

CASE REPORT

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Infant Kawasaki disease complicated with supraventricular tachycardia: a case report and literature review

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Abstract

Background The occurrence of arrhythmias as a complication of Kawasaki disease (KD) is extremely rare. Moreover, previous literature showed a low incidence of arrhythmias during the acute phase of KD, and the majority occurred in the subacute and chronic phases. To date, we have found only 17 sporadically reported global cases in the available literature.

Case presentation We present the first documented case of an infant with KD complicated with supraventricular tachycardia (Atrioventricular reentrant tachycardia) during the acute phase. The arrhythmia resolved promptly after the combination therapy of intravenous Immunoglobulin (IVIG) and steroids during the acute phase since the inflammation subsided. Additionally, we conducted a review and summary of cases involving KD-related arrhythmias.

Conclusions KD rarely causes arrhythmias, which might be associated with myocarditis and myocardial ischemia attributed to scar formation and/or excessive inflammatory factors damaging the conduction system. Strengthening the early identification and management of complications in patients with KD and personalized follow-up strategies for high-risk children during the chronic phase can enhance patients' prognosis.

Keywords Kawasaki disease, Arrhythmia, Supraventricular tachycardia, Atrioventricular reentrant tachycardia

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Background

KD is an acute vasculitis that primarily affects children, and its exact etiology remains unclear. Due to its impact on the coronary arteries and the heart (including the myocardium, pericardium, and cardiac valves), KD has emerged as a leading cause of acquired cardiac conditions in the pediatric population [1, 2]. Notably, inflammatory processes within cardiac tissues, scar formation, and abnormalities in electrical signal conduction are all potential factors that can lead to the development of arrhythmias. The type of arrhythmia, its duration, and the overall health status of the patient can significantly influence the cardiac function of affected children, potentially posing severe threats to their lives.

After a comprehensive literature review, we found 17 reported cases of KD having complicated arrhythmias [3–13]. It showed that such arrhythmias generally occurred in the chronic and subacute phases of KD; nonetheless, rare was found in the acute phase. Furthermore, although various types of arrhythmias such as atrial fibrillation, ventricular premature contractions, ventricular tachycardia, bundle branch block, and atrio-ventricular block have been reported, supraventricular tachycardias are rarely reported in the literature. There were only two cases on record [3, 10]. Additionally, even among KD patients with regression of coronary artery lesions or absence of coronary artery lesions, there remains a risk of developing arrhythmias [3, 7–10].

Herein, we reported a Chinese KD patient experiencing supraventricular tachycardia (SVT) during the acute phase of the disease for the first time. Furthermore, we reviewed and summarized available cases involving KD-related arrhythmias, aiming to enhance pediatric awareness of this condition in KD.

Case presentation

On June 21, 2023, a 6-month-old and 13-day-old male infant was brought to the Emergency Department of West China Second University Hospital, Sichuan University, due to an unexplained persistent fever for 4 days and mild cough. Blood cell examination revealed a significant increase in white blood cells (WBC count of $16.6 \times 10^9/L$, normal range $4.03\text{--}11.09 \times 10^9/L$; neutrophils percentage 73.1%, normal hemoglobin (133 g/L, normal range 108–144 g/L), normal platelet count ($398 \times 10^9/L$, normal range $128\text{--}420 \times 10^9/L$), and elevated C-reactive protein (CRP 42.1 mg/L, normal range 0–8 mg/L). The erythrocyte sedimentation rate (ESR) was elevated (54 mm/h, normal range <25 mm/h). Initial assessment suggested that the fever might be caused by an acute upper respiratory tract infection, and ceftriaxone was administered for 2 days. However, he had a persistent high fever without any significant worsening of infection symptoms (only mild cough and diarrhea). On the 6th day of illness, new

symptoms appeared, including generalized congestive rash, conjunctival congestion without exudate, flushed and chapped lips, limb swelling, enlarged lymph nodes in the right neck, and redness at the Bacille Calmette-Guérin (BCG) vaccination site. Breath sounds in both lungs were symmetrical, with no rales. The abdomen was soft, and the liver and spleen were not palpable or enlarged, and neurological examination showed negative pathological signs. These symptoms were typical clinical manifestations of KD. Due to a fever persisting for more than five days, no response to antimicrobial treatment, and the manifestation of six clinical symptoms that meeting with the diagnostic criteria for complete KD [1, 2], in conjunction with elevated ESR, WBC, and CRP levels, we are considering a diagnosis of KD. Further examinations revealed a significant increase in WBC (WBC count of $15.4 \times 10^9/L$, neutrophils 59.2%), a slight decrease in hemoglobin (106 g/L), a slight increase in platelet count ($430 \times 10^9/L$), and elevation of CRP (55.5 mg/L). Liver enzymes were markedly elevated (Alanine Aminotransferase (ALT) 280U/L, normal range $<49U/L$; Aspartate Aminotransferase (AST) 231U/L, normal range $<40U/L$). Electrolyte levels were within normal ranges (Na^+ , K^+ , Ca^{2+}). Cardiac injury markers were within normal limits (cTnI, CK-MB, Myoglobin). Further, infectious diseases and other immune diseases were excluded by pathogen-specific examinations such as respiratory pathogen tests, stool routine and culture, blood culture, sputum culture, and serological testing for autoantibodies. Two novel coronavirus nucleic acid tests were negative (ruling out the possibility of multisystem inflammatory syndrome in children - MIS-C [14, 15]). The echocardiogram revealed the presence of a small coronary artery aneurysm in the left coronary artery (LCA=2.7 mm, Z-score=3.27), dilation in the left anterior descending artery (LAD=2.0 mm, Z-score=2.01), while the left circumflex artery and right coronary artery showed normal dimensions (LCX=1.7 mm, Z-score=1.52; RCA=2.0 mm, Z-score=1.94). The dimensions of the atria and ventricles were normal, and both systolic and diastolic functions of the heart appeared normal.

Based on the above findings, the diagnosis of KD was proposed. He was transferred to our inpatient department. However, upon admission, we observed a significantly elevated heart rate (190 beats per minute) during cardiac auscultation, with a regular rhythm and normal heart sounds. While sinus tachycardia was initially suspected, the child exhibited no signs of restlessness, remained quiet at rest, had a body temperature of $38^\circ C$, normal urine output, normal limb circulation, normal blood pressure (80/46 mmHg), normal oxygen saturation (SpO_2 97%), and normal respiratory rate (32 breaths per minute). Absence of irritability, high fever, shock, hypoxia, and anemia ruled out underlying causes for

sinus tachycardia. Of greater significance, an electrocardiogram (ECG) revealed a ventricular rate of 219 beats per minute, demonstrating a regular rhythm, normal QRS waveform, consistent PR and RR intervals (RP interval < PR interval, RP interval was 93 ms), and retrograde P' waves observed in lead aVR and II (Fig. 1A). Consequently, the final diagnosis was SVT (Atrioventricular

reentrant tachycardia) rather than sinus tachycardia. He had a negative history of autoimmune or cardiac diseases. Considering the patient's underlying KD and the absence of prior clinical episodes of SVT, our experience and literature reports led us to speculate this condition might be associated with the inflammatory burden of KD.

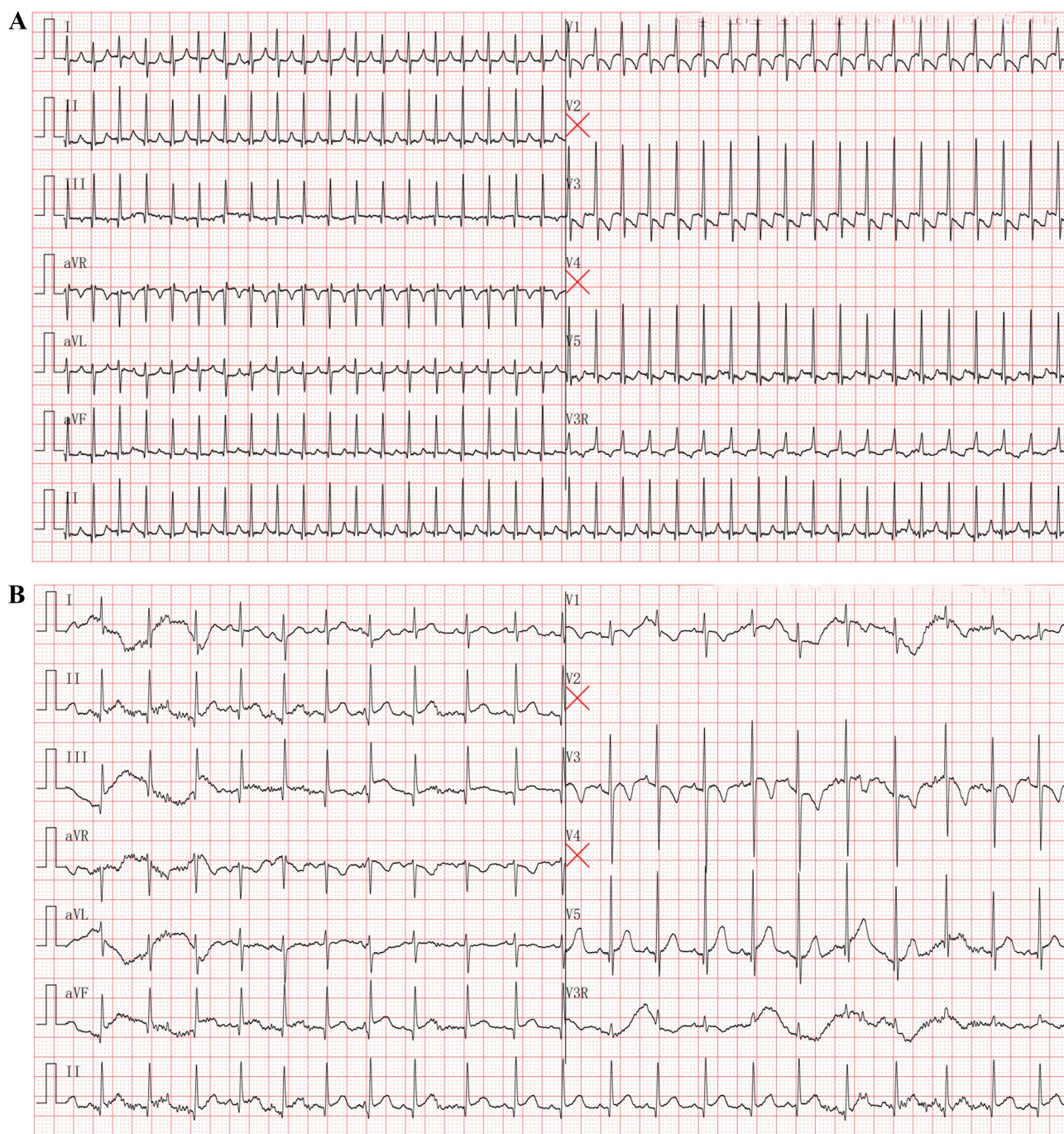


Fig. 1 Comparison of electrocardiograms (ECGs) before (B) and after (A) anti-inflammatory treatment. (A) The ECG revealed a ventricular rate of 219 beats per minute, demonstrating a regular rhythm, normal QRS waveform, consistent PR and RR intervals, and retrograde P' waves observed in lead aVR. (B) Repeated ECGs showed normal sinus rhythm with normal PR and QT intervals and the absence of SVT's δ waves

Because of his hemodynamic stability and possibly the aforementioned cause of SVT, we initially did not perform antiarrhythmic treatment but continued to monitor his breathing rate, heart rate, blood pressure, and capillary refill time. At the same time, we quickly initiate anti-inflammatory measures, including oral aspirin (30 mg/kg/day) and IVIG at a single dose of 2 g/kg, and methylprednisolone (2 mg/kg twice a day), according to the treatment protocol for KD [1, 2]. Fortunately, one hour later, repeated ECGs showed normal sinus rhythm with normal PR and QT intervals and the absence of SVT's δ waves (Fig. 1B). The child's body temperature gradually decreased. However, after 48 h of treatment, the child experienced a recurrence of fever, suggesting the possibility of IVIG resistance. Considering the patient's young age, intense inflammatory response, concurrent arrhythmia, and impaired liver function, a second round of IVIG treatment (single dose of 2 g/kg) was initiated. Finally, the child's symptoms gradually improved, and there was no further occurrence of fever two days later. On the 11th day of illness, blood indicators, 12-lead ECG, Holter ECG, and echocardiography were all normal. The child was discharged with oral aspirin (3 mg/kg/day) for six weeks. In his regular follow-ups at 1 month, 3 months, 6 months, and 1 year, no further arrhythmias were found. Echocardiography showed normal coronary arteries and cardiac function. However, we did not rule out the possibility of a congenital bypass tract in the conduction system or other external factors triggering the event. Since SVT occurred only once at the onset of the illness, his parents declined an invasive cardiac catheter electrophysiology study. We also informed the family that if SVT occurs again in the future, it would be necessary to consider conducting an electrophysiological study.

Discussion

In this study, we present the documented case of an infant complicating with SVT during the acute phase of KD for the first time. Previous literature showed arrhythmias rarely occurred during the acute phase of KD in children, which were predominantly found in the subacute and chronic phases. Until now, we found only 17 cases were sporadically available worldwide in the literatures [3–7, 9–13] (Table 1). Our study showed pediatric clinicians should enhance the awareness of arrhythmia occurring in patients with KD. Moreover, it was critical to recognize the arrhythmia other than Coronary Artery Lesions (CALs) during the short- and long-term follow-up of KD.

Subsequently, we further reviewed 17 patients with KD (11 males and 5 females, aged 4 months to 34 years) complicating with arrhythmias, which predominantly occurred during the chronic phase of the illness, ranging from 2 days to 33 years. It indicated 70.6% (12/17)

patients experiencing arrhythmias in the chronic phase, 17.6% (3/17) in the subacute phase, and 5.9% (1/17) in the acute phase. Among these 17 patients with tachycardia, supraventricular tachycardia accounted for 2/17 cases (1 in the subacute phase and 1 in the chronic phase), atrial fibrillation for 2/17, premature ventricular contractions for 5/17, ventricular tachycardia for 7/17, and ventricular fibrillation for 1/17. The mechanism of arrhythmias as a complication of KD remained complex. Fujiwara et al. [16] conducted a pathological study that revealed inflammatory and edematous changes in the sinoatrial node and atrioventricular conduction system during the acute phase of the disease (days 0–9). However, no necrosis was observed in the conduction cells. During the subacute phase of the disease (days 21–31), severe edematous changes and mild fibrosis were observed in the perivascular areas, while the conduction cells exhibited widespread compression, degeneration, and necrosis. These changes can potentially lead to abnormalities in electrical activity. In addition, changes in coronary artery morphology [17] and vascular endothelial dysfunction caused by inflammatory responses [18] can contribute to impaired perfusion of the arteries supplying the conduction system (sinus node and atrioventricular node), ultimately leading to disruptions in electrical signal conduction. After entering the chronic phase of the disease, the formation of scars following the resolution of inflammation becomes a high-risk factor for developing arrhythmias [12, 19]. On the other hand, changes in coronary artery morphology can lead to abnormalities in blood flow perfusion and the formation of blood clots. The formation of coronary artery thrombosis and coronary artery stenosis can result in myocardial ischemia, ultimately leading to malignant arrhythmias. In our case, the child's presence of IVIG resistance, Coronary artery aneurysms (CAAs), and increased levels of inflammatory indicators showed a severe inflammatory burden, which might account for arrhythmia during the acute phase of KD. The arrhythmia resolved promptly as the inflammation subsided after receiving the combination therapy of aspirin, IVIG, and steroids. However, it is necessary to be aware of arrhythmia development attributed to the formation of myocardial scars following inflammatory damage, and long-term follow-up is critically required.

In addition, the occurrence of CAAs is a significant clinical concern in patients with KD. It was widely recognized that the development of CAAs associated with intensive inflammatory burden, which more likely occurred during the acute phase of KD. Therefore, the abnormal presence of the cardiac conduction system during the acute phase of KD, such as arrhythmias, might be an independent risk factor for CAAs in KD. On the other hand, the sustained presence of these aneurysms may potentially impact myocardial and conduction cells'

Table 1 Literature review of Kawasaki disease cases complicated by arrhythmias

Year/Author	Types of arrhythmia	Age (Years)	KD onset (Years)	Gender	Duration of fever	Incomplete Kawasaki Disease	Coronary Artery condition	ECG Evaluation and Symptom Assessment of MI	Cardiac function status	Number of IVIG/ Steroid hormone	Antiarrhythmic therapy	Surgical treatment	Treatment outcome
1995/Haney, I. [11]	Ventricular arrhythmia	-	3 W	-	-	-	-	-	-	3/Yes	-	-	Normal
1996/Nakada, T. [9]	FPVCs; VT	11 Y	9 Y	Female	11 d	-	Normal	1/-	Normal	-	No	-	Normal
1998/Seymour, J. J. [5]	SVT	12 Y	10 Y	Female	-	-	Normal	Normal/Yes (Palpitations and chest tightness)	Normal	-	Valsalva, Carotid massage, and Adenosine	-	Normal
2005/Yagi, Syusuke. [8]	PVCs; VT; RBBB	34 Y	33 Y	Man	1 M	-	RCA-stenosis; LAD-occlusion of GCA	2,3/-	LV dysfunction	-	Beta-blocker	CABG; Radio-frequency catheter ablation; ICD	-
	PVCs; NSVT	32 Y	-	Man	-	-	RCA-stenosis	2/Yes	LV dysfunction	-	Beta-blocker	ICD	Normal
2008/Sumitomo, Naokata. [4]	PVCs	9.9 Y	3.3 Y	Man	-	-	LCA-aneurysm	-	-	-	-	-	-
	SNDF	9.9 Y	1.6 Y	Man	-	-	LCA-LCX-RCA-regression of CA	-	-	-	-	-	-
	VF	11.9 Y	1.6 Y	Man	-	-	RCA-dilatation	-	-	-	-	-	-
	SNDF	13 Y	2.3 Y	Man	-	-	LCA-stenosis; RCA-obstruction of CA	-	-	-	-	-	-
	VT	13.9 Y	0.4 Y	Female	-	-	LCA-regression of CA; RCA-CA	-	-	-	-	-	-
	NSAF	19.9 Y	2 Y	Man	-	-	LCA-regression of CA; RCA-CA	-	-	-	-	-	-
2010/Uusimaa, P. [12]	Mobitz type II-AVB	20.8 Y	10.9 Y	Female	-	-	LCA-calcification of CA	-	-	-	-	-	-
	AT	28 Y	27.7 Y	Man	-	-	Aneurysmal dilatation of the proximal segments of all coronary arteries.	-	LV dysfunction	-	-	Radiofrequency catheter ablation	Normal
2016/Chou, Chia-Pei. [3]	PSVT	4M10d	16d	Man	2d	No	Normal	-	-	1	Adenosine, Amiodarone loading and Propranolol	-	Normal
2018/Hu, Fan [10]	PVCs; VT	1 Y	2d	Female	5d	Yes	Normal	Normal	Normal	1	Mexiletine	-	Normal

Table 1 (continued)

Year/Author	Types of arrhythmia	Age (Years)	KD onset (Years)	Gender	Duration of fever	Incomplete Kawasaki Disease	Coronary Artery condition	ECG Evaluation and Symptom Assessment of MI	Cardiac function status	Number of IVIG/ Steroid hormone	Antiarrhythmic therapy	Surgical treatment	Treatment outcome
2018/Komaki, Hisaaki. [7]	VT	26Y	22Y	Man	-	-	LAD-CA, LCX-occlusion of CA	-	LV dysfunction	-	-	Radiofrequency catheter ablation; ICD	Normal
2019/Nakagawa, Naomi [13]	Sinus arrest; AF	5.5Y	10d	Man	6d	No	Normal	Normal	Normal	2/Yes	Flecainide	-	Normal

Y, Year; W, Week; M, Month; d, day; FPVCs, Frequent Premature Ventricular Contractions; AT, left atrial tachycardia; VT, Ventricular Tachycardia; NSVT, Non-sustained VT; VF, ventricular fibrillation; RBBB, Right bundle-branch block; AVB, Atrioventricular block; NSAF, Nonsustained Atrial Fibrillation; SNDF, Sinus node dysfunction; PSVT, Paroxysmal supraventricular tachycardia; MI, Myocardial ischemia; RCA, Right coronary artery; LAD, left anterior descending artery; GCA, giant coronary aneurysm; LV, Left Ventricle; ECG, Electrocardiogram; 1 = ST segment depression; 2 = Q wave; 3 = QS pattern; CABG, Coronary artery bypass grafting; ICD, Implantable defibrillator

blood supply (especially when coronary artery thrombosis occurs) and increase the risk of long-term myocardial fibrosis and myocardial infarction. As a result, patients with persistent CAAs might also be prone to develop arrhythmias during the chronic phase of KD. Previous studies suggested that patients with CAA z-scores ≥ 5 were prone to exercise stress test-induced arrhythmias [20]. This also emphasized the importance of strengthening the management of chronic CAAs and evaluating myocardial ischemia in KD patients with concurrent medium to large coronary artery aneurysms.

In summary, arrhythmias were rare in patients with KD, however, this condition might occur during the acute, subacute, and chronic stages of KD. Whether it's conduction impairment and cell compression during the acute phase or myocardial ischemia, infarction, and scar formation in the chronic phase, we speculated the inflammatory response during the acute phase serves as an initiating factor for arrhythmia development. Therefore, it is crucial to timely control the inflammatory reaction during the acute phase to prevent subsequent cascading reactions. Additionally, for KD patients with concurrent CAAs in the chronic phase, improving the management of CAAs and myocardial ischemia can reduce the occurrence of long-term cardiovascular events in these patients, such as arrhythmias.

Conclusions

The occurrence of arrhythmias as a complication of KD was exceedingly rare. This phenomenon may be related to myocarditis and myocardial ischemia caused by scar formation, or excessive inflammatory factors caused by the release of immune damage to the conduction system and other factors. Strengthening the early identification and management of complications in patients with KD and personalized follow-up strategies for high-risk children during the chronic phase can enhance patients' prognosis. Furthermore, besides evaluating coronary arteries, it might also be essential to evaluate the cardiac conduction system by ECG for individuals with a history of KD and personalize follow-up strategies.

Abbreviations

- KD Kawasaki disease
- IMG Intravenous Immunoglobulin
- SVT Supraventricular tachycardia
- WBC White blood cells
- CRP C-reactive protein
- ESR Erythrocyte sedimentation rate
- ALT Alanine Aminotransferase
- AST Aspartate, Aminotransferase
- MIS-C Multisystem inflammatory syndrome in children
- ECG Electrocardiogram
- CALs Coronary Artery Lesions
- CAAs Coronary artery aneurysms

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Author contributions

Nanjun Zhang drafted the manuscript and approved the final submission. Bowen Li and Yu Yan contributed to the literature collection, provided Table, and approved the final manuscript. Shuran Shao, Yimin Hua, and Hongyu Duan contributed to the study design and approved the final manuscript. Hongyu Duan and Xiaoliang Liu provided the Figure, and funding support, and also approved the final manuscript. All abstracts were reviewed by Nanjun Zhang, Bowen Li, and Yu Yan, and checked by Kaiyu Zhou, Chuan Wang, and Xiaoliang Liu. Chuan Wang also provided financial support and approved the final manuscript.

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Data availability

The original contributions presented in the study are included in the article, further inquiries can be directed to the corresponding authors.

Declarations

Competing interests

All authors have no conflict of interest or any grants to declare.

Ethics approval, consent to participate and consent for publication

Informed consent has been obtained from the guardian, and the guardian has agreed to the publication of all images, clinical data, and other data included in the manuscript.

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