammatory disorder, and autoimmune disorder, and other various diseases, who were given pulse steroid.

Exclusion criteria:

- Patients with electrolyte abnormality,
- Thyroid dysfunction,
- children with pre-existing arrhythmias

Sample size calculation

Sample size was estimated with OpenEpi v3.01 using parameters from a prior study [(8)] with $\alpha = 0.05$ and type II error = 0.2; the

minimal required sample was 50 participants.

Ethical consideration

The local ethics commission at the Faculty of Medicine at Benha University approved the entire study design. After explaining the value of the study and the procedures that would be commenced, an informed written consent was obtained from the guardian of every participant before being included in the study. Confidentiality and personal privacy were respected in all the levels of the study. **Approval code:** MS 8-2-2024

The following data were collected for all included patients: detailed history taking including cause of pulse steroid, complete clinical examination including vital signd and systemic examination, laboratory investigation, including: complete blood count, C-reactive protein, random blood sugar, sodium, potassium and calcium Liver and kidney function tests, all investigations were done 4 hour before and 12 hour after intravenous infusion of methyl prednisolone

Automated Electrocardiography (ECG): was done 4 hours before and 12 hour after intravenous infusion of methyl prednisolone, including; **QT interval,** is the interval of time between the beginning of the QRS complex and the conclusion of the T wave. **Corrected QT interval,** is formula which correct QT interval to the QT at normal heart rate. **JT interval,** is the difference between QRS complex and QT interval. **Tpeak-Tend interval,** is the time interval between the peak of the T wave and end of the QT interval. **(Tp-e)/QTc ratio** [9]**.**

Follow up until discharge or death, reported complications, response to treatment and length of hospital stay were recorded.

Statistical Analysis

To edit, code, and tabulate the data that was obtained, we utilized the Statistical Package for the Social Sciences (IBM Corp., 2017). Watson Data Science for Windows, Version 25.0, published by IBM Corporation in Armonk, New York, USA. The data for each parameter were displayed and analyzed in accordance with the type of data acquired. To ensure that the data was normal, the Shapiro-Wilk test was used. Statistical descriptions: If the numerical data is parametric, the measures used are mean and standard deviation (± SD). If the data is non-parametric, the measures used are median and range. Frequency and proportion of non-numerical data. For the purpose of analytical statistics, the difference between the pre- and post-treatment data was evaluated using a Paired T Test.

Results

This study included 50 children, 20 females and 30 males, their mean age was 7.1±4.5 years. the mean weight was 36.2±15 kg, the mean height was 131.9±21.9 cm and the mean BMI was 18.3±3.9. the most common indications of pulse steroid in this study were nephrotic syndrome (16%), then ADEM (10%), GBS (8%), autoimmune hemolytic anemia (8%), autoimmune encephalitis (6%), ITP (6%), SLE (6%), autoimmune cerebellitis (4%), multiple sclerosis (4%), optic neuritis (4%), BPD (4%), bronchial asthma exacerbation (4%), dermatomyocities (4%), Juvenile idiopathic arthritis (4%). Figure 1.

Heart rate, systolic and diastolic blood pressure were significantly higher after pulse steroid compared to before pulse steroids. While there was no statistical difference as regarding to respiratory rate and temperature. Table 1

TLC and neutrophils were significantly higher after pulse steroid while lymphocytes decreased significantly compared to before pulse steroids. While there was no statistical difference as regarding to hemoglobin and platelets. Table 2

Random blood sugar were significantly higher after pulse steroid, while CRP, urea, creatinine decreased significantly

compared to before pulse steroids. While there was no statistical difference as regarding to ALT, AST, sodium, potassium or calcium. Table 3.

Regarding ECG; HR, cQT, Tp-e, Tp-e/QTc were significantly higher after pulse steroid compared to before pulse steroids, PR were significantly lower after pulse steroid compared to before pulse steroids. While there was no statistical difference as regarding to QRS or JTc. Table 4.

In the current study, 46% of children had side effects during oulse steroids administration; 18% had fatigue & weakness, 22% had headache, 6% had irritability, 4% had blurred vision, 10% had sleep disturbance, 14%% had GIT irritation, 6% had hotness, 10% had palpitation. 86% of children responded to pulse steroids while 14% required IVIG, All case survived, the mean length of hospital stay was 14.2±3.9 days, Table 5

Table 1: Vital signs of the studied group before and after pulse steroid therapy

		Study group		Test	P value
		Before	After		
Heart rate/min.	**Mean ±SD**	103.0±22.9	112.8±11.7	Pt=6.7	<0.001*
	Range	80-165	82-130		
Temperature	**Mean ±SD**	37.2±0.4	37.0±0.3	Pt=1.1	0.51
	Range	36.5-37.5	36.5-37.5		
Respiratory rate.min	**Mean ±SD**	45.8±7.1	41.3±5.1	Pt=1.9	0.061
	Range	24-56	20-41		
Systolic Bl.p	**Mean ±SD**	87.8±16.1	94.3±9.3	Pt=4.9	<0.001*
	Range	55-131	61-135		
Diastolic Bl.p	**Mean ±SD**	55.2±12.1	59.2±8.1	Pt=3.7	<0.001*
	Range	45-92	50-99		

Pt: Paired sample t-test, *: significant

Table 2: Complete blood count of the studied group before and after pulse steroid therapy

		Study group		Test	P value
		Before	After		
Hemoglobin (mg/dl)	**Mean ±SD**	9.3±2.1	9.5±1.7	Pt=0.88	0.42
	Range	3.9-13	8.2-12.5		
Platelets ($x10^3$/L)	**Mean ±SD**	197.8±110.3	215.2±81.3	Pt=0.91	0.36
	Range	12-541	79-412		
TLC ($x10^3$/L)	**Mean ±SD**	11.4±6.3	15.3±5.1	Pt=2.1	0.011*
	Range	4.3-26.6	5.5-22.9		
Lymphocytes	**Mean ±SD**	4.4±1.5	2.3±1.3	Pt=3.7	<0.001*
	Range	0.5-6.7	1.1-4.9		
Neutrophils	**Mean ±SD**	8.3±6.3	11.3±5.1	Pt=3.1	0.002*
	Range	2-25.4	4.9-16.7		

Pt: Paired sample t-test, *: significant

Table 3: Laboratory investigations in the studied group before and after pulse steroid therapy

		Study group		Test	P value
		Before	**After**		
C-reactive protein (mg/dl)	**Mean ±SD**	34.4±21.0	13.4±7.7	Pt=3.6	<0.001*
	Range	3-196	4-56		
ALT (IU/L)	**Mean ±SD**	64.8±22.2	47.0±7.3	Pt=1.2	0.21
	Range	22-243	25-112		
AST (IU/L)	**Mean ±SD**	87.5±26.8	41.3±5.1	Pt=1.4	0.16
	Range	25-272	20-141		
Urea (mg/dl)	**Mean ±SD**	72.6±41	44.3±9.3	Pt=2.6	0.009*
	Range	19-212	18-110		
Creatinine (mg/dl)	**Mean ±SD**	1.0±0.6	0.6±0.3	Pt=2.7	0.003*
	Range	0.3-3.3	0.3-1.2		
Sodium (mEq/L)	**Mean ±SD**	139.6±4.9	138.8±5.7	Pt=0.7	0.72
	Range	124-143	134-145		
Potassium (mEq/L)	**Mean ±SD**	4.1±0.7	3.7±0.3	Pt=1.1	0.51
	Range	3.4-5	3.5-4.8		
Calcium (mmol/L)	**Mean ±SD**	9.2±0.7	8.9±1.1	Pt=1.6	0.085
	Range	7.6-10.3	7.4-10.3		
Random blood sugar (mg/dl)	**Mean ±SD**	104.2±11.3	124.3±19.3	Pt=6.9	<0.001*
	Range	85-162	98-235		

Pt: Paired sample t-test, *: significant

Table 4: ECG of the studied group before and after pulse steroid therapy

		Study group		Test	P value
		Before	**After**		
HR/min.	**Mean ±SD**	97.6±**7.9**	112.8±11.7	Pt=8.8	<0.001*
	Range	80-110	90-138		
PR(msn)	**Mean ±SD**	132.8±13.4	125.7±11.8	Pt=3.5	0.001*
	Range	110-165	110-155		
QRS duration(msn)	**Mean ±SD**	93.2±11.6	93.1±12.5	Pt=0.07	0.94
	Range	70-130	70-130		
cQT (msn)	**Mean ±SD**	393.7±12.3	406.2±14.1	Pt=6.9	<0.001*
	Range	371-415	382-432		
Tp-e (msn)	**Mean ±SD**	74.6±10.3	80.8±8.5	Pt=4.4	<0.001*
	Range	55-90	60-95		
JTc(msn)	**Mean ±SD**	300.5±18.5	305.6±21.3	Pt=1.9	0.067
	Range	276-353	276-378		
(Tp-e)/QTc (msn)	**Mean ±SD**	0.19±0.03	0.20±0.02	Pt=2.7	0.008*
	Range	0.14-0.23	0.15-0.24		

Pt: Paired sample t-test, *: significant, HR: heart rate, cQTd: Corrected QT interval, JTc: Corrected JT interval, Tp-e: peak-to-end interval of the T wave.

Table 5: Adverse effects of pulse steroid therapy in the studied group

			N=50	%
Adverse effects	**Cardiovascular**	**Fatigue & weakness**	9	18.0%
	Non-cardiovascular	**Palpitation**	5	10.0%
		Irritability	3	6.0%
		Blurred vision	2	4.0%
		Sleep disturbance	5	10.0%
		GIT irritation	7	14.0%
		Hotness	3	6.0%
		Headache	11	22.0%
	No adverse effects		27	54.0%
Respond to treatment		**Improved**	43	86.0%
		Required IVIG	7	14.0%
Survival		**Yes**	50	100.0%
		No	0	0.0%
Length of hospital stay (days)		**Mean ±SD**	14.2±3.9	
		Range	8-26	

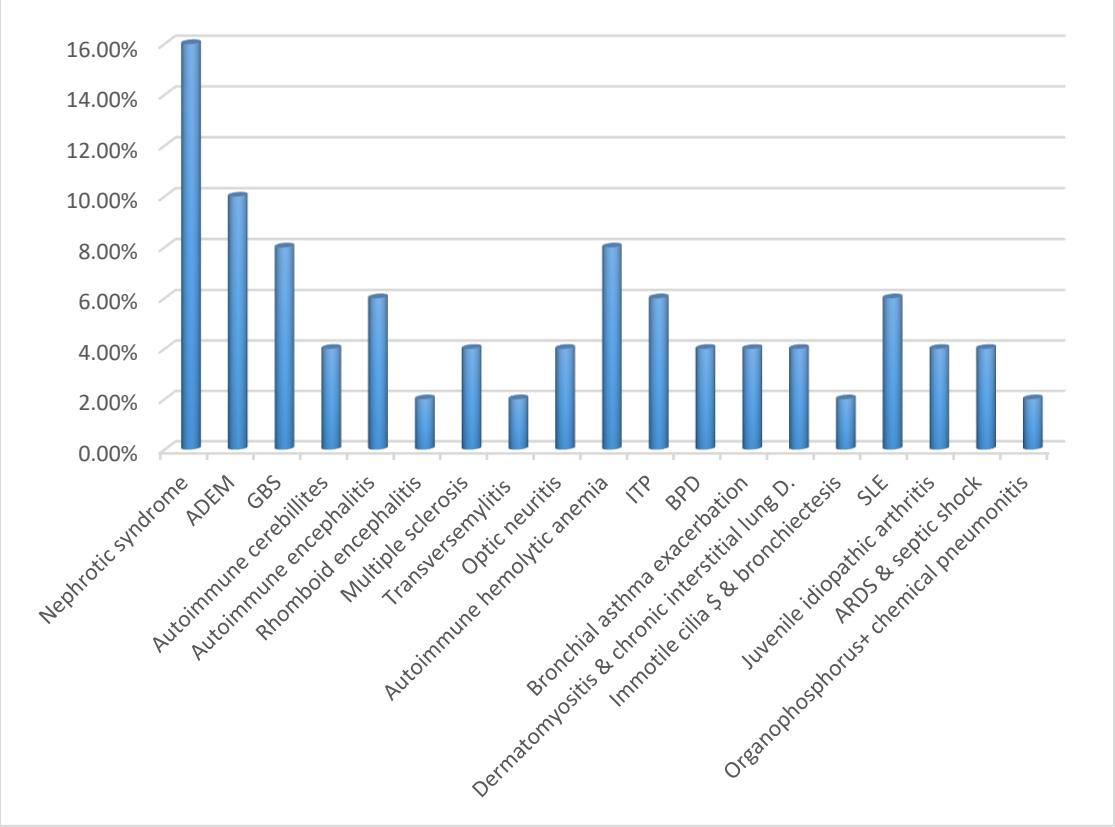

Figure 1: Cause of admission in the studied group

Discussion

The literature has documented the occurrence of cardiovascular disorders and a variety of arrhythmias during corticosteroid pulse therapy. It is hypothesized that cardiovascular physiology can be influenced by the excessive dose of steroid infusion. Although it is unknown how much mechanical cardiac alteration this mode of administration causes, corticosteroid pulse treatment has been linked to sudden deaths [(4)].

Since it is still unknown what specifically causes deaths after steroid medication, we aimed to ascertain the effect of intravenous pulse steroid delivery on ventricular repolarization indices. We found that ventricular repolarization markers, which are suggestive of malignant arrhythmias, were extended in response to pulse steroid treatment. We discovered that HR, cQT, Tp-e, and Tp-e/QTc were significantly higher after pulse steroids than before. Conversely, PR were significantly reduced after pulse steroids than before. QRS and JTc did not exhibit any statistically significant differences.

This was agreed with *Pishgahi et al.,* [(8)], They found that when steroid pulse treatment was administered, the heart rate-corrected QT (cQT) dispersion value increased significantly. Prior to starting pulse therapy, the adjusted QT dispersion was 19.92 ñ 8.2 ms. But after the treatment, it increased to 40.68 ± 18.12 ms (P-value < 0.001). Within the range of -1.00 to 56.00 ms, the average decrease in cQT dispersion (cQT interval dispersion after pulse therapy − cQT dispersion before therapy) was 20.76 ± 14.44 ms. Age was found to be correlated with changes in cQT dispersion (P-value < 0.001; B: 1.33). In univariate analysis, gender did not show any correlation with QT alteration (B: -2.65; P-value: 0.52). They noted that sinus tachycardia, the most prevalent arrhythmia both prior to and following steroid pulse therapy, was significantly more prevalent after steroid pulse therapy. Before receiving steroid pulse therapy, sinus tachycardia was observed in 8 (16%) patients, while it was observed in 25 (50%) patients afterward (P value<0.004).

Also, *Altunbas et al.,* [(2)], Pulse steroid therapy contributed to a significantly longer QT interval (361.0±29.91 vs 388.20±42.84 p:0,001) and corrected QT interval (QTc) (401.60±19.79 vs 413.72±26.38 p:0.01). Furthermore, the JT interval index (JTI%) and JT interval were significantly lengthened (118.18±17.54 vs. 110.56±13.92 p:0.01), with a difference of 273.0±28.73 vs. 299.60±45.66 p:0.001. Following high-dose steroid treatment, the Tp-e interval was significantly prolonged (74.60±13.12 vs. 83.80±13.68, p:0.001). The ratio of Tp-Te to QTc was also significantly increased as a consequence of pulse steroid therapy (0.18±0.03 vs 0.20±0.03, p=0.009).

The mechanisms responsible for the cardiac effects of steroid pulse therapy have not yet been fully understood. Hypopolarization of cell membranes, which makes them vulnerable to arrhythmias and conduction interruptions, may result from a temporary change in electrolytes across the cell membranes that does not affect their serum levels. An intensified response of the blood vessels to the body's own vasopressors might lead to cardiac fatigue. Additionally, it seems that cardiac cells' activation threshold is changed by intravenous methylprednisolone. The administration of pulse dosages affects both serum potassium and potassium excretion as well as sodium excretion. The electrolyte transfers across the membranes of myocardial cells may be affected by these modifications [(10)].

There is a dearth of literature that delineates the impact of high-dose steroid treatment on cardiovascular physiological functions.

Sakamoto et al, [(11)], an account of three instances of ventricular fibrillation that

transpired during corticosteroid therapy. Even though they were taking oral steroids for their conditions, they still thought that attacks were mostly brought on by hypokalemia from corticosteroids. It is well-known that using steroids can increase the incidence of new-onset atrial fibrillation. In addition to the number of case reports, the risk of atrial fibrillation is significantly elevated following the administration of high concentrations of corticosteroids in large series.

Depolarization and repolarization of the ventricles are indicated by the QT and corrected QT (QTc) intervals. The risk of precipitous mortality is well-established for QT and QTc durations that are either congenital or acquired [12]. The JT interval is suggested as a marker of arrhythmic risk, particularly when the QRS is protracted. *Zulqarnain et al,* [13] In patients whose QT intervals were extended (≥120 msec), a longer JT interval was more suggestive of an increased risk of mortality than a longer QT interval. Pulsed steroid treatment raises the risk of deadly arrhythmias, particularly in the short term, due to the combined effect of much longer QTc and JT intervals.

On the other side; *Tripathy et al.,* [14]**,** An 11-year-old girl with polyarticular juvenile idiopathic arthritis developed bradycardia as a consequence of pulse methylprednisolone therapy, as reported. She experienced bradycardia on the second day of her methylprednisolone infusion, with a heart rate that fluctuated between 50 and 60 pulses per minute.. An electrocardiogram revealed sinus bradycardia. Dyselectrolytemia (Na—141 mmol/l, K—3.54 mmol/l, Ca—8.72 mg/l) and sepsis were not observed.

At present, the precise pathophysiology of corticosteroid-induced bradycardia remains unknown. A change in the baroreceptor reflex due to volume expansion and steroid-induced hypertension is one of the hypothesized explanations. In addition, an idiosyncratic reaction, hypokalemia (caused by mineralocorticoid activity), and reduced catecholamine sensitivity in the heart are noted. Bradycardia develops when there is preexisting cardiac and renal illness and when the infusion rate is very high. Only a few instances of dyselectrolytemia were documented [15]**.**

Yet now, there still a lot of conflicting results, which warranted a more extensive studies to be assured.

In the present study, 46% of children had side effects during pulse steroids administration; 18% had fatigue & weakness, 22% had headache, 6% had irritability, 4% had blurred vision, 10% had sleep disturbance, 14%% had GIT irritation, 6% had hotness, 10% had palpitation.

Our results run in accordance with *Nasser et al.,* [16]**,** who studied efficacy and tolerability of pulse steroid as an add-on therapy in management of refractory epilepsy in children, they showed that they were infrequent, mild and transient side effects as weight gain (26.67%), GIT upset (20%), irritability (20%) and transient hyperglycemia (6.67%) were reported.

And *Bast et al.,* [17]**,** According to reports, 38 (70%) of the minors had negative effects, most of which were modest and temporary. Children in 18 cases had vague symptoms including mood swings, exhaustion, or irritation. Two children had recurrent abnormal blood glucose levels (maximum 172 mg/dl), two children had mildly increased blood pressure, and six children experienced a brief cushingoid appearance.

While *Koncak et al.,* [18]**,** In the patients, the most frequently observed adverse effects were psychiatric (31.4%), dermatological (48.5%), and gastrointestinal (31.4%). Seven patients (20%) were diagnosed with hypertension after treatment. Dermatological abnormalities were found in 17 patients (48.5%), 16 patients (45.7%) had edema, 12 patients (34%), 8 patients (22.8%) had hirsutism, 4 patients (11.4%) had acne, 4 patients (11.4%) had striae, and 1 patient

(2.8%) had acanthosis. A number of patients had different dermatological results. There were two patients (5.7%) with anxiety disorders and nine patients (25.7%) with mood disorders. A total of eleven patients (31.4%) had mental symptoms. There were 11 patients (31.4%) who experienced gastrointestinal symptoms. No gastrointestinal hemorrhage was detected. Upon completion of treatment, seven patients (20%) were diagnosed with hypertension, and three patients (8.5%) remained hypertensive. The antihypertensive treatment was subsequently administered to these patients and was sustained for an average of six months. One patient was diagnosed with sinus tachycardia during steroid infusion; however, no specific treatment was required.

On the other side; *Pera et al.,* [19] In the 24 hours following the infusion, only a modest transient hyperglycemia was observed; no significant or persistent adverse effects were observed. Also, *Yassin et al.,* [20]**,** reported that pulse corticosteroids was not associated with increased risk for severe adverse effects compared to oral corticosteroids.

In the present study, 86% of children responded to pulse steroids while 14% required IVIG, all case survived, the mean length of hospital stay was 14.2±3.9 days.

In the study by *Koncak et al.,* [18]**,** According to their findings, 24 patients responded to treatment, 11 patients did not respond, and one patient passed away. However, *Edel et al.,* [21]**,** concluded that pulse corticosteroids was not associated with an increase risk of severe side effects and should be regarded as safe

Finally, we acknowledge that this study had several limitations; the first of all is the small sample size which didn't allow for a more analytic studies and to detect these differences in every specific disease. Also due to short time of follow up, we couldn't detect the prolonged side effects of pulse steroids in those patients.

Additionally, there is a scarcity of research on the adverse effects of pulse steroid therapy, particularly in the context of minors. In order to assess the efficacy and complications associated with its use, multicentre and randomized controlled studies are required, which should include a variety of patient populations.

Conclusion

Our research demonstrates that pulse steroid treatment significantly prolongs indices of ventricular repolarization. These results suggest that the administration of high-dose intravenous methylprednisolone to minors is associated with an elevated risk of arrhythmias.

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Author contribution

Authors contributed equally in the study.

Conflicts of interest

No conflicts of interest

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