Effect of Immunoglobulin Plus Prednisolone in Reducing Coronary Artery Lesion in Patients with Kawasaki Disease

Trial Registration ID: NCT04078568

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Statistical Analysis Plan

The Data Management and Statistical Analysis Plan is directed to support the aims of the study

Version 3.0

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SAP version history		
Version Date	SAP Version	Details of Changes
July 30, 2020	*1.1*	*Draft*
May 11, 2021	*1.2*	*Adding more secondary outcomes*
May 19, 2023	*1.3*	*Adding more detail description of GEE model and secondary outcomes analyzation*
June 15, 2023	*1.4*	*Adding solutions for non-convergence of GEE model*
August 08, 2023	*2.0*	*Adjusting the structure of Introduction, as well as a few document styles and formats; adding more details about the definition of per-protocol population, primary outcome analyses and graphical displays.*
October 31, 2025	*3.0*	● *Adding final solution for non-convergence of GEE model;* ● *Adding eight sensitivity analyses, with three of which suggested by reviewer;* ● *Adding post-hoc exploratory outcome analysis.*

Catalogue

1.Introduction

Kawasaki disease (KD), an acute systematic vasculitis, has been reported worldwide nowadays and more prevalent in Asia. Coronary artery lesion (CAL) is the most important complication and one of the main predictors to the long-term prognosis of KD. CAL can induce coronary artery aneurysm (CAA), occlusion, myocardial ischemia, myocardial infarction and even death, making KD a major cause of the acquired heart diseases in children. The large dose of intravenous immunoglobulin, together with aspirin (abbreviated as IVIG treatment) administered during the acute phase of KD has been well proved in reducing the occurrence of CALs. Given certain proportion of patients response poorly to this treatment, which is commonly linked with higher risk of CAL, it is worth exploring whether more aggressive treatments are able to achieve better effectiveness in reducing the incidence of CAL or not. Corticosteroid, preferred in the treatments of majority of vasculitis, remains controversial in the acute phase treatment of KD. Several studies suggest that the therapy time, regimen and the targeted people require careful considerations in the corticosteroid's treatment in KD. It is still under arguing that using corticosteroids in the initial treatment benefits all the KD patients rather than those with higher risk of IVIG resistance.

The purpose of this Statistical Analysis Plan (SAP) is to define the outcome variables, statistical methods and analysis strategies for a multicenter open-label randomized controlled trial. The trial aims to evaluate the effects of two therapies in reducing the incidence of CAL for children with acute KD: conventional IVIG treatment plus prednisolone (the experimental arm) and only IVIG treatment (the control arm).

2. Study Objective and Outcomes

2.1 Study Objective

This is a superiority randomized controlled trial, aiming to provide high quality evidence for

supporting effects and safety of current treatment combining steroid (corticosteroids) in the IVIG treatment plan for newly onset Kawasaki disease pediatric patients. The primary objective is to evaluate the hypothesis that IVIG treatment plus prednisolone will reduce the incidence of CAL at 1 month of illness comparing with the conventional IVIG treatment. The secondary objectives include comparing the need of additional treatment or not and duration of fever after initial treatment, occurrence of CAL after 1 month of illness onset, changes in laboratory data between two treatment groups, and serious adverse events.

2.2 Outcomes

2.2.1 Primary Outcome

The primary outcome is the occurrence of coronary artery lesions (CAL) at one month from disease onset. Two-dimensional echocardiography will be performed to evaluate CAL at six time points (at admission before treatment as the baseline, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days), and 12th month (±5 days), respectively). The examination included the diameter of the left main coronary artery (LMCA), the left anterior descending artery (LAD), the left circumflex coronary artery (LCX), and the proximal and middle segments of the right coronary artery (RCA). Z scores of each coronary artery will be calculated[1], and the occurrence of CAL is defined as $z \geq 2$ of any coronary artery, including LMCA, LAD, LCX, and the proximal and middle segment of the RCA.

(Time Frame: at one month of illness) (Type: repeatedly measured binary variable)

2.2.2 Secondary Outcomes

1) Changes in z score of LMCA from admission. Data will be measured at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

   (Time Frame: from admission to 12 months of illness) (Type: repeatedly measured continuous

variable)

2) Changes in z score of LAD from enrollment. Data will be measured at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

   (Time Frame: from admission to 12 months of illness) (Type: repeatedly measured continuous variable)

3) Changes in z score of LCX from enrollment. Data will be measured at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

   (Time Frame: from admission to 12 months of illness) (Type: repeatedly measured continuous variable)

4) Changes in z score of the proximal segment of RCA from enrollment. Data will be measured at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

   (Time Frame: from admission to 12 months of illness) (Type: repeatedly measured continuous variable)

5) Changes in z score of the middle segment of RCA from enrollment. Data will be measured at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

   (Time Frame: from admission to 12 months of illness) (Type: repeatedly measured continuous variable)

6) Need for additional treatment or not. The acute KD patients in both arms resistant to the initial IVIG therapy will be given additional treatment, which will be determined by the prescription record.

   (Time Frame: at one month of illness) (Type: binary variable)

7) Duration (hours) of fever (defined as an axillary temperature <37.5 for more than 24 hours) after initiation of initial IVIG among participates of two arms.

   (Time Frame: from initiation of initial IVIG infusion to the first record of being afebrile) (Type: time-to-event variable)

8) Change in serum C-reactive protein (CRP) concentration from admission to 72 hours after

completion of initial IVIG infusion.

(Time Frame: from admission to 72 hours after completion of initial IVIG infusion) (Type: continuous variable)

9) Serious adverse events (including death, hypertension, severe infection, allergic reactions, heart failure, thrombosis, etc.) will be recorded.

(Time Frame: from admission to 3 months of illness) (Type: character variable)

2.2.3 Exploratory Outcomes

The following exploratory outcomes were added by authors during manuscript drafting or suggested by reviewers during revision.

1) Occurrence of medium-to-giant CAAs. CAL classification was based on the maximum z score according to the 2017 American Heart Association guideline[2]. Medium CAA was defined as a maximum z score ≥5 to <10, and all internal diameters <8 mm; large or giant CAA was defined as a maximum z score ≥10, or any internal diameter ≥8 mm. Data were and will be collected at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

(Time Frame: from admission to 12 months of illness) (Type: repeatedly measured binary variable)

2) Occurrence of large/giant CAAs. Data were and will be collected at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

(Time Frame: from admission to 12 months of illness) (Type: binary variable)

3) Occurrence of CAL progression within 3 months of illness onset in all participants and those with CAL at baseline. CAL progression was defined as an increment in the z score ≥1 from baseline in any coronary artery (LMCA, LAD, LCX, proximal and middle segments of RCA) at any given time point within 3 months of illness onset.

(Time Frame: from admission to 3 months of illness) (Type: binary variable)

4) Changes in absolute diameters of LMCA, LAD, LCX, the proximal and middle segments of

RCA from enrollment. Data were and will be collected at six time points of illness: enrollment, 2th week (±2 days), 1th month (±5 days), 3th month (±5 days), 6th month (±5 days) and 12th month (±5 days), respectively.

(Time Frame: from admission to 12 months of illness) (Type: repeatedly measured continuous variable)

3. Study Design

3.1 Design

The trial is a multicenter, superiority, open-label, phase III randomized controlled trial. Patients fulfil the eligibility criteria as outlined in the protocol are invited to participate the trial consecutively and randomized individually to the control or experimental group. Randomization is based on the random block design (block size of 4).

3.2 Trial Sites

The trial will be conducted in 33 hospitals in China: Children's hospital of Fudan university, Jiangxi Provincial Children's Hospital, Qingdao Women and Children's Hospital, Chengdu Women's and Children's Central Hospital, Guangzhou Women and Children's Medical Center, Children's Hospital of Chongqing Medical University, Kaifeng Children's Hospital, Children's hospital of Soochow university, Xuzhou Children's Hospital, Affiliated Hangzhou First People's Hospital, Zhejiang University School of Medicine, Shenzhen Children's Hospital, Shengjing Hospital of China Medical University, First Hospital of Jilin University, the third affiliated hospital of Zhengzhou university, Liuzhou Maternity and Children Healthcare Hospital, Inner Mongolia Autonomous Region People's Hospital, Hunan First People's Hospital, Henan Children's Hospital, Sichuan Academy of Medical Sciences & Sichuan Provincial People's Hospital, Children's Hospital of Nanjing Medical University, Union Hospital, Tongji Medical College, Huazhong University of

Science and Technology, Sun Yat-sen Memorial Hospital, Qilu Hospital of Shandong University, the First Affiliated Hospital of Zhengzhou University, Yuying Children's Hospital of Wenzhou Medical University, Xi'an Children's Hospital, Lanzhou University Second Hospital, Children's Hospital, Capital Institute of Pediatrics, Taihe Hospital Affiliated Hospital of Hubei University of Medicine, Anhui Children's Hospital, Shanghai Children's Hospital, and Bengbu First People's Hospital.

3.3 Interventions

3.3.1 Experimental arm: IVIG + Aspirin + Prednisolone

Patients in this arm will receive conventional IVIG treatment plus additional prednisolone 2 mg/kg per day in the initial treatment. The details are as follows:

In both arms, IVIG treatment is administered within 12 to 24 hours with the maximum dose of 60g. The dose of aspirin will be reduced to 3 to 5 mg/kg per day when fever subsides for 3 days and CRP is normal, and continued for at least 6 weeks after onset of KD. If patients resistant to initial IVIG therapy will be given rescue therapy, including a second dose of IVIG (2 g/kg), or a high dose of methylprednisolone (10 to 30 mg/kg per day), or infliximab (5 mg/kg), or other immunosuppressive agents, or a combination with two or more drugs, or even more aggressive treatment such as plasmapheresis, depending on patients' condition and physicians' experience. IVIG resistance is defined as recurrent or persistent fever (axillary temperature ≥37.5°C or rectal temperature ≥38°C) after 36 hours of completion of initial IVIG infusion.

Patients in the experimental group will additionally receive intravenous methylprednisolone 1.6 mg/kg per day which is given in 2 doses (equal to prednisolone 2 mg/kg, and the maximum dose is 60 mg of prednisolone), then switch to oral prednisolone 2 mg/kg when fever subsides for 3 days. If CRP returns to normal, the prednisolone dose will be tapered over 15 days in 5-day steps, from 2 mg/kg per day to 1 mg/kg per day then to 0.5 mg/kg per day. During corticosteroids administration, 0.5 mg/kg H2-blocker omeprazole will be given per day.

3.3.2 Control arm: Standard Treatment: IVIG + Aspirin

Patients in this arm will receive conventional treatment including, IVIG 2 g/kg and oral aspirin 30 mg/kg per day (given at 3 divided doses) in the initial treatment, which has been elaborated in the corresponding section of 3.3.1.

3.4 Randomization and Blinding

Randomization is stratified by trial sites and done in block size of 4. According to the random seeds, a sequence of study numbers and allocation plan will be generated by the SAS software (version 9.4). Each allocation sequence will be placed in four small, opaque and sealed envelopes numbered in order from 1 to 4, and then enclosed in a larger, opaque and sealed envelope marked with the block number. The randomized plan and concealed envelopes will be prepared by an independent statistician team from the Clinical Trial Unit (CTU) of the Children's Hospital of Fudan University, and delivered to and kept by the site coordinate nurse. Recruited patients will be randomly allocated in a 1:1 ratio to the control or experimental group based on the randomization plan. At each site, after obtaining signed informed consent from the eligible participant, the trained physician will meet with the prespecified institutional nurse to achieve the allocation plan as indicated by the corresponding envelope. The coordinate nurse will open the envelopes in numbered order according to the enrollment order, reveal the treatment allocation to the physician, and record the assignments of the enrolled patient.

Patients and physicians who treat patients will not be masked to the allocation, while pediatric cardiologists who assess CAL by echocardiography will be blinded to the allocation. All researchers conducting outcome assessments will be masked in the trial. The trial statistician will also be blinded regarding the treatment code when developing the statistical analysis plan and writing the statistical programs, which will be validated and completed using dummy randomization codes. The actual allocation will only be provided to the study team after lock of the database.

3.5 Sample Size

This study is a superiority trial. Sample size calculation is performed based on the difference of the primary outcome between the two arms. According to the previous study[3], we assume a difference in the proportion of patients with CAL occurred within the first month of illness between the two groups of 3.3% (12.4% vs 9.1%, odds ratio [OR] = 0.73 for the control arm and the experimental arm, respectively). With an α of 0.05 and a power of 0.8, a sample size of 1400 cases in each group will be needed. With an assumed dropout rate of 10%, a total of 3200 patients are planned for recruitment (1600 per arm).

4. Analysis Populations

4.1 Study Population Data Sets

Three study populations will be considered in the analysis. The primary analysis for the primary objective will be based on the intention-to-treatment population.

Intention-to-Treat population

Intention-to-treat (ITT) population will be defined at the moment the randomization is performed, including all subjects who are randomized.

Participants will be excluded from the ITT analysis if the primary outcome is missing, forming a modified ITT population (mITT).

Per-protocol population

Participants will be excluded from the per-protocol (PP) population if they:

- Being randomized but not receiving any treatment.

- Not adhering to allocated treatment due to:
  - aspirin allergy in either group.
  - being allocated to the experimental arm, while not completing the prednisolone treatment as recommended by the protocol (i.e., stopped early, took less than the recommended doses or tapered more quickly than the recommended period).
  - being allocated to the control arm but were prescribed additional prednisolone treatment due to discovered with IVIG resistance or other co-morbidities, such as juvenile idiopathic arthritis and necrotic lymphadenitis.

The treatment groups in the PP analysis will be determined according to what the participant actually received. This population will be used for the supportive analyses.

Safety population

Participants given any medicine-relate-treatments will be included in this population. Adverse reactions and events will be recorded. This population will be used for the safety analyses.

4.2 Study Close Date

The data collection close date is the date on which the last patient completed 6-month follow-up. All the participants will be followed up for 12 months.

4.3 Data Monitoring and Cleaning

Each center has a data manager dedicated to managing the data. Feedback on the inclusion of the week's study subjects is provided weekly in a WeChat group. All databases are synchronized in the cloud using NutCloud. The center data administrator verifies each sub-center database on a monthly basis. The data will then be checked to ensure that there are no erroneous entries and that all missing data is properly coded.

4.4 Data Check-up

Once all data have been inputted and checked, the database will be locked and a data download request made. The data will be downloaded into SAS, R and Stata formats for statistical analyses.

5. Statistical Analyses

5.1. Primary Outcome Analysis

5.1.1 ITT analysis of the primary outcome - the primary analysis

The primary analysis will be based on the ITT population as defined above. The primary outcome is a binary outcome: CAL occurs or not within 1 month of illness onset. Since CAL will be repeatedly examined at enrollment, 2 weeks, 1 month, 3 months, 6 months and 12 months respectively, generalized estimating equation (GEE) model will be performed, with treatment, visit and interaction between treatment with visit as fixed effect, patient ID as cluster effect, the mean of 5 coronary artery baseline z scores (described in section 2.2.1) as covariate variable. Risk difference (RD) and two-sided 95% confidence intervals (CI) between the two treatment groups will be estimated from the model using a binomial distribution and identity link function. OR with 95% CI at each time point will be obtained using GEE model with binomial distribution and logit link function. Binomial distribution and log link function will be used to obtain estimation of risk ratio (RR) and 95% CI. Exchangeable correlation structure is assumed for within-group variation for all the GEE models. The P value for the interaction term will be used to determine the statistical significance of the group difference at specific time point.

If the above GEE model does not converge, a poisson distribution will be used for the family function. A negative binomial distribution will be subsequently used for family function in case the GEE model based on poisson distribution does not converge. RD, OR and RR with their 95% CI

estimates will be reported by using identity, logit and log link function, respectively. In the final analysis, a normal family distribution was used because none of the three prespecified distribution models converged. RD and 95% CI estimates were derived using identity link function.

5.1.2 Sensitivity analysis

5.1.2.1 Per-protocol analysis of the primary outcome

A supportive analysis of the primary outcome will also be performed on the per-protocol population. Statistical methods will be the same as used in Section 5.1.1.

5.1.2.2 Covariate adjusted analysis of the primary outcome

An analysis of the primary endpoint will be adjusted for baseline information including age, body mass index (BMI), gender and complete or incomplete KD (following the KD diagnostic criteria from the American Heart Association released in 2017). From the above model, the adjusted estimates of RD and two-sided 95% CIs will be derived. The OR and RR with their 95% CIs will also be estimated from the GEE model using logit and log link function as described in Section 5.1.1.

Given the GEE model in the primary analysis has converged, if the GEE model doesn't converge when all covariates are introduced into the model simultaneously, the adjusted model will be established by removing a covariate one by one until the model converges.

5.1.2.3 Accounting for cluster effects of trial sites

A separate GEE model was applied to account for the cluster effects of trial sites, using the statistical method as described in Section 5.1.1. The RD with its 95% CI were estimated.

5.1.2.4 Best-case scenario

Participants with missing primary outcomes were assumed to have the best possible outcome, i.e., CAL did not occur at one month of illness onset in all of them. The statistical method was identical to that used in Section 5.1.1.

5.1.2.5 Worst-case scenario

Participants with missing primary outcomes were assumed to have the worst possible outcome, i.e., CAL occurred at one month of illness onset in all of them. The statistical method was identical to that used in Section 5.1.1.

5.1.2.6 Japanese criteria of CAL

The primary analysis (Section 5.1.1) was repeated using the Japanese Ministry of Health and Welfare criteria for CAL[4], defined as follows:

- In children <5 years, any coronary artery diameter ≥3 mm; in children ≥5 years, ≥4 mm;
- Or ≥1.5 times the diameter of an adjacent segment.

5.1.2.7 Z threshold ≥2.5 for CAL

The occurrence of CAL was re-defined as a z score ≥2.5 in any of the LMCA, LAD, LCX, or proximal/middle RCA segments. The same statistical method as in Section 5.1.1 was used.

5.1.2.8 CAL defined based on LAD and proximal RCA

The occurrence of CAL was re-defined as a z score ≥2 in either the LAD or proximal RCA segment. The same statistical method as in Section 5.1.1 was used.

5.1.2.9 Adjusting for the baseline maximum z score

Using the same method as in Section 5.1.1, this sensitivity analysis adjusted for the maximum z score at baseline instead of the mean of 5 coronary artery baseline z scores.

5.1.2.10 GLM model analysis

This analysis used generalized linear model (GLM) treating response variable as a simple binary outcome without adjustment for any covariate. RD and 95% CI were estimated using a binomial distribution and an identity link function.

Sensitivity analyses 5.1.2.3 – 5.1.2.7 were conducted during manuscript preparation, and analyses 5.1.2.8 – 5.1.2.10 were added in response to reviewers' suggestions during manuscript revision.

5.1.3 Subgroup analysis of the primary outcome

Subgroup analyses will be performed for the primary outcome using GEE model as the primary analysis. We will stratify the analysis by age group (disease onset age <=1 or >1 year), gender (male or female), BMI group (according to different BMI distributions of age and gender in Chinese children, BMI will be stratified into three groups, underweight, normal weight, and overweight and obese, BMI percentile for age and gender of P10 and P85), complete or incomplete KD, IVIG respond or not and CAL presence or not at baseline. Same strategy and methods will be used as described in section 5.1.1.

Same strategy will be used when sensitivity analysis is performed for the secondary outcomes.

5.2 Secondary Outcome Analysis

All secondary outcomes will be analyzed as for a superiority designed trial. Two-sided 95% CIs for

the treatment differences in these outcomes between two groups will be calculated and reported. Secondary outcome analyses will be based on the ITT population unless specified.

5.2.1 Analysis of binary outcomes

Repeated measured binary outcomes will be analyzed with the same tactics as that used for the primary outcome.

Non-repeated measured binary outcomes, referring to need for additional treatment or not, will be summarized by number (%) of participants by treatment group. The proportion difference will be tested by χ^2 methods or fisher exact test. If necessary for analysis, OR with their two-sided 95% CIs of the two groups will be derived from logistic regression. RR and 95% CI were also estimated using a binomial regression with log link function.

5.2.2 Analysis of continuous outcomes

The continuous outcome will be summarized using number of subjects (n), mean, standard deviation (SD), minimum, and maximum by intervention group. The repeated measured outcomes (including changes in z score of LMCA; changes in z score of LAD; changes in z score of LCX; changes in z score of the proximal segment of RCA; changes in z score of the middle segment of RCA) will be analyzed by generalized linear mixed model (GLMM) with intervention, visit and interaction between treatment with visit as fixed effect, patient ID as random effect, their baseline value as covariate variable. The outcomes which are not repeated measures (including change in serum C-reactive protein (CRP) concentration from admission to 72 hours after completion of initial IVIG infusion) will be analyzed by GLM. Gaussian distribution and identity link function will be used in the model. Difference in mean changes of outcome with their two-sided 95% confidence intervals between two groups will be estimated.

5.2.3 Analysis of time to event outcomes

Kaplan-Meier method will be used to plot cumulative event rate of time to first event data. If the Kaplan-Meier curves don't cross, log-rank test will be performed to detect a difference between groups. Cox regression will be performed when the proportional hazards assumption is met and hazard ratio and 95% CI will be estimated.

If the Kaplan-Meier curves cross or the proportional hazards assumption is not satisfied, the win ratio method[5] will be performed.

5.2.4 Analysis of other secondary outcomes (Exploratory Outcome Analysis)

The post-hoc secondary outcomes were analyzed using the same statistical methods described above unless otherwise specified. Specifically, these exploratory outcomes included: occurrence of medium-to-giant CAAs among all participants (Section 5.2.1, repeated measured binary outcomes); CAL progression among all participants (Section 5.2.1, non-repeated measured binary outcomes); and changes in absolute diameters of LMCA, LAD, LCX, the proximal and middle segments of RCA (Section 5.2.2, repeated measured continuous outcomes).

Considering the low incidence of large/giant CAAs (<1%), between-group RRs and two-sided 95% CIs were estimated from a GLM with a negative binomial distribution and log link, adjusting for the mean baseline z scores. Participants with large/giant aneurysms at baseline were excluded from this analysis.

For analysis of CAL progression in participants with baseline CAL, the model was further adjusted for age, sex, KD type, baseline sodium level, CRP concentration, and white blood cell count. RR and corresponding 95% CI were estimated.

5.3 Handling of Missing data

Missing baseline covariates will be imputed using simple imputation methods in the covariate adjusted analysis based on the covariate distributions, should the missing values for a particular covariate be less than 5%. For a continuous variable, missing values will be imputed from random values from a permutated normal distribution with mean and SD calculated from the available sample. For a categorical variable, missing values will be imputed from random values from a uniform distribution with probabilities P1, P2, …, and Pk from the sample. The seed to be used is 20230605. We do not assume the condition that missing of covariate variable at baseline will be over 5%.

6. General Considerations for Data Analyses

SAS (version 9.4) will be used to perform all data analyses and generate majority of data displays. STATA® (version 16.0) or R may also be used for some data analyses and generating statistical graphs. All analyses for the primary and secondary outcome variables will be primarily based on ITT population. Two-side tests and P values will be reported. Regression models for repeated measurement will be used for outcomes that are repeatedly measured, group differences at the prespecified timepoint will be computed and reported.

6.1 Data Summaries

Continuous variables will be summarized according to number of subjects with non-missing data (n), mean, standard deviation (SD), median, minimum, maximum and range interquartile (IQR). The confidence intervals will be reported on summaries of continuous effectiveness variables. Categorical variables will be summarized according to the absolute frequency and percentage of subjects (%) in each category level. The denominator for the percentages is the number of subjects in the treatment arm with data available, unless noted otherwise.

6.2 Graphical Displays

Table 1. Baseline characteristics of patients

Analysis Set: ITT population

Variable	Statistic	Treatment A (N=)	Treatment B (N=)	All (N=)
Gender, n (%)	Female			
	Male			
Age (years)	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			
Height (cm)	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			
Weight (kg)	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			
LMCA	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			
LAD	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			
LCX	n			
	Mean (SD)			
	Median (IQR)			

Variable	Statistic	Treatment A (N=)	Treatment B (N=)	All (N=)
	Min-Max			
RCA (prox)	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			
RCA (mid)	n			
	Mean (SD)			
	Median (IQR)			
	Min-Max			

Table 2. Summary for primary outcomes by generalized estimating equation model

Analysis Set: ITT population

		Participants, No. (%)		Generalized estimating equation model analysis		
Primary outcome	Visit	Treatment A	Treatment B	Risk difference (95% CI)	Odds Ratio (95% CI)	Risk ratio (95% CI)
Covariate unadjusted[a]						
CAL	1 month					
	3 months					
	6 months					
	12 months					
Covariate adjusted[b]						
CAL	1 month					
	3 months					
	6 months					
	12 months					

[a]The GEE model only includes the mean of 5 coronary artery baseline z scores as covariate.

[b]The GEE model was adjusted by including …as covariates.

Table 3. Subgroup analysis of primary outcome by generalized estimating equation model

Analysis Set: ITT population

			Participants, No. (%)		Generalized estimating equation model analysis		
Primary outcome	Variable	Visit	Treatment A	Treatment B	Risk difference (95%CI)	Odds ratio (95%CI)	Risk ratio (95%CI)
CAL	Age (<=1 year)	1 month					
		3 months					
		6 months					
		12 months					
	Age (>1 year)	1 month					
		3 months					
		6 months					
		12 months					
	Gender (F)	1 month					
		3 months					
		6 months					
		12 months					
	Gender (M)	1 month					
		3 months					
		6 months					
		12 months					
	BMI (l)	1 month					
		3 months					
		6 months					
		12 months					
	BMI (N)	1 month					
		3 months					
		6 months					
		12 months					
	BMI (H)	1 month					
		3 months					
		6 months					

			Participants, No. (%)		Generalized estimating equation model analysis		
Primary outcome	Variable	Visit	Treatment A	Treatment B	Risk difference (95%CI)	Odds ratio (95%CI)	Risk ratio (95%CI)
		12 months					

Table 4. Secondary analysis by generalized estimating equation model

Analysis Set: ITT population

		n, mean (SD)		Generalized estimating equation model analysis	
Secondary outcome	Visit	Treatment A (N=)	Treatment B (N=)	Risk difference (95% CI)	P value
LMCA	1 month				
	3 months				
	6 months				
	12 months				
LAD	1 month				
	3 months				
	6 months				
	12 months				
LCX	1 month				
	3 months				
	6 months				
	12 months				
RCA (prox)	1 month				
	3 months				
	6 months				
	12 months				
RCA (mid)	1 month				
	3 months				
	6 months				
	12 months				

Additional tables will be generated if more analyses are conducted.

7. Study Variable List

Table 1 Variable list

Note: T, text variable; D, The date type; N, Continuous variable; B, binary variable; C, categorical variable

No	Variable	Variable Type	Variable interpretation
1	center	T	Name of center
2	id_center	T	Number of center
3	name_pat	T	Acronym of subject
4	patid	N	Study ID of subject
5	time_start	D	Date of enrollment
6	time_end	D	Date of end
7	investigator		Investigator signature
8	inc_1	B	Meeting the diagnostic criteria of Kawasaki disease
9	inc_2	B	Age≥1 month and weight≤30kg
10	inc_3	B	Fever≤10 days
11	inc_4	B	All coronary z values are <10 before IVIG administration
12	inc_5	B	Able to complete follow-up
13	inc_6	B	The guardian is fully informed and signs the informed consent
14	exclusion_1	B	Use of hormones or other immunosuppressants 1 month before KD onset
15	exclusion_2	B	Recurrence of KD

16	exclusion_3	B	Body temperature is normal at the time of inclusion（T≤37.5≥24h）
17	exclusion_4	B	With the following infections: sepsis, septic meningitis, peritonitis, bacterial pneumonia, varicella, and influenza
18	exclusion_5	B	History of serious immune diseases, such as immunodeficiency, or chromosomal abnormalities
19	eligibility	B	Eligibility or not
20	arm	T	Intervention allocation
21	gender	B	Gender
22	birth	D	Date of birth
23	tel	T	Telephone
24	height	N	Height at baseline
25	weight	N	Body weight at baseline
26	id_hospital	T	Hospital admission number
27	date_fisrt_fever	D	Date of first fever
28	date_ivig_on	D	Start date of IVIG administration
29	hour_ivig_on	N	Start time of IVIG administration
30	manifes_fever	B	Clinical feature - fever
31	manifes_fever_days	N	Clinical feature - febrile days
32	manifes_fever_high	N	Clinical feature - maximum heating temperature
33	manifes_conjestion	B	Clinical feature - conjunctivitis
34	manifes_rash	B	Clinical feature - rash
35	manifes_tongue	B	Clinical feature - oral changes
36	manifes_swollen_hand	B	Clinical feature - extremity changes
37	manifes_lymph	B	Clinical feature - cervical lymphadenopathy
38	manifes_crissum	B	Clinical feature - perianal vulva flushing and

			molting
39	maniffes_scar	B	Clinical feature - red and swollen kaba
40	manifes_oth	B	Clinical feature - other
41	manifes_oth_txt	T	Clinical feature - other text
42	a_tempstart	N	Body temperature at IVIG administration begin
43	a_notestart	T	Note of body temperature at IVIG administration begin
44	a_temp0h	N	Body temperature at IVIG administration end
45	a_note0h	T	Note of body temperature at IVIG administration end
46	a_temp6h	N	Body temperature at 6 hours after IVIG administration
47	a_note6h	T	Note of body temperature at 6 hours after IVIG administration
48	a_temp12h	N	Body temperature at 12 hours after IVIG administration
49	a_note12h	T	Note of body temperature at 12 hours after IVIG administration
50	a_temp18h	N	Body temperature at 18 hours after IVIG administration
51	a_note18h	T	Note of body temperature at 18 hours after IVIG administration
52	a_temp24h	N	Body temperature at 24 hours after IVIG administration
53	a_note24h	T	Note of body temperature at 24 hours after IVIG administration
54	a_temp30h	N	Body temperature at 30 hours after IVIG

			administration
55	a_note30h	T	Note of body temperature at 30 hours after IVIG administration
56	a_temp36h	N	Body temperature at 36 hours after IVIG administration
57	a_note36h	T	Note of body temperature at 36 hours after IVIG administration
58	a_temp42h	N	Body temperature at 42 hours after IVIG administration
59	a_note42h	T	Note of body temperature at 42 hours after IVIG administration
60	a_temp48h	N	Body temperature at 48 hours after IVIG administration
61	a_note48h	T	Note of body temperature at 48 hours after IVIG administration
62	a_temp54h	N	Body temperature at 54 hours after IVIG administration
63	a_note54h	T	Note of body temperature at 54 hours after IVIG administration
64	a_temp60h	N	Body temperature at 60 hours after IVIG administration
65	a_note60h	T	Note of body temperature at 60 hours after IVIG administration
66	b1_wbc0	N	White blood cell counts before IVIG administration
67	b1_wbc3d	N	White blood cell counts at 72 hours after IVIG administration
68	b1_wbc6d	N	White blood cell counts at 6 days after IVIG administration

69	b1_wbc9d	N	White blood cell counts at 9 days after IVIG administration
70	b2_neutro0	N	Neutrophil counts before IVIG administration
71	b2_neutro3d	N	Neutrophil counts at 72 hours after IVIG administration
72	b2_neutro6d	N	Neutrophil counts at 6 days after IVIG administration
73	b2_neutro9d	N	Neutrophil counts at 9 days after IVIG administration
74	b3_platelet0	N	Blood platelet before IVIG administration
75	b3_platelet3d	N	Blood platelet at 72 hours after IVIG administration
76	b3_platelet6d	N	Blood platelet at 6 days after IVIG administration
77	b3_platelet9d	N	Blood platelet at 9 days after IVIG administration
78	b4_hemoglob10	N	Hemoglobin before IVIG administration - 1
79	b4_hemoglob20	N	Hemoglobin before IVIG administration - 2
80	b4_hemoglob13d	N	Hemoglobin at 72 hours after IVIG administration - 1
81	b4_hemoglob23d	N	Hemoglobin at 72 hours after IVIG administration - 2
82	b4_hemoglob16d	N	Hemoglobin at 6 days after IVIG administration - 1
83	b4_hemoglob26d	N	Hemoglobin at 6 days after IVIG administration - 2
84	b4_hemoglob19d	N	Hemoglobin at 9 days after IVIG administration - 1

85	b4_hemoglob29d	N	Hemoglobin at 9 days after IVIG administration - 2
86	b4_hemoglobunit	T	Unit of hemoglobin
87	b5_hct0	N	HCT before IVIG administration
88	b5_hct3d	N	HCT at 72 hours after IVIG administration
89	b5_hct6d	N	HCT at 6 days after IVIG administration
90	b5_hct9d	N	HCT at 9 days after IVIG administration
91	b6_crp10	N	CRP before IVIG administration - 1
92	b6_crp20	N	CRP before IVIG administration - 2
93	b6_crp13d	N	CRP at 72 hours after IVIG administration - 1
94	b6_crp23d	N	CRP at 72 hours after IVIG administration - 2
95	b6_crp16d	N	CRP at 6 days after IVIG administration - 1
96	b6_crp26d	N	CRP at 6 days after IVIG administration - 2
97	b6_crp19d	N	CRP at 9 days after IVIG administration -1
98	b6_crp29d	N	CRP at 9 days after IVIG administration - 2
99	b6_crpunit	T	Unit of CRP
100	b7_saa0	N	Serum amyloid A before IVIG administration
101	b7_saa3d	N	Serum amyloid A at 72 hours after IVIG administration
102	b7_saa6d	N	Serum amyloid A at 6 days after IVIG administration
103	b7_saa9d	N	Serum amyloid A at 9 days after IVIG administration
104	c1_esr0	N	Erythrocyte sedimentation rate (ESR) before IVIG administration
105	c1_esr4	N	Erythrocyte sedimentation rate (ESR) after

			IVIG administration
106	c2_alb0	N	Albumin before IVIG administration
107	c2_alb4	N	Albumin after IVIG administration
108	c3_prealb10	N	Prealbumin before IVIG administration - 1
109	c3_prealb14	N	Prealbumin after IVIG administration - 1
110	c3_prealb20	N	Prealbumin before IVIG administration - 2
111	c3_prealb24	N	Prealbumin after IVIG administration - 2
112	c3_prealb30	N	Prealbumin before IVIG administration - 3
113	c3_prealb34	N	Prealbumin after IVIG administration - 3
114	c3_prealbunit	T	Unit of prealbumin
115	c4_gpt10	N	Glutamic-pyruvic transaminase before IVIG administration - 1
116	c4_gpt14	N	Glutamic-pyruvic transaminase after IVIG administration - 1
117	c4_gpt20	N	Glutamic-pyruvic transaminase before IVIG administration - 2
118	c4_gpt24	N	Glutamic-pyruvic transaminase after IVIG administration - 2
119	c4_gptunit	T	Unit of glutamic-pyruvic transaminase
120	c5_got10	N	Glutamic oxalacetic transaminase before IVIG administration - 1
121	c5_got14	N	Glutamic oxalacetic transaminase after IVIG administration - 1
122	c5_got20	N	Glutamic oxalacetic transaminase before IVIG administration - 2
123	c5_got24	N	Glutamic oxalacetic transaminase after IVIG administration - 2
124	c5_gotunit	T	Unit of glutamic oxalacetic transaminase
125	c6_glucose10	N	Blood glucose before IVIG administration -

			1
126	c6_glucose14	N	Blood glucose after IVIG administration - 1
127	c6_glucose20	N	Blood glucose before IVIG administration - 2
128	c6_glucose24	N	Blood glucose after IVIG administration - 2
129	c6_glucoseunit	T	Unit of blood glucose
130	c7_ckmb10	N	Creatine kinase isoenzyme before IVIG administration - 1
131	c7_ckmb14	N	Creatine kinase isoenzyme after IVIG administration - 1
132	c7_ckmb20	N	Creatine kinase isoenzyme before IVIG administration - 2
133	c7_ckmb24	N	Creatine kinase isoenzyme after IVIG administration - 2
134	c7_ckmb30	N	Creatine kinase isoenzyme before IVIG administration - 3
135	c7_ckmb34	N	Creatine kinase isoenzyme after IVIG administration - 3
136	c7_ckmunit	T	Unit of creatine kinase isoenzyme
137	c8_bnp0	N	B-type natriuretic peptide before IVIG administration
138	c8_bnp4	N	B-type natriuretic peptide after IVIG administration
139	c9_probnp10	N	N-terminal moiety of B-type natriuretic peptide before IVIG administration - 1
140	c9_probnp14	N	N-terminal moiety of B-type natriuretic peptide after IVIG administration - 1
141	c9_probnp20	N	N-terminal moiety of B-type natriuretic peptide before IVIG administration - 2

142	c9_probnp24	N	N-terminal moiety of B-type natriuretic peptide after IVIG administration - 2
143	c9_probnpunit	T	Unit of N-terminal moiety of B-type natriuretic peptide
144	c10_sodium0	N	Sodium before IVIG administration
145	c10_sodium4	N	Sodium after IVIG administration
146	c11_troponin10	N	Serum troponin before IVIG administration - 1
147	c11_troponin14	N	Serum troponin after IVIG administration - 1
148	c11_troponin20	N	Serum troponin before IVIG administration - 2
149	c11_troponin24	N	Serum troponin after IVIG administration - 2
150	c11_troponin30	N	Serum troponin before IVIG administration - 3
151	c11_troponin34	N	Serum troponin after IVIG administration - 3
152	c11_troponin40	N	Serum troponin before IVIG administration - 4
153	c11_troponin44	N	Serum troponin after IVIG administration - 4
154	c11_troponinunit	T	Unit of serum troponin
155	c12_tspot0	N	T-SPOT before IVIG administration
156	c12_tspot4	N	T-SPOT after IVIG administration
157	c13_tbil0	N	Serum total bilirubin before IVIG administration
158	c13_tbil4	N	Serum total bilirubin after IVIG administration

159	c14_tg10	N	Triglyceride before IVIG administration - 1
160	c14_tg14	N	Triglyceride after IVIG administration - 1
161	c14_tg20	N	Triglyceride before IVIG administration - 2
162	c14_tg24	N	Triglyceride after IVIG administration - 1
163	c14_tgunit	T	Unit of Triglyceride
164	c15_tc10	N	Total cholesterol before IVIG administration - 1
165	c15_tc14	N	Total cholesterol after IVIG administration - 1
166	c15_tc20	N	Total cholesterol before IVIG administration - 2
167	c15_tc24	N	Total cholesterol after IVIG administration - 2
168	c15_tcunit	T	Unit of total cholesterol
169	c16_ldl10	N	LDL before IVIG administration - 1
170	c16_ldl14	N	LDL after IVIG administration - 1
171	c16_ldl20	N	LDL before IVIG administration - 2
172	c16_ldl24	N	LDL after IVIG administration - 2
173	c16_ldlunit	T	Unit of LDL
174	c17_dimer10	N	D-dimer before IVIG administration - 1
175	c17_dimer14	N	D-dimer after IVIG administration - 1
176	c17_dimer20	N	D-dimer before IVIG administration - 2
177	c17_dimer24	N	D-dimer after IVIG administration - 2
178	c17_dimer30	N	D-dimer before IVIG administration - 3
179	c17_dimer34	N	D-dimer after IVIG administration - 3
180	c17_dimer40	N	D-dimer before IVIG administration - 4
181	c17_dimer44	N	D-dimer after IVIG administration - 4
182	c17_dimer50	N	D-dimer before IVIG administration - 5
183	c17_dimer54	N	D-dimer after IVIG administration - 5

184	c17_dimerunit	T	Unit of D-dimer
185	c18_pct0	N	Calcitonin before IVIG administration
186	c18_pct4	N	Calcitonin after IVIG administration
187	c19_il60	N	IL-6 before IVIG administration
188	c19_il64	N	IL-6 after IVIG administration
189	c20_tnf0	N	TNF before IVIG administration
190	c20_tnf4	N	TNF after IVIG administration
191	d1_date_ucg0	D	Date of ultrasonic cardiogram before IVIG administration
192	d1_date_ucg5	D	Date of ultrasonic cardiogram at 2 weeks of KD onset
193	d1_date_ucg6	D	Date of ultrasonic cardiogram at 1 month of KD onset
194	d1_date_ucg2m	D	Date of ultrasonic cardiogram at 2 months of KD onset
195	d1_date_ucg7	D	Date of ultrasonic cardiogram at 3 months of KD onset
196	d1_date_ucg8	D	Date of ultrasonic cardiogram at 6 months of KD onset
197	d1_date_ucg9	D	Date of ultrasonic cardiogram at 12 months of KD onset
198	d2_lmca0	N	LMCA in ultrasonic cardiogram before IVIG administration
199	d2_lmca5	N	LMCA in ultrasonic cardiogram at 2 weeks of KD onset
200	d2_lmca6	N	LMCA in ultrasonic cardiogram at 1 month of KD onset
201	d2_lmca2m	N	LMCA in ultrasonic cardiogram at 2 months of KD onset

202	d2_lmca7	N	LMCA in ultrasonic cardiogram at 3 months of KD onset
203	d2_lmca8	N	LMCA in ultrasonic cardiogram at 6 months of KD onset
204	d2_lmca9	N	LMCA in ultrasonic cardiogram at 12 months of KD onset
205	d3_lad0	N	LAD in ultrasonic cardiogram before IVIG administration
206	d3_lad5	N	LAD in ultrasonic cardiogram at 2 weeks of KD onset
207	d3_lad6	N	LAD in ultrasonic cardiogram at 1 month of KD onset
208	d3_lad2m	N	LAD in ultrasonic cardiogram at 2 months of KD onset
209	d3_lad7	N	LAD in ultrasonic cardiogram at 3 months of KD onset
210	d3_lad8	N	LAD in ultrasonic cardiogram at 6 months of KD onset
211	d3_lad9	N	LAD in ultrasonic cardiogram at 12 months of KD onset
212	d4_lcx0	N	LCX in ultrasonic cardiogram before IVIG administration
213	d4_lcx5	N	LCX in ultrasonic cardiogram at 2 weeks of KD onset
214	d4_lcx6	N	LCX in ultrasonic cardiogram at 1 month of KD onset
215	d4_lcx2m	N	LCX in ultrasonic cardiogram at 2 months of KD onset
216	d4_lcx7	N	LCX in ultrasonic cardiogram at 3 months

			of KD onset
217	d4_lcx8	N	LCX in ultrasonic cardiogram at 6 months of KD onset
218	d4_lcx9	N	LCX in ultrasonic cardiogram at 12 months of KD onset
219	d5_rca_prox0	N	Proximal segment of RCA in ultrasonic cardiogram before IVIG administration
220	d5_rca_prox5	N	Proximal segment of RCA in ultrasonic cardiogram at 2 weeks of KD onset
221	d5_rca_prox6	N	Proximal segment of RCA in ultrasonic cardiogram at 1 month of KD onset
222	d5_rca_prox2m	N	Proximal segment of RCA in ultrasonic cardiogram at 2 months of KD onset
223	d5_rca_prox7	N	Proximal segment of RCA in ultrasonic cardiogram at 3 months of KD onset
224	d5_rca_prox8	N	Proximal segment of RCA in ultrasonic cardiogram at 6 months of KD onset
225	d5_rca_prox9	N	Proximal segment of RCA in ultrasonic cardiogram at 12 months of KD onset
226	d6_rca_mid0	N	Middle segment of RCA in ultrasonic cardiogram before IVIG administration
227	d6_rca_mid5	N	Middle segment of RCA in ultrasonic cardiogram at 2 weeks of KD onset
228	d6_rca_mid6	N	Middle segment of RCA in ultrasonic cardiogram at 1 month of KD onset
229	d6_rca_mid2m	N	Middle segment of RCA in ultrasonic cardiogram at 2 months of KD onset
230	d6_rca_mid7	N	Middle segment of RCA in ultrasonic cardiogram at 3 months of KD onset

231	d6_rca_mid8	N	Middle segment of RCA in ultrasonic cardiogram at 6 months of KD onset
232	d6_rca_mid9	N	Middle segment of RCA in ultrasonic cardiogram at 12 months of KD onset
233	e1_ecg0	C	ECG before IVIG administration
234	e1_ecg_txt0	T	Text of ECG before IVIG administration
235	e1_ecg1	C	ECG after IVIG administration
236	e1_ecg_txt1	T	Text of ECG after IVIG administration
237	e2_xray0	C	X-ray before IVIG administration
238	e2_xray_txt0	T	Text of X-ray before IVIG administration
239	e2_xray1	C	X-ray after IVIG administration
240	e2_xray_txt1	T	Text of X-ray after IVIG administration
241	e3_mra0	C	MRA before IVIG administration
242	e3_mra_txt0	T	Text of MRA before IVIG administration
243	e3_mra1	C	MRA after IVIG administration
244	e3_mra_txt1	T	Text of MRA after IVIG administration
245	e4_mpi0	C	Myocardial perfusion imaging before IVIG administration
246	e4_mpi_txt0	T	Text of myocardial perfusion imaging before IVIG administration
247	e4_mpi1	C	Myocardial perfusion imaging after IVIG administration
248	e4_mpi_txt1	T	Text of myocardial perfusion imaging after IVIG administration
249	ae	B	Adverse events
250	f1_infection	B	Serious infection
251	f1_infection_hour	N	Occurrence time of serious infection
252	f1_infection_duration	N	Duration of serious infection
253	f1_infection_relation	C	Relation between serious infection and

			drugs
254	f1_infection_intensity	C	Intensity of serious infection
255	f1_infection_treat	C	Treatment of serious infection
256	f1_infection_dose	N	Dose adjustment of serious infection
257	f1_infection_t1	T	Time to start the adjustment of serious infection
258	f1_infection_t2	T	Time to end the adjustment of serious infection
259	f1_infection_outcome	N	Outcome of serious infection
260	f2_skin	B	Skin allergy
261	f2_skin_hour	N	Occurrence time of skin allergy
262	f2_skin_duration	N	Duration of skin allergy
263	f2_skin_relation	C	Relation between skin allergy and drugs
264	f2_skin_intensity	C	Intensity of skin allergy
265	f2_skin_treat	C	Treatment of skin allergy
266	f2_skin_dose	N	Dose adjustment of skin allergy
267	f2_skin_t1	D	Time to start the adjustment of skin allergy
268	f2_skin_t2	D	Time to end the adjustment of skin allergy
269	f2_skin_outcome	C	Outcome of skin allergy
270	f3_heartfailure	B	Heart failure
271	f3_heartfailure_hour	N	Occurrence time of heart failure
272	f3_heartfailure_duration	N	Duration of heart failure
273	f3_heartfailure_relation	C	Relation between heart failure and drugs
274	f3_heartfailure_intensity	C	Intensity of heart failure
275	f3_heartfailure_treat	C	Treatment of heart failure
276	f3_heartfailure_dose	N	Dose adjustment of heart failure
277	f3_heartfailure_t1	D	Time to start the adjustment of heart failure
278	f3_heartfailure_t2	D	Time to end the adjustment of heart failure

279	f3_heartfailure_outcome	C	Outcome of heart failure
280	f4_thromb	B	Thrombogenesis
281	f4_thromb_hour	N	Occurrence time of thrombogenesis
282	f4_thromb_duration	N	Duration of thrombogenesis
283	f4_thromb_relation	C	Relation between thrombogenesis and drugs
284	f4_thromb_intensity	C	Intensity of thrombogenesis
285	f4_thromb_treat	C	Treatment of thrombogenesis
286	f4_thromb_dose	N	Dose adjustment of thrombogenesis
287	f4_thromb_t1	D	Time to start the adjustment of thrombogenesis
288	f4_thromb_t2	D	Time to end the adjustment of thrombogenesis
289	f4_thromb_outcome	C	Outcome of thrombogenesis
290	f5_hypertension	B	Hypertension
291	f5_hypertension_hour	N	Occurrence time of hypertension
292	f5_hypertension_duration	N	Duration of hypertension
293	f5_hypertension_relation	C	Relation between hypertension and drugs
294	f5_hypertension_intensity	C	Intensity of hypertension
295	f5_hypertension_treat	C	Treatment of hypertension
296	f5_hypertension_dose	N	Dose adjustment of hypertension
297	f5_hypertension_t1	D	Time to start the adjustment of hypertension
298	f5_hypertension_t2	D	Time to end the adjustment of hypertension
299	f5_hypertension_outcome	C	Outcome of hypertension
300	f6_othae	B	Other adverse events
301	f6_othae_txt	T	Text of other adverse events
302	f6_othae_hour	N	Occurrence time of other adverse events

303	f6_othae_duration	N	Duration of other adverse events
304	f6_othae_relation	C	Relation between other adverse events and drugs
305	f6_othae_intensity	C	Intensity of other adverse events
306	f6_othae_treat	C	Treatment of other adverse events
307	f6_othae_dose	N	Dose adjustment of other adverse events
308	f6_othae_t1	D	Time to start the adjustment of other adverse events
309	f6_othae_t2	D	Time to end the adjustment of other adverse events
310	f6_othae_outcome	C	Outcome of other adverse events
311	f7_death	B	Death due to adverse events
312	f7_death_time	N	Occurrence time of death due to adverse events
313	ae_solve	B	Adverse events are solved or not
314	remedy	B	Remedy is used or not
315	remedy_ivig	B	Second IVIG administration in the remedy
316	remedy_ivig_dose	T	Dose of second IVIG administration in the remedy
317	remedy_mp	B	Methylprednisolone in the remedy
318	remedy_mp_dose	T	Dose of methylprednisolone in the remedy
319	remedy_ifx	B	Infliximab in the remedy
320	remedy_ifx_dose	T	Dose of infliximab in the remedy
321	remedy_ims	B	Other immunosuppressants in the remedy
322	remedy_ims_dose	T	Dose of other immunosuppressants in the remedy
323	remedy_pte	B	Plasma exchange in the remedy
324	remedy_oth	B	Other remedy
325	remedy_oth_txt	T	Text of other remedy

326	date_tempcover	D	Date of final body temperature recovery
327	date_revisit	D	Date of last revisit
328	compliance	B	Completing the protocol or not
329	quit_person	C	Proposer of quitting
330	quit_person_oth	T	Other proposer of quitting
331	quit_reason	C	Reason of quitting
332	quit_reason_txt	T	Text of other reason of quitting
333	date_crf	D	Date of CRF

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