Association between the miRNA-149 rs2292832 T>C polymorphism and Kawasaki disease susceptibility in a southern Chinese population

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Abstract

Background: Kawasaki disease (KD), which is characterized by vasculitis, is prone to occur in patients under 5 years of age, has an ambiguous etiology, and displays coronary artery lesions as the chief complication. Previous studies have linked miRNA-149 to cancers, and rs2292832 T>C is related to allergic diseases and inflammatory bowel disease, which both show immune system disorders and coronary artery disease. Therefore, we performed a study concentrating on the association between the miRNA-149 rs2292832 T>C polymorphism and KD susceptibility.

Methods: The subjects enrolled were 532 children with KD and 623 controls. We used TaqMan real-time PCR to obtain the genotypes of the rs2292832 T>C polymorphism.

Results: Ultimately, no significant association was found between the miRNA-149 rs2292832 T>C polymorphism and KD susceptibility, even in stratification analysis.

Conclusion: Our results indicated that in southern Chinese patients, the miRNA-149 rs2292832 T>C polymorphism did not affect KD susceptibility, which needs to be further confirmed.

KEYWORDS
Kawasaki disease, miRNA-149, polymorphism

Abbreviations: CAA, coronary artery aneurysm; CAL, coronary artery lesion; CI, confidence interval; GWAS, genome-wide association study; KD, Kawasaki disease; miRNA, microRNA; OR, odds ratio.

Li and Wang are contributed equally to this work.

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1 | INTRODUCTION

Kawasaki disease (KD), an autoimmune syndrome characterized by vasculitis, is prone to emerge in children under 5 years of age and was first detected in 1967.1 Currently, acquired heart disease is most likely caused by KD in numerous developed countries.2 Multiple complications of KD affect the cardiovascular and non-cardiovascular systems. As common complications in the cardiovascular system, coronary artery aneurysms are increasingly being studied along with coronary artery dilatation.3

According to some investigations, the annual incidence of KD has gradually increased in recent years in multiple places, such as Italy, Australia, South Korea, Japan, Inner Mongolia, and Jilin.4-9 Therefore, the incidence of KD is gradually rising in most regions, which is one of the reasons for studying this disease. The number of boys suffering from KD is approximately twice that of girls, and the mean age of onset in most regions is approximately 28 months.4,5,9-12 In individuals with KD, the occurrence rate of coronary artery lesions (CALs) was 23.5%, 32.6%, 40.2%, 41%, and 41.4% in Australia, Athens, Inner Mongolia, Jordan, and Jilin, respectively.4,5,8,13,14

Thus far, the pathogenesis of KD has not been fully elucidated. Single or multiple pathogens could stimulate an excessive immune response in susceptible individuals, easily inducing a systemic inflammatory response, which may give rise to KD.15-17 Through the analysis of bacteria and KD in the pharynx and serum, researchers found that streptococcus might be related to the onset of KD.18 The American Heart Association introduced criteria to distinguish the classic and incomplete forms of KD from other diseases. Classic KD is diagnosed in patients sustaining a fever for at least 4 days who have ≥4 chief clinical signs. Uncommon KD presents as a fever with an unexplained cause, <4 chief clinical signs, and corresponding echocardiographic and laboratory findings.2

MicroRNAs (miRNAs) are noncoding RNA molecules of approximately 20 nucleotides that can affect the expression of some genes.19,20 More than 25 miRNAs have been identified for differentiating patients with KD from patients with fever using next-generation sequencing. Quantitative polymerase chain reaction was selected for further analysis of some of these miRNAs, but researchers did not find a connection between miRNA-149 and KD.21 In searching the literature, we found that miRNA-149 has a definite association with some cancers, such as lung cancer, colorectal cancer, laryngeal squamous cell carcinoma, hepatocellular carcinoma, and neuroblastoma.22-26 To a certain extent, the alteration of rs2292832 T allele could enhance the severity of inflammatory bowel disease, which is similar to KD, as it is caused by abnormal activation of the immune system by a pathogen.29

Nevertheless, no study has examined the association of the miRNA-149 rs2292832 T>C polymorphism with KD susceptibility. Based on data from our medical center, we examined 532 KD children and 623 controls to carry out a case-control study and evaluate the relationship between the miRNA-149 rs2292832 T>C polymorphism and KD susceptibility in a southern Chinese population.

2 | MATERIALS AND METHODS

2.1 | Ethics statement

The present study conducted at Guangzhou Women and Children’s Medical Center satisfied the criterion for human subjects in the Declaration of Helsinki. The Review Committee (2014073009) of the medical center approved this study. Written informed consent was obtained from the legal guardians of all participants.

2.2 | Study subjects

All participants recruited were unrelated Chinese Han individuals from southern China. A total of 623 healthy controls and 532 patients diagnosed with KD were recruited from January 2012 to January 2017 as we described previously.30-32 Two milliliters of fresh blood was collected from each participant, and 200 µL of each blood sample was employed to extract DNA.

2.3 | DNA extraction and genotyping

A TIANamp Blood DNA Kit (Centrifugal column, Tiangen) was used to extract DNA from the blood samples according to the manufacturer’s instructions.34-36 TaqMan real-time PCR assays were set up in 384-well plates containing positive and negative samples and were carried out in an ABI Q6 system (Applied Biosystems) to genotype the miRNA-149 rs2292832 T>C polymorphism.37-39

2.4 | Statistical analysis

Utilizing two-sided chi-square test and chi-square test, we obtained the distribution of various parameters and the genotype distribution between cases and controls. A goodness-of-fit chi-square test was used to assess the control genotype distributions for Hardy-Weinberg equilibrium. Odds ratios (ORs) and 95% confidence intervals (CIs) of homozygotes (CC in comparison with TT), heterozygotes (TC in comparison with TT), the recessive model (CC in comparison with TT + TC), and the dominant model (CC + TC in comparison with TT) were obtained by univariate logistic regression and used to
illustrate the relationship between the miRNA-149 rs2292832 T>C polymorphism and KD susceptibility. As data were divided into subgroups on the basis of coronary lesion (CAL), coronary artery aneurysm (CAA), age, gender, and genotypes (TT, TC/CC), associations between KD susceptibility and the polymorphism were considered in depth via stratification. In accordance with the Japanese Kawasaki Disease Research Committee as well as the coronary z score, artery lesions with a luminal diameter ≥4.0 mm in children ≥5 years of age or ≥3.0 mm in children <5 years of age, a section with a diameter ≥1.5 times larger than a proximal segment or clearly irregular profile of the artery, were defined as CALs. Depending on the diameter of the personal coronary artery, CAAAs were defined as major damage of the coronary artery.40 SAS software (version 9.4; SAS Institute) was applied to implement all statistical analyses.

3 | RESULTS

3.1 | Population features

A total of 623 healthy controls and 532 KD children were included in the current study (Table S1). The average age of morbidity of KD patients was 28.39 months, and in contrast, the average age of the controls was 28.48 months. There were 167 (31.39%) female KD patients, a lower number than the 365 (68.61%) male KD patients. There were 167 (31.39%) female KD patients, and in contrast, the average age of the controls was 28.48 months. There were 167 (31.39%) female KD patients, a lower number than the 365 (68.61%) male KD patients.

3.2 | The miRNA-149 rs2292832 polymorphism and KD susceptibility

We successfully genotyped 507 KD patients and 612 healthy controls, as illustrated in Table 1. We employed the goodness-of-fit chi-square test, and the *P* value obtained was .791, satisfying Hardy-Weinberg equilibrium. The genotype distributions of miRNA-149 rs2292832 were as follows: 67.26% (TT), 23.67% (TC), and 9.07% (CC) in the KD patients and 70.10% (TT), 22.06% (TC), and 7.84% (CC) in the healthy controls. No significant association was discovered between the miRNA-149 rs2292832 T>C polymorphism and KD susceptibility (TC compared with TT: OR = 1.12, 95% CI = 0.84-1.48; CC compared with TT: OR = 1.20, 95% CI = 0.78-1.84; CC compared with TT + TC: OR = 1.17, 95% CI = 0.76-1.78, CC + TC compared with TT: OR = 1.14, 95% CI = 0.88-1.47).

3.3 | Stratification analysis

We assessed the connection between the rs2292832 T>C polymorphism and KD susceptibility after stratification by age, gender, CAL, and CAA. As shown in Table 2, the adjusted *P* values of the differences between the associations of CAL, NCAL, CAA, and no coronary artery aneurysm (NCAA) with the TT genotype and the TC/CC genotypes of rs2292832 were 0.306, 0.507, 0.178, and 0.452, respectively. No notable association was observed in any of the subgroups.

4 | DISCUSSION

There are few studies on miRNAs and KD, and there is no research on the rs2292832 T>C polymorphism and KD susceptibility. In the first genome-wide association study (GWAS) for KD, variants of several genes (such as Inx1, CAMK2D, ZFHX3, CSM1D, and tcp1) were suggested to be associated with KD susceptibility.41 In addition, SNPs in rs2254546, rs2857151, rs4813003, rs16921209, rs7922552, rs17076896, rs12068753, rs4786091, and rs2833195 and some SNPs in BLK, CD40, and FCGR2A may be relevant to susceptibility, clinical presentations (such as CAA and CAL), laboratory data (such as CRP), treatment of KD, and gender in KD in GWASs.42-48

Therefore, we examined the relationship between the occurrence of KD and the miRNA-149 rs2292832 T>C polymorphism in this case-control trial. We failed to acquire meaningful positive results that could assist future experiments. KD-related allergic diseases, inflammatory bowel disease, and coronary artery disease may be related to the rs2292832 T>C polymorphism. A study of the relationship between the rs2292832 T>C polymorphism and KD is necessary. The negative results of this article will not only assist others in excluding the possibility of this association in a southern Chinese population but will also provide similar ideas for further studies, such as studying the nexus between the rs2292832 T>C polymorphism and KD susceptibility in other populations around the world.

As mentioned above, 168 (31.58%) children suffering from CAL were reported in southern China. In Inner Mongolia, Jilin, and Jordan, the incidence of CAL in KD was approximately 40%.4,8,13 In Australia, the incidence of CAL in KD was approximately 23%.7 The incidences of CAL in Western countries, such as Ireland, Italy, and Athens, Greece, were 2.9%, 15.6%, and 32.6%, respectively, which was close to that in southern China.14,49,50 The distinct occurrence of CAL in KD patients worldwide may be related to the frequency of rs2292832 genotypes due to differences in race based on diverse hereditary susceptibility, geography, time, definition of coronary artery injury, and other factors.

Studies have shown that genetic variants may increase the susceptibility to asthma and allergic rhinitis, and asthma was suggested to be connected with the miR-149 rs2292832 T>C polymorphism in a study by Hu et al.29 Similar to KD, these are all inflammatory diseases, and correlations with these diseases are possible. From a laboratory and clinical perspective, according to the relevant literature and Harada score, age, gender, plasma albumin, CRP, WBC, and Hc, all could be risk factors for CAL in KD patients.51-53 Bai et al.54 reported that increased CRP levels (≥30 mg/L), hepatomegaly, ESR acceleration (≥ 40 mm/h), and IVIG ineffectiveness had a significant effect on CAL. Particularly in patients aged <24 months, age-dependent
increases in the LDH level, white blood cell count, erythrocyte sedimentation rate, and platelet count in Korean patients diagnosed with KD were caused by polymorphisms in two genes, ITPKC and SLC11A1. The diverse incidences of CAL may be due to different averages in various laboratory indices. According to epidemiology reports from Athens, 42.2% of sufferers with classic KD had CAL, but only 4.5% of sufferers with incomplete KD had CAL. We suspect that the ratio of typical KD to incomplete KD is also one of the factors contributing to the diverse incidence of CAL.

The miRNA-149 rs2292832 T>C polymorphism might not have an influence on the occurrence of KD in our current research. Nevertheless, our finding of no positive relationship is needed to assess further related aspects or exclude other interferential conditions. For instance, our study subjects were limited to children in southern China, an uncharacteristic Chinese population. The effect of low-penetrance hereditary variation on the result may not have been detected because of the small sample size and relatively high morbidity. In addition, some relevant mutation sites or genes exert a substantial effect on the final result. The functions of the rs2292832 T>C polymorphism might be concealed by interacting elements. The mechanism of KD caused by multiple factors is still unclear. Therefore, our research is not convincing without other useful data, such as disease history and living environment. Based on these results, we will try to address these issues in the future.

5 | CONCLUSIONS

In conclusion, we found that the miRNA-149 rs2292832 T>C polymorphism did not affect KD susceptibility in southern Chinese patients, which needs to be further investigated in other populations.

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AUTHOR CONTRIBUTIONS
All authors contributed significantly to this work. JL, ZJ, XR, XG, CJ, LZ, and HZ performed the research study and collected the samples and data; JL and XG analyzed the data; XG and MC designed the research study; JL and JW wrote the study; and JL and XS prepared all the Tables. All authors reviewed the study. In addition, all authors have read and approved the study.

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DATA AVAILABILITY STATEMENT
Data are available upon request.

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SUPPORTING INFORMATION
Additional supporting information may be found online in the Supporting Information section.

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