Appendix 1

Two-Sample T-Test Power Analysis

Numeric Results for Two-Sample T-Test Null Hypothesis: Mean1=Mean2. Alternative Hypothesis: Mean1>Mean2 The standard deviations were assumed to be known and unequal.

Allocation									
Power	N1	N2	Ratio	Alpha	Beta	Mean1	Mean2	S1	S2
0.90459	38	112	2.947	0.05000	0.09541	60.0	55.0	8.7	9.9
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Two-Sample T-Test Power Analysis

Chart Section



References

Machin, D., Campbell, M., Fayers, P., and Pinol, A. 1997. Sample Size Tables for Clinical Studies, 2nd Edition. Blackwell Science. Malden, MA. Zar, Jerrold H. 1984. Biostatistical Analysis (Second Edition). Prentice-Hall. Englewood Cliffs, New Jersey.

Report Definitions

Power is the probability of rejecting a false null hypothesis. Power should be close to one.

N1 and N2 are the number of items sampled from each population. To conserve resources, they should be small. Alpha is the probability of rejecting a true null hypothesis. It should be small.

Beta is the probability of accepting a false null hypothesis. It should be small.

Mean1 is the mean of populations 1 and 2 under the null hypothesis of equality.

Mean2 is the mean of population 2 under the alternative hypothesis. The mean of population 1 is unchanged. S1 and S2 are the population standard deviations. They represent the variability in the populations.

Summary Statements

Group sample sizes of 38 and 112 achieve 90% power to detect a difference of 5.0 between the null hypothesis that both group means are 60.0 and the alternative hypothesis that the mean of group 2 is 55.0 with known group standard deviations of 8.7 and 9.9 and with a significance level (alpha) of 0.05000 using a one-sided two-sample *t*-test.



Figure S1 Flowchart of study participant inclusion according to study eligibility criteria. KD, Kawasaki disease; LGE, late gadolinium enhancement.



Figure S2 Normal Q-Q plots of study variables in acute KD patients. Q-Q plot for assessing normality of the data. The x-axis represents the theoretical quantiles of the standard normal distribution, while the y-axis represents the sample quantiles. Points lying close to the reference line indicate that the data follows a normal distribution, whereas deviations from the line suggest departures from normality. BSA, body surface area; HR, heart rate; CMR, cardiac magnetic resonance; LVEF, left ventricular ejection fraction; KD, Kawasaki disease.



Figure S3 Normal Q-Q plots of study variables in chronic KD patients. Q-Q plot for assessing normality of the data. The x-axis represents the theoretical quantiles of the standard normal distribution, while the y-axis represents the sample quantiles. Points lying close to the reference line indicate that the data follows a normal distribution, whereas deviations from the line suggest departures from normality. HR, heart rate; BSA, body surface area; LVEF, left ventricular ejection fraction; CMR, cardiac magnetic resonance; KD, Kawasaki disease.



Figure S4 Normal Q-Q plots of study variables in normal control. Q-Q plot for assessing normality of the data. The x-axis represents the theoretical quantiles of the standard normal distribution, while the y-axis represents the sample quantiles. Points lying close to the reference line indicate that the data follows a normal distribution, whereas deviations from the line suggest departures from normality. BSA, body surface area; CMR, cardiac magnetic resonance; LVEF, left ventricular ejection fraction; HR, heart rate.



Figure S5 Central illustrative figure: myocardial and hepatic T1 value between patients with LGE⁺ and LGE⁻ in different courses of KD. * means significant difference between any 2 groups (* represents P<0.05, ** represents P<0.01). KD, Kawasaki disease; LGE, late gadolinium enhancement.



Figure S6 Bland-Altman plots were used to assess the agreement of T1 mapping in both hepatic and cardiac. Bland-Altman plots with limits of agreement (95% confidence intervals) demonstrating the interobserver reproducibility of cardiac and hepatic

KD patients with myocardial LGE	Acute phase (n=10), n (%)	Chronic phase (n=14), n (%)
Proportion in all KD patients	10 (20.00)	14 (21.54)
Longitudinal distribution pattern of LGE in the LV		
Basal	5 (50.00)	8 (57.14)
Middle	6 (60.00)	10 (71.43)
Apex	2 (20.00)	3 (21.43)
LGE location		
Lateral wall	4 (40.00)	9 (64.29)
Anterior wall	1 (10.00)	3 (21.43)
Septal wall	3 (30.00)	7 (50.00)
Inferior wall	2 (20.00)	3 (21.43)
LGE pattern		
Subepicardial	3 (30.00)	2 (14.29)
Transmural	1 (10.00)	2 (14.29)
Subendocardial	2 (20.00)	11 (78.57)
Midmyocardial	4 (40.00)	2 (14.29)
Matching of LGE + segments with dilated coronary arteries	2 (20.00)	11 (78.57)

Table S1 Distribution characteristics of myocardial late gadolinium enhancement in KD patients

KD, Kawasaki disease; LGE, late gadolinium enhancement; LV, left ventricular.