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Dynamic Electrocardiogram Assessment and Clinical Value of Myocardial Damage in Children with Mycoplasma Pneumoniae Pneumonia

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Abstract: To evaluate the clinical application value of 24-hour dynamic electrocardiography (DCG) and heart rate variability (HRV) indices in the early assessment of myocardial damage in children with Mycoplasma pneumoniae pneumonia (MPP), this retrospective analysis of 120 pediatric patients with Mycoplasma pneumoniae pneumonia (MPP) evaluated the diagnostic utility of 24-hour dynamic electrocardiography (DCG) and heart rate variability (HRV) indices for the early assessment of myocardial damage. Subjects were stratified into myocardial damage (n=48) and non-damage (n=72) cohorts based on clinical and biomarker criteria. DCG demonstrated a significantly higher abnormality detection rate of 56.67% compared to 19.17% for routine ECG (x^2=35.42, P<0.001). while all HRV time-domain parameters, including SDNN, SDANN, RMSSD, and pNN50, were significantly attenuated in the myocardial damage group (all P<0.001). Receiver operating characteristic (ROC) curve analysis for SDNN yielded an area under the curve (AUC) of 0.837; at an optimal cut-off value of 95.95ms, the index provided 90.3% specificity and 64.6% sensitivity for predicting myocardial involvement. These findings indicate that DCG is superior to routine ECG in detecting occult cardiac abnormalities and that diminished HRV indices reflect significant autonomic dysfunction, identifying SDNN as a reliable, non-invasive tool for early risk stratification of parenchymal myocardial damage in pediatric MPP.

1. Introduction

Accumulating immunopathological evidence indicates that *Mycoplasma pneumoniae* pneumonia (MPP) extends beyond a localized respiratory infection, manifesting as a broader systemic disease. The mechanisms underlying MPP-associated myocardial damage are multifactorial. Current understandings emphasize direct pathogen invasion, immune complex-driven hypersensitivity, and endothelial injury resulting from robust inflammatory cytokine cascades [1]. Epidemiological studies estimate that 15% to 25% of MPP patients experience concurrent myocardial injury. If left

unmanaged, a critical proportion of these pediatric cases face the risk of progressing to fulminant myocarditis or sustaining long-term arrhythmias.

While cardiac biomarkers such as cTnI and BNP remain the cornerstone for diagnosing myocardial damage, their fluctuating serum concentrations limit their utility in continuously monitoring electrophysiological stability. Furthermore, the brief recording window of standard 12-lead electrocardiography (ECG) inherently restricts its ability to detect transient or evolving cardiac abnormalities. Conversely, 24-hour dynamic electrocardiography (DCG, or Holter monitoring) substantially enhances the diagnostic yield for paroxysmal arrhythmias. Additionally, it provides a unique advantage in quantifying autonomic nervous system equilibrium through time- and frequency-domain parameters of heart rate variability (HRV) [2].

To address these diagnostic limitations, this retrospective cohort study analyzed 120 pediatric MPP cases to evaluate the efficacy of DCG in detecting occult cardiac electrophysiological changes. By examining the relationship between HRV alterations and the extent of myocardial involvement, we aim to validate a non-invasive, continuous monitoring approach to optimize risk stratification in pediatric patients with severe MPP.

2. Materials and Methods

2.1 Study Population

A total of 120 pediatric patients diagnosed with MPP and admitted to our hospital between January 2024 and December 2025 were enrolled. Based on the presence or absence of myocardial damage, the patients were assigned to either the myocardial damage group (n=48) or the non-myocardial damage group (n=72).

2.2 Methods

2.2.1 Instruments and Procedures

All patients underwent continuous 24-hour monitoring using a 12-lead fully digital dynamic electrocardiogram recorder (DM Software Inc., Stateline, NV, USA).

2.2.2 Operational Specifications

Electrodes were placed by dedicated technicians according to the standard 12-lead configuration. Prior to monitoring, the anterior chest skin was prepped, and specialized disposable electrodes were applied to reduce skin impedance and minimize motion artifacts.

2.2.3 Monitoring Management

Guardians were instructed to maintain a 24-hour daily log for the patients, recording periods of sleep, physical activity, emotional fluctuations, and the exact onset times of symptoms such as dyspnea, palpitations, and chest tightness. This facilitated the retrospective correlation between clinical symptoms and electrocardiographic alterations.

2.2.4 Valid Recording Criteria

The total monitoring duration was required to be≥ 22h, with valid data accounting for 90% of the total time. Records failing to meet these criteria were deemed invalid, and the monitoring was repeated.

2.2.5 Observational Indices and Diagnostic Criteria

Electrocardiographic evaluations were manually verified and audited by two senior attending physicians from the ECG department using a double-blind approach. Diagnostic criteria were based on *Practical Pediatric Arrhythmology* and *Pediatrics*:

2.2.5.1 Arrhythmia Criteria

(1) Premature beats: The total occurrences of atrial premature beats (APB) and ventricular premature beats (VPB) within 24 hours were recorded, and the severity was assessed according to the Lown grading system. (2) Conduction blocks: Including sinoatrial block and all degrees of atrioventricular block (AVB).

2.2.5.2 ST-T Changes

For the ST segment, measured 60–80 ms after the J point, a horizontal or down-sloping depression of≥0.05mv, or an elevation exceeding the normal range for the corresponding lead, was considered abnormal. For the T wave, flattening (amplitude <1/10of the R wave in the same lead), biphasic morphology, or inversion lasting for≥1min were recorded.

2.2.5.3 Clinical Criteria for Myocardial Damage (Grouping Validation)

Myocardial damage was defined based on the elevated serum levels of cardiac troponin I (cTnI) and creatine kinase-myocardial band (CK-MB).

2.2.6 Heart Rate Variability (HRV) Analysis

Time-domain analysis was performed using the built-in HRV analysis software. Prior to analysis, artifacts and all supraventricular and ventricular ectopic beats were manually eliminated by the physicians. Calculations were exclusively based on normal sinus R-R intervals:

① SDNN (ms): Standard deviation of all normal sinus R-R intervals over 24 hours, reflecting the overall regulatory capacity of the autonomic nervous system.

② SDANN (ms): Standard deviation of the averages of normal R-R intervals in all 5-minute segments, reflecting variations in sympathetic tone.

③ RMSSD (ms): Root mean square of successive differences between adjacent normal R-R intervals, primarily reflecting parasympathetic (vagal) activity.

④ PNN50 (%): Percentage of adjacent R-R intervals with a difference>50 ms, reflecting the sensitivity of vagal modulation.

2.3 Data Collection and Statistical Analysis

Patient data, including inflammatory markers at admission (CRP, WBC, ESR, IL-6), *M. pneumoniae* antibody titers (MP-IgM/IgG), and imaging findings, were retrieved via the hospital's Electronic Medical Record (EMR) system. The number of abnormal events detected by DCG, maximum ST-segment deviation, and all HRV parameters were recorded in Microsoft Excel 2021. All statistical analyses were performed using R software (version 4.4.2).

3. Results

3.1 Baseline Characteristics of the Study Population

A total of 120 patients were included in this study, comprising 48 cases with myocardial damage

and 72 cases without myocardial damage. There were no statistically significant differences between the two groups in demographic characteristics, including age, gender, and body mass index (all $P > 0.05$).

Regarding clinical manifestations and disease course, the duration of fever in the myocardial damage group was significantly longer than that in the non-damage group (P=0.014), and the pre-admission disease duration was also significantly prolonged (8.2±3.1 days vs. 6.4±2.5 days, P = 0.001). Concurrently, the incidence of accompanying chest tightness or palpitations was significantly higher in the myocardial damage group (37.50% vs. 6.94%, P<0.001).

Table 1 Comparison of Clinical Characteristics between Patients with and Without Myocardial Injury

Variables	**Myocardial Injury Group (*n*=48)**	**Non-Myocardial Injury Group (*n*=72)**	**Statistic**	***P* value**
Demographic characteristics				
Age (years, mean±SD)	6.8 ± 2.4	6.4±2.1	0.965	0.336
Male sex, *n* (%)	26 (54.17)	40 (55.56)	0.023	0.879
Body mass index (kg/m², mean±SD)	16.2±2.1	15.9±1.8	0.832	0.407
Clinical manifestations and disease course				
Duration of fever (days, median [IQR])	7.0 (5.0, 9.5)	5.5 (4.0, 7.5)	-2.451	0.014
Disease course before admission (days, mean±SD)	8.2±3.1	6.4±2.5	3.485	0.001
Chest tightness/palpitations, n (%)	18 (37.50)	5 (6.94)	18.24	<0.001
Comorbidities, *n* (%)				
Pleural effusion	12 (25.00)	8 (11.11)	4.021	0.045
Atelectasis	7 (14.58)	6 (8.33)	1.185	0.276
Laboratory findings				
White blood cell count (×10⁹/L, mean±SD)	9.8±3.2	8.4±2.8	2.521	0.013
C-reactive protein (mg/L, median [IQR])	42.5 (28.2, 65.4)	18.6 (10.4, 32.8)	-5.124	<0.001
Lactate dehydrogenase (U/L, mean±SD)	485±112	312±85	9.542	<0.001
Myocardial injury biomarkers				
Creatine kinase-MB (U/L, mean±SD)	38.4±12.5	16.2±5.4	13.56	<0.001
Cardiac troponin I (μg/L, median [IQR])	0.12 (0.08, 0.25)	0.02 (0.01, 0.04)	-7.845	<0.001

Notes:

① Continuous variables with normal distribution are presented as mean ± standard deviation (SD) and compared using the independent samples t-test.

② Non-normally distributed variables are expressed as median (interquartile range, IQR) and

analyzed using the Mann–Whitney U test.

③ Categorical variables are presented as number (percentage) and compared using the Chi-square test.

In terms of pulmonary complications, the incidence of pleural effusion was higher in the myocardial damage group than in the control group (25.00% vs. 11.11%, P=0.045), whereas the difference in the incidence of atelectasis was not statistically significant (P=0.276).

Regarding laboratory parameters, the white blood cell (WBC) count was significantly elevated in the myocardial damage group (9.8±3.2×10⁹/L vs. 8.4±2.8×10⁹/L, P=0.013). The level of the inflammatory marker C-reactive protein (CRP) was also markedly higher than that in the control group [42.5 (28.2, 65.4) mg/L vs. 18.6 (10.4, 32.8) mg/L, P<0.001]. Furthermore, the lactate dehydrogenase (LDH) level was significantly increased in the myocardial damage group (485±112 U/L vs. 312±85 U/L, P<0.001).

Concerning myocardial injury biomarkers, the CK-MB level in the myocardial damage group was significantly higher compared to the non-damage group (38.4±12.5 U/L vs. 16.2±5.4 U/L, P<0.001), and the cardiac troponin I (cTnI) level was also significantly elevated [0.12 (0.08, 0.25) μg/L vs. 0.02 (0.01, 0.04) μg/L, P<0.001]. Details are summarized in Table 1.

3.2 Comparison of Detection Efficiency between Routine ECG and Dynamic Electrocardiogram (DCG)

The detection rates of various electrocardiographic abnormalities were compared between routine ECG and 24-hour DCG in 120 pediatric patients with MPP. As summarized in Table 2, DCG demonstrated significantly higher sensitivity in identifying cardiac electrophysiological changes compared to routine ECG (all P < 0.05). Specifically, the incidence of atrial premature beats (APB) detected by DCG was 18.33% (22/120), markedly surpassing the 6.67% (8/120) identified by routine ECG (x^2=7.124, P=0.008). Similarly, DCG yielded higher detection rates for ventricular premature beats (VPB) (10.00% vs. 2.50%, P=0.016) and atrioventricular block (AVB) (7.50% vs. 1.67%, P=0.028). Notably, the detection rate of ST-T segment changes via DCG (20.83%) was approximately 2.5-fold higher than that of routine ECG (8.33%, x^2= 7.351, P=0.007).The overall abnormality rate captured by DCG reached 56.67% (68/120), which was significantly superior to the 19.17% (23/120) recorded by routine ECG (x^2= 35.42, P<0.001), suggesting that DCG is a more robust diagnostic tool for identifying occult myocardial involvement in children with MPP.

Table 2 Comparison of Abnormality Detection Rates between Two Electrocardiographic Methods in 120 Pediatric Patients [n (%)]

Variables	Routine ECG (n=120)	24-h DCG (n=120)	x^2	P value
Atrial premature beats	8 (6.67)	22 (18.33)	7.124	0.008
Ventricular premature beats	3 (2.50)	12 (10.00)	5.828	0.016
ST-T changes	10 (8.33)	25 (20.83)	7.351	0.007
Atrioventricular block	2 (1.67)	9 (7.50)	4.821	0.028
Total abnormality rate	23 (19.17)	68 (56.67)	35.42	<0.001

3.3 Comparison of Heart Rate Variability (HRV) Indices between the Two Groups

As shown in Table 3, the HRV time-domain parameters derived from 24-h DCG were compared between the two groups. The levels of SDNN (reflecting overall autonomic regulation), SDANN (reflecting sympathetic tone), as well as RMSSD and PNN50 (reflecting parasympathetic/vagal

activity) in the myocardial damage group were significantly lower than those in the non-damage group (all $P<0.001$). These findings indicate that MPP complicated with myocardial damage is associated with pronounced autonomic dysfunction and suppressed vagal modulation.

Receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive efficacy of the dynamic electrocardiogram time-domain parameter, SDNN, for myocardial damage in children with MPP (Figure 1). The analysis revealed an area under the curve (AUC) of 0.837 for SDNN. Based on the maximization of the Youden Index, the optimal clinical cut-off value for SDNN was determined to be 95.95 ms, yielding a sensitivity of 64.6% and a specificity of 90.3%. These findings indicate that a reduction in SDNN exhibits high specificity in predicting myocardial damage.

Table 3 Comparison of HRV Time-Domain Parameters between the Two Groups of Children with MPP ($\bar{x} \pm s$)

Group	*n*	SDNN (ms)	SDANN (ms)	RMSSD (ms)	pNN50 (%)
Myocardial damage	48	92.4 ±15.6	81.2 ±14.8	28.5 ±8.2	14.2 ±4.8
Non-damage	72	118.6 ±18.2	105.4 ±16.5	39.4 ±10.3	22.4 ±6.5
t	—	-8.14	-8.23	-6.15	-7.96
P value	—	<0.001	<0.001	<0.001	<0.001

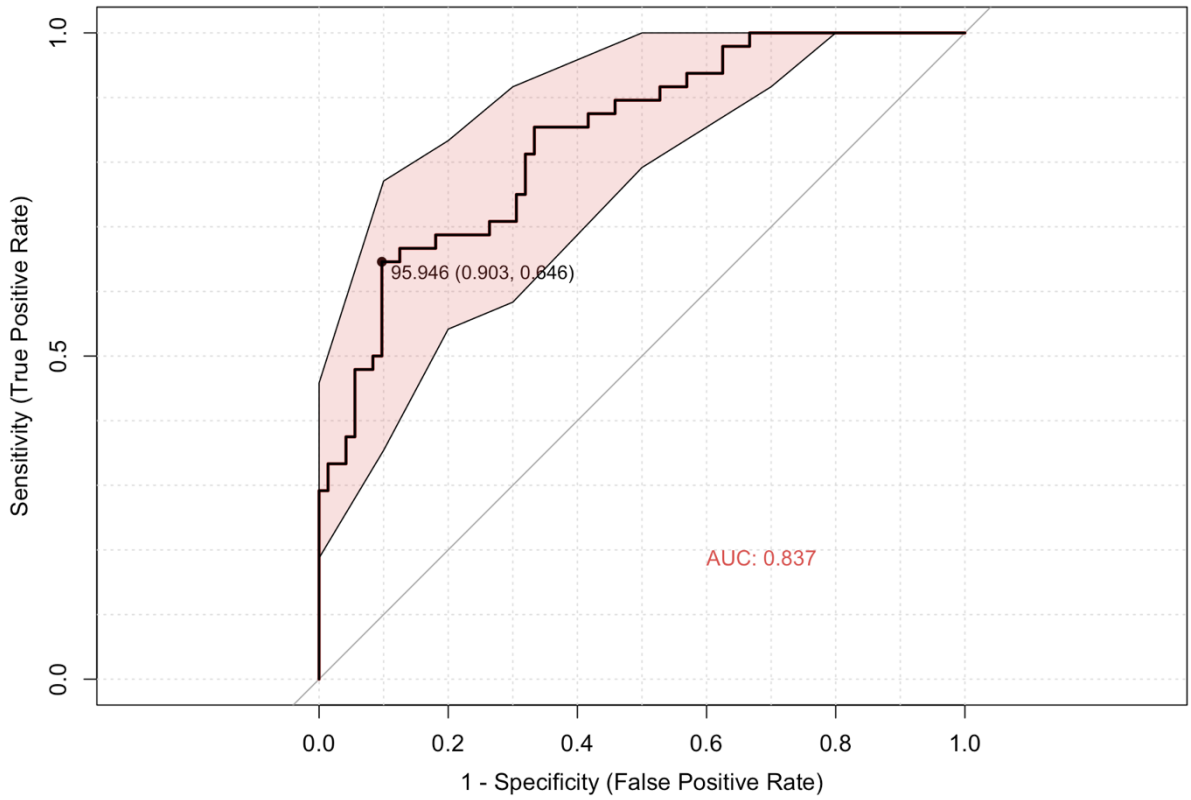

Figure 1 Receiver operating characteristic (ROC) curve of SDNN for predicting myocardial damage in pediatric MPP patients

Note: ROC, receiver operating characteristic; SDNN, standard deviation of the N-N intervals; AUC, area under the curve; 95% CI, 95% confidence interval. The bold black line represents the ROC curve. The shaded pink area indicates the 95% CI band of the curve. The black circular point marks the optimal cut-off value based on the maximum Youden Index: 95.95 ms (Specificity: 90.3%, Sensitivity: 64.6%).

4. Discussion

Mycoplasma pneumoniae (MP) represents a primary etiologic agent of pediatric community-acquired pneumonia. Driven by the emergence of macrolide-resistant strains, the clinical burden of severe and refractory MP pneumonia (MPP) has escalated considerably [1]. Beyond localized pulmonary injury, MP frequently triggers extra-pulmonary complications. Myocardial involvement, a prominent extra-pulmonary sequela, typically presents with insidious early symptoms that are easily overshadowed by respiratory distress, frequently resulting in delayed clinical recognition [2]. Consequently, the present study sought to evaluate the utility of 24-hour dynamic electrocardiography (DCG) and heart rate variability (HRV) indices for the early detection of MPP-associated myocardial injury.

While the exact pathogenesis of MP-induced myocardial injury remains incompletely defined, prevailing evidence points toward a synergistic effect of direct myocardial invasion, immune complex-driven hypersensitivity, and overwhelming systemic cytokine release [2]. These inflammatory cascades disrupt the vascular endothelium and microcirculation, provoking regional myocardial ischemia and focal necrosis that subsequently derange cardiomyocyte electrophysiology. In our cohort, standard 12-lead ECG identified abnormalities in merely 19.17% of MPP cases with cardiac involvement, whereas 24-hour DCG successfully detected aberrations in 56.67%. The diagnostic superiority of DCG was particularly evident in identifying premature atrial/ventricular contractions and transient ST-T segment deviations. These findings imply that cardiac electrophysiological perturbations in pediatric MPP are predominantly paroxysmal. Unlike standard brief ECG recordings, which carry a high risk of false negatives, continuous Holter monitoring captures subtle, state-dependent diurnal variations across different activity levels and sleep cycles [3].

HRV serves as a validated, non-invasive metric for assessing autonomic modulatory effects on the sinoatrial node. Severe systemic infections are known to disrupt neurohumoral homeostasis [4]. Our data revealed that patients with myocardial injury exhibited profoundly depressed global time-domain metrics (SDNN) and sympathetic tone indicators (SDANN) compared to unaffected counterparts, reflecting a compromised overall cardiac autonomic capacity. Furthermore, significant decrements in RMSSD and pNN50 highlighted concurrent parasympathetic (vagal) withdrawal. This profound uncoupling of sympathetic and parasympathetic antagonism inherently lowers the ventricular fibrillation threshold, creating an arrhythmogenic substrate highly susceptible to malignant events during severe pediatric infections [5].

ROC curve analysis further substantiated the prognostic utility of SDNN, yielding an AUC of 0.837 for predicting myocardial damage. Crucially, a predefined cut-off threshold of 95.95 ms provided a striking specificity of 90.3%. Epidemiologically, this robust specificity offers substantial "rule-in" diagnostic confidence: an MPP patient presenting with a 24-hour SDNN below this critical value warrants immediate clinical suspicion for parenchymal cardiac injury. Such findings should prompt physicians to accelerate biomarker reassessments and consider early cardioprotective interventions to avert fulminant myocarditis. Conversely, the moderate sensitivity (64.6%) observed suggests that patients with mild or early-stage myocardial involvement may maintain adequate autonomic compensation. Consequently, an SDNN above the threshold cannot definitively exclude cardiac injury; holistic clinical correlation remains indispensable.

Several limitations must be acknowledged. The retrospective, single-center design with a modest sample size inherently introduces potential selection biases. Additionally, the lack of stratified subgroup analyses comparing different clinical severities (e.g., mild versus severe MPP) restricts broader extrapolations. We also lacked longitudinal DCG follow-up data to assess autonomic recovery post-treatment with macrolides and cardioprotective agents. Future large-scale, multicenter prospective investigations are essential to map the longitudinal trajectory of HRV and establish its

definitive causal links with long-term cardiovascular outcomes in severe MPP cohorts.

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