

Brief Report

Cite this article: Akagawa D, Motoki N, Hachiya A, Akazawa Y. (2019) Transient Q-waves in an infant with asymptomatic myocardial infarction due to Kawasaki disease. *Cardiology in the Young* 29: 231–234. doi: 10.1017/S1047951118001919

Received: 10 May 2018
Revised: 13 September 2018
Accepted: 30 September 2018
First published online: 6 December 2018

Key words:

Kawasaki disease; myocardial infarction; transient Q-wave; supergiant aneurysm

Author for correspondence:

Noriko Motoki, MD, PhD, Department of Pediatrics, Shinshu University School of Medicine, Asahi 3-1-1, Matsumoto 390-8621, Japan. Tel: 81-263-37-2642; E-mail: nmotoki@shinshu-u.ac.jp

Transient Q-waves in an infant with asymptomatic myocardial infarction due to Kawasaki disease

Daisuke Akagawa, Noriko Motoki, Akira Hachiya and Yohei Akazawa

Department of Pediatrics, Shinshu University School of Medicine, Asahi 3-1-1, Matsumoto, Japan

Abstract

Giant coronary artery aneurysms are a complication of Kawasaki disease and can be fatal if associated with thrombosis. We describe the clinical outcome of a boy with Kawasaki disease who exhibited “supergiant” coronary artery aneurysms at the age of 14 months and, despite treatment with anticoagulant and antiplatelet medication, developed a left coronary artery thrombosis and presented following a myocardial infarction at 2 years old. Although his symptoms were minimal, the myocardial infarction was identified by abnormal Q-waves and giant negative T-waves in precordial leads of routine electrocardiography. Intensive anticoagulant therapy combining heparin injections and high-dose warfarin was successful. The abnormal Q-waves and negative T-waves had completely disappeared 2 weeks later, likely in association with confirmed reperfusion. On the basis of prompt identification of abnormal Q-waves by electrocardiography, the patient could avoid thrombolytic therapy and catheter or surgical intervention.

Kawasaki disease is an acute febrile childhood illness that is classified as a systemic vasculitis syndrome. Together with aspirin, high-dose intravenous immunoglobulin therapy is generally effective to resolve inflammation in Kawasaki disease and reduce the occurrence of coronary artery abnormalities.^{1–3} Although “giant” and “supergiant” coronary aneurysms of ≥ 8 and ≥ 10 mm, respectively, are uncommon, Kawasaki disease patients may be predisposed to acute thrombotic occlusion leading to acute myocardial infarction and mortality.^{4,5} Patients with giant coronary artery aneurysms therefore need lifelong anticoagulant therapy. In a recent Japanese study of 245 patients, 15 deaths were caused by Kawasaki disease, among which 13 were directly due to myocardial infarction.⁶

We here report the clinical findings of an infant boy who suffered a myocardial infarction caused by a thrombus in a “supergiant” aneurysm. Since his symptoms were mild, routine electrocardiography played a crucial role in myocardial infarction identification. Anticoagulant treatment including combined heparin injection and high-dose warfarin was effective.

Case report

A 14-month-old Japanese boy with Kawasaki disease was admitted to our hospital for suspected coronary artery aneurysms. Initial treatment with prednisolone 2 mg/kg/day, aspirin 30 mg/kg/day, and intravenous immunoglobulin therapy 2 g/kg was administered on day 4, as he was predicted to be a non-responder to intravenous immunoglobulin therapy by conventional risk scores. On day 8, he was unresponsive to the second round of intravenous immunoglobulin and soon after developed coronary aneurysms of 4.6 mm in diameter in the proximal left anterior descending coronary artery and 4.7 mm in diameter in the right coronary artery. On day 12, he commenced infliximab 5 mg/kg through intravenous injection. Although he became afebrile and other Kawasaki disease symptoms disappeared, he eventually exhibited “supergiant” coronary aneurysms that were 8.6 mm in diameter in the proximal left anterior descending coronary artery and 12.6 mm in diameter in the right coronary artery (Fig 1a and b) that required urgent anticoagulant therapy with warfarin. At discharge, he was prescribed aspirin, warfarin, and dipyridamole. Propranolol and enalapril maleate were also added to control heart rate and blood pressure and prevent aneurysm rupture. According to the guidelines for Kawasaki disease management in Japan, warfarin intake was adjusted to maintain a therapeutic range of prothrombin time based on the international normalised ratio of 2.0:2.5.⁷

At the age of 2 years, the patient experienced upper abdominal pain and sudden vomiting but recovered spontaneously in several hours. One week later, he visited our hospital for

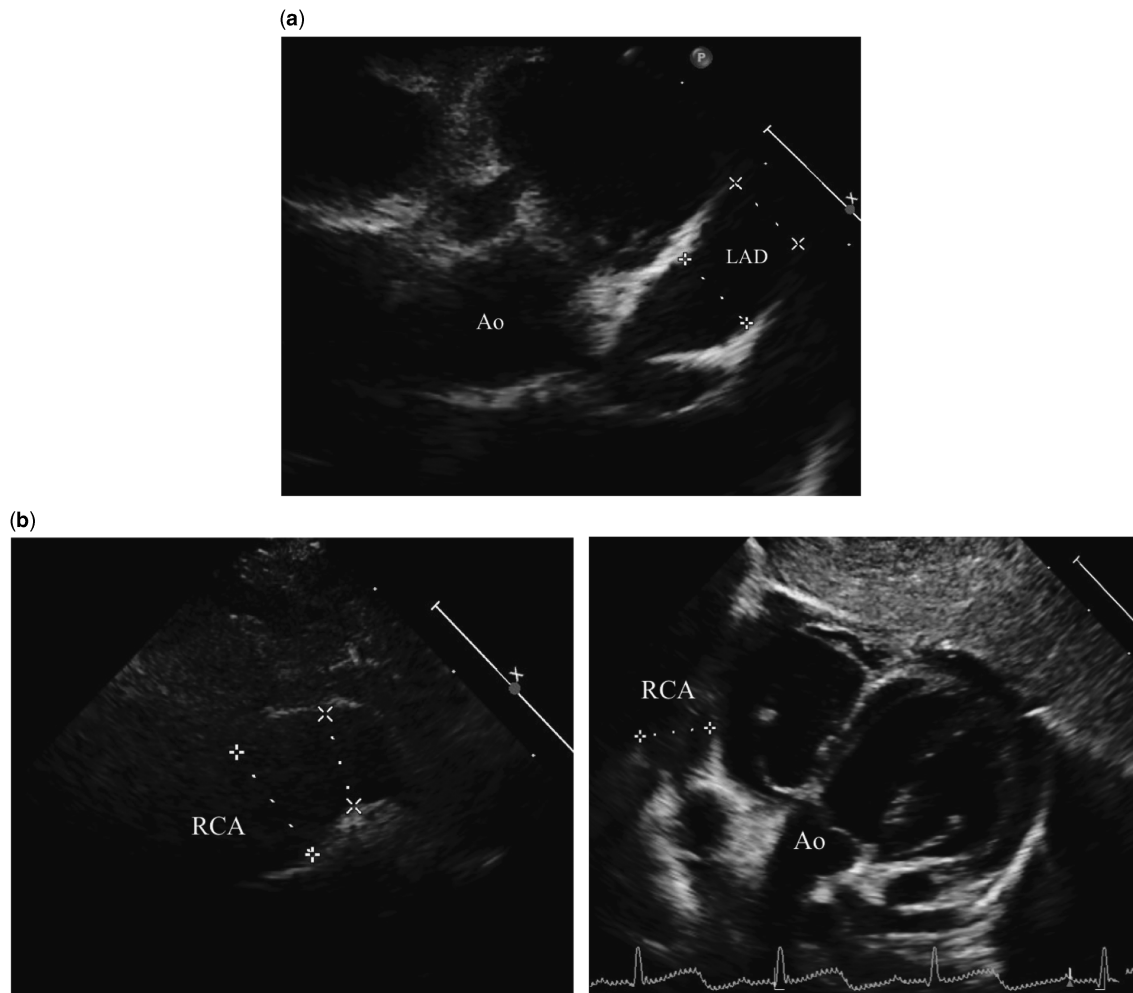


Figure 1. Echocardiography at the acute phase of Kawasaki disease. Echocardiography revealed diffuse dilatation and “supergiant” aneurysms in the left anterior descending coronary artery (a) and right coronary artery (b) on the 20th day of acute Kawasaki disease. The left anterior descending coronary artery was 8.6 mm and the right coronary artery was 12.6 mm in maximum diameter.

scheduled coronary angiography and showed no obvious symptoms suggestive of myocardial infarction; however, standard 12-lead electrocardiography revealed abnormal Q-waves in V1-3 and giant negative T-waves in V2-4 (Fig 2a). Laboratory data showed brain natriuretic peptide of 188.9 pg/ml, the normal range being <20 pg/ml, prothrombin time based on the international normalised ratio of 2.17, and normal cardiac enzymes. Echocardiography also uncovered a large thrombus of 7.9 × 17.3 mm in the left anterior descending coronary artery aneurysm (Fig 2b) without any apparent dyskinesia of the left ventricular wall. Left ventricular systolic and diastolic functions were preserved. Subsequent 99m-technetium myocardial perfusion scintigraphy revealed decreased myocardial blood flow in the anterior wall both at rest and during adenosine stress. He was diagnosed of having suffered an anterior myocardial infarction without apparent symptoms or cardiac dysfunction. We decided against thrombolytic therapy, such as urokinase or tissue-plasminogen activator, since more than 1 week had passed from the myocardial infarction, indicating that his minor gastrointestinal symptoms had been caused by myocardial infarction and his cardiac condition had been preserved. Anticoagulant therapy with combination of heparin injection and warfarin was initiated. Warfarin dose was adjusted to maintain a prothrombin time based on the

international normalised ratio between 2.5 and 3.0. One week later, angiography of the left coronary artery confirmed a thrombus in the aneurysm of the left anterior descending coronary artery and total occlusion of the distal left anterior descending coronary artery (Fig 2c). Furthermore, angiographic imaging of the right coronary artery disclosed collateral circulation from the right coronary artery to the distal left anterior descending coronary artery. Intensive anticoagulant therapy with heparin and warfarin was continued to resolve the remaining thrombus. Carvedilol instead of propranolol was administered to prevent myocardial fibrosis.

Daily electrocardiography measurements indicated shrinking of the thrombus in the left anterior descending coronary artery. It was also notable that the abnormal Q-waves and negative T-waves in the precordial leads had completely disappeared in 2 weeks (Fig 3a). Owing to the success of the aggressive anticoagulant treatment, heparin was discontinued and high-dose warfarin was maintained by prothrombin time based on the international normalised ratio 2.5:3.0. Three months after the myocardial infarction, follow-up coronary angiography revealed that the thrombus had almost completely disappeared and that perfusion of the left anterior descending coronary artery was restored (Fig 3b). Finally, his myocardial infarction because of a

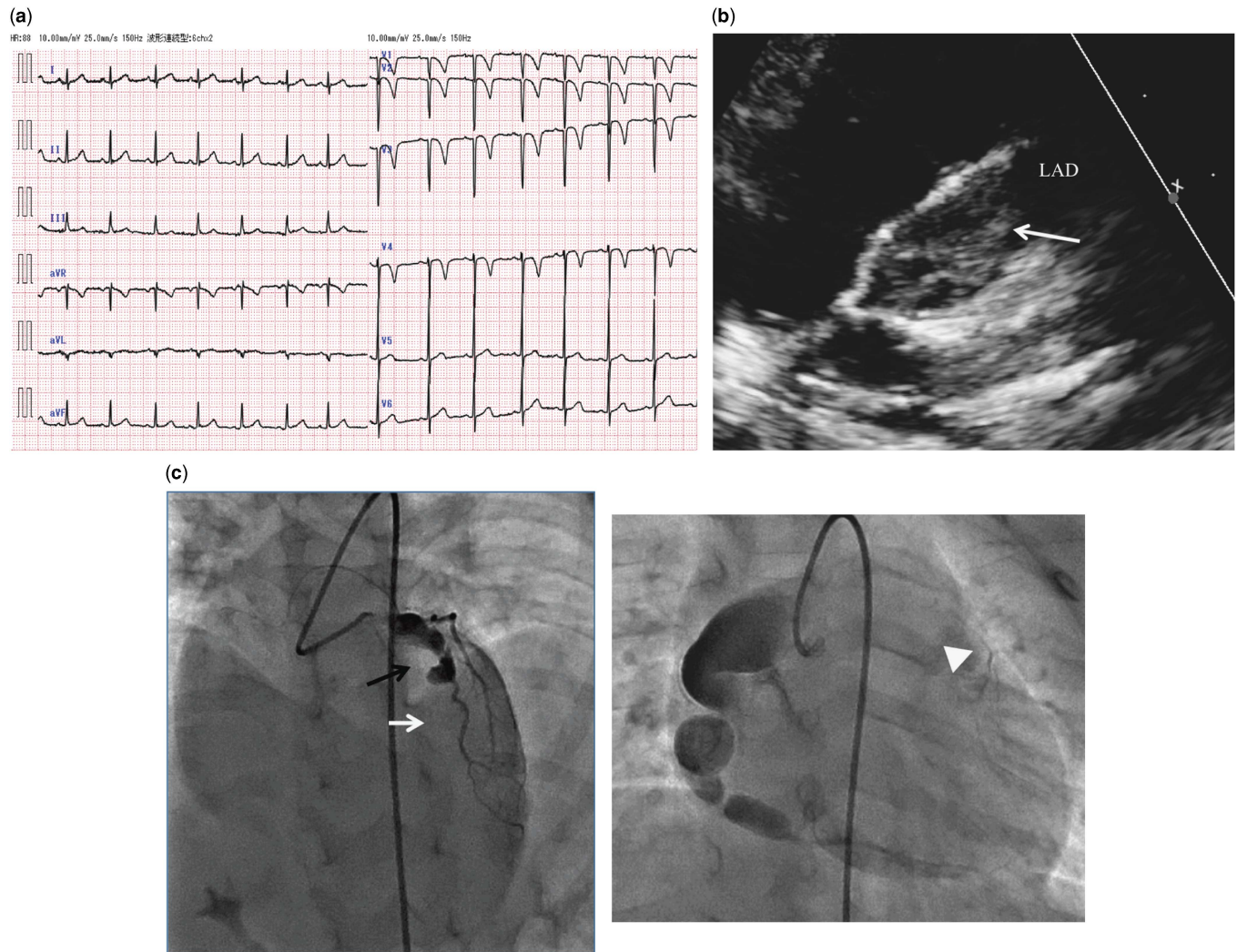


Figure 2. Cardiovascular examination on admission for myocardial infarction due to a thrombus in a coronary aneurysm. (a) Standard 12-lead electrocardiography at scheduled coronary angiography. Abnormal Q waves in V1-3 and giant negative T waves in V2-4 were evident. (b) Echocardiography revealed a large thrombus (white arrow; 7.9 × 17.3 mm) in the giant aneurysm of the left anterior descending coronary artery. (c) Coronary angiography a week after starting anticoagulation therapy disclosed a large thrombus in the left anterior descending coronary artery aneurysm (black arrow) and total occlusion of the distal left anterior descending coronary artery (white arrow). Collateral circulation had extended from the right coronary artery to the distal left anterior descending coronary artery (white arrowhead).

thrombus in a giant aneurysm was successfully managed without any apparent ischaemic signs or cardiac dysfunction, although he ultimately required continuous therapy with warfarin.

Discussion

Giant aneurysms secondary to Kawasaki disease increase the risk of sudden cardiac death because of myocardial infarction caused by a massive thrombus.⁸ Myocardial infarction occurs most commonly in the first year after the disease onset.⁶ However, its diagnostic difficulty stems from the rarity of acute myocardial infarction in young patients and is compounded by its diverse clinical features and the inability of infants to describe their symptoms. Consequently, the diagnosis of acute myocardial infarction is likely to be delayed in infants and young children, resulting in a diminished chance of timely reperfusion.⁶ Because most cases of acute myocardial infarction involve Kawasaki disease patients with giant aneurysms, close follow-up is needed, particularly within the first year after Kawasaki disease diagnosis.

In the present case, changes in electrocardiography findings helped diagnose asymptomatic myocardial infarction and enable successful reperfusion. In the absence of QRS confounders, abnormal Q-waves are usually diagnostic of myocardial necrosis. However, they may regress or disappear over time in as many as 25–63% of patients with a history of Q-wave myocardial infarction.^{9,10} Some investigators have reported that Q-wave appearance after myocardial infarction depends on infarct size.^{11,12} Patients with myocardial infarction showed Q-wave regression trend towards a smaller amount of necrotic myocardium and a significantly larger amount of stunned myocardium.¹² Transient ischaemic Q-waves may appear acutely or be present chronically with the potential of disappearing when coronary perfusion is restored. Oedema and inflammation may also play a part in rendering the myocardium electrically inert, and their disappearance may explain a loss of Q-waves.¹³ Electrical stunning, which presents as transient abnormal Q-waves, may be associated with the myocardial “stunning” following episodes of acute transient coronary ischaemia. In our case, at least 1 week had

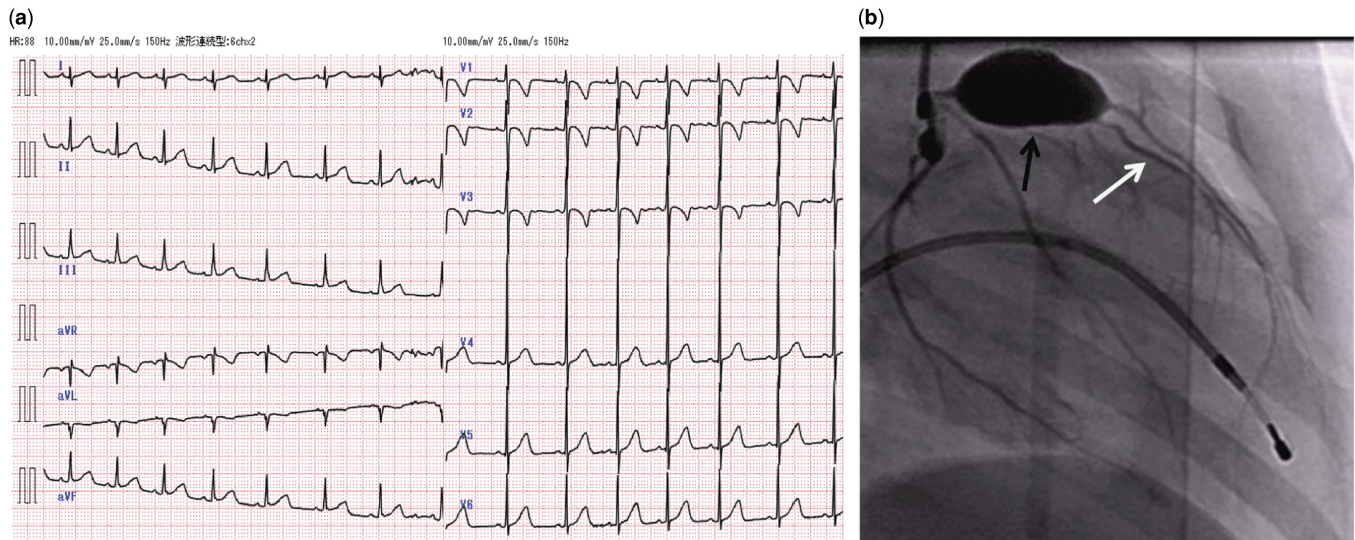


Figure 3. Cardiovascular examination after anticoagulant therapy. (a) Standard 12-lead electrocardiography 2 weeks after starting aggressive anticoagulant therapy. The abnormal Q waves in V1-3 and negative T waves in V4 had disappeared. (b) Coronary angiography 3 months after the myocardial infarction. The thrombus had shrunk (black arrow), and perfusion of the distal left anterior descending coronary artery was restored (white arrow).

passed from the suspected asymptomatic myocardial infarction. He could have had a stunned myocardium due to ischaemia rather than an infarction, and therefore the abnormal Q-waves disappeared by reperfusion using anticoagulant therapy. However, the infarct size was small and it was thought that myocardial viability remained preserved in the stunned myocardium.

Conclusion

We described in this study a Kawasaki disease patient with “supergiant” coronary aneurysms, who suffered an asymptomatic myocardial infarction because of a large thrombus in the left anterior descending coronary artery. Abnormal Q-waves in electrocardiography played a key role in diagnosing the disease, as well as in the early monitoring of anticoagulant therapy progress. Electrocardiography is a simple, non-invasive, widely available tool. Combination heparin and warfarin treatment enabled successful reperfusion without additional catheter or surgical intervention.

Acknowledgements. The authors sincerely thank Dr Ryuji Fukazawa at Nippon Medical School for his suggestions regarding the present patient and for providing clinical data. We also thank Dr Takashi Shimizu at Saku Central Hospital for his routine medical care, as well as Yūka Miyajima and Trevor Ralph for their language assistance.

Financial Support. No funding was received for this study. The authors have no financial relationships relevant to this article to disclose.

Conflicts of Interest. The authors have no conflicts of interest to disclose.

References

1. Newburger JW, Takahashi M, Burns JC. Kawasaki disease. *J Am Coll Cardiol* 2016; 67: 1738–1749.
2. Oates-Whitehead RM, Baumer JH, Haines L, et al. Intravenous immunoglobulin for the treatment of Kawasaki disease in children. *Cochrane Database Syst Rev* 2003; (4): CD004000.
3. Newburger JW, Takahashi M, Beiser AS, et al. A single intravenous infusion of gamma globulin as compared with four infusions in the treatment of acute Kawasaki syndrome. *N Engl J Med* 1991; 324: 1633–1639.
4. Elkici F, Varan B, Kocabaş A, Erdoğan İ, Eminoğlu S, Aktaş D. Multiple giant aneurysms and stenosis of the coronary and systemic arteries in an infant with Kawasaki disease at the early stage of convalescent period. *Echocardiography*. 2014; 31: E147–E150.
5. Motozawa Y, Uozumi H, Maemura S, et al. Acute myocardial infarction that result from poor adherence to medical treatment for giant coronary aneurysm. *Int Heart J* 2015; 56: 551–554.
6. Tsuda E, Hamaoka K, Suzuki H, et al. A survey of the 3-decade outcome for patient with giant aneurysms caused by Kawasaki disease. *Am Heart J* 2014; 167: 249–258.
7. JCS Joint Working Group. Guidelines for diagnosis and management of cardiovascular sequelae in Kawasaki disease (JCS 2013). *Circ J* 2014; 78: 2521–2562.
8. Kato H, Sugimura T, Akagi T, et al. Long-term consequences of Kawasaki disease. A 10- to 21-year follow-up study of 594 patients. *Circulation* 1996; 94: 1379–1385.
9. Yasuda M, Iida H, Itagane H, et al. Significance of Q wave disappearance in the chronic phase following transmural acute myocardial infarction. *Jpn Circ J* 1990; 54: 1517–1524.
10. Voon WC, Chen YW, Hsu CC, Lai WT, Sheu SH. Q-wave regression after acute myocardial infarction assessed by TI-201 myocardial perfusion SPECT. *J Nucl Cardiol* 2004; 11: 165–170.
11. Delewi R, Ijff G, van de Hoef TP, et al. Pathological Q wave in myocardial infarction in patients treated by primary PCI. *JACC Cardiovasc Imaging* 2013; 6: 324–331.
12. Nagase K, Tamura A, Mikuriya Y, Nasu M. Significance of Q-wave regression after anterior wall acute myocardial infarction. *Eur Heart J* 1998; 19: 742–746.
13. Barold SS, Falkoff MD, Ong LS, Heinle RA. Significance of transient electrocardiographic Q waves in coronary artery disease. *Cardiol Clin*. 1987; 5: 367–380.