

Document heading

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## Acute myocardial infarction in a 35-year-old man with coronary artery aneurysm most probably caused by Kawasaki disease

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### PEER REVIEW

#### Peer reviewer

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#### Comments

In this case report, the clinical course of the disease has been well illustrated. The contents seem rather concise and thorough, and the discussion is thoughtfully relevant. It is also well drafted, and the scientific picture regarding the clinical significance of late onset cardiac complications of Kawasaki disease is clearly described. Details on Page S51

### ABSTRACT

We present a 35-year-old man with history of Kawasaki disease who referred with myocardial infarction, and angiography, revealing aneurysm of left main and left anterior descending coronary arteries. The patient underwent percutaneous coronary intervention and thrombectomy and was discharged after 6 d. Coronary artery sequels of Kawasaki disease should be considered as one of the underlying causes of acute myocardial infarction in young adults.

### KEYWORDS

Kawasaki disease, Myocardial infarction, Coronary aneurysm, Angiography

## 1. Introduction

A 35-year-old man with history of Kawasaki disease at the age of five years presented to the emergency department 2 h after sudden onset of chest pain. On physical examination, his blood pressure was 130/94 mmHg and heart rate was 62 beats/min. Chest and heart examinations were unremarkable. A 12-lead electrocardiogram (ECG) showed sinus rhythm with ST elevations in leads V<sub>1</sub>–V<sub>5</sub>, I, aVL and

ST depression in leads II, III, aVF and V<sub>6</sub>. In addition, raised cardiac enzymes (creatinine kinase myocardial bound/creatinine kinase: 198/641 U/L and elevated troponin-I level: 21 ng/mL) were recognized.

Also, echocardiography revealed ejection fraction of 30%, anterior and antro-septal wall akinesia, inferoseptal wall severe hypokinesia and smoky pattern in left ventricular apex. Results of laboratory assessments included normal levels of electrolytes and glucose. The blood urea nitrogen

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level was 15 mg per deciliter and creatinine level was 1.1 mg per deciliter. The white blood cell count was 19600 per cubic milliliter, with 61% neutrophils. The hematocrite was 49.5% and the platelet count was 309000 per cubic milliliter. The prothrombin time and partial-thromboplastin time were also normal.

Emergency coronary angiography was performed which revealed aneurysm of the distal portion of left main and proximal part of left anterior descending coronary arteries, and occluded left anterior descending artery after aneurysm without distal run off. Right coronary artery showed narrowing at mid portion (Figure 1). Left ventriculography was not performed due to high left ventricular end diastolic pressure (40 mmHg). Accordingly, 325 mg aspirin and 600 mg clopidogrel were loaded and patient was scheduled for primary percutaneous coronary intervention in the setting of acute ST-elevation myocardial infarction. After engagement with a 6 French left Judkins guiding catheter and wiring with BMW wire, thrombectomy was performed with Diver-C aspiration catheter and two boluses of intracoronary eptifibatid were immediately injected and continued intravenously at 2 µg/kg/min for 18 h. Then a 3.5\*12 mm integrity stent was deployed on left anterior descending and grade III TIMI flow was established.



**Figure 1.** A: RAO caudal view; B: LAO caudal view of left coronary angiogram showing the aneurysm of distal portion of left main coronary and proximal part of left anterior descending coronary arteries; C: LAO view of right coronary artery showing narrowing at mid portion of right coronary artery; D: RAO cranial view of left coronary artery after thrombectomy and PCI showing successful revascularization.

Twelve-lead ECG was performed 7 d after percutaneous coronary angioplasty which showed sinus rhythm with Q waves in  $V_1$ – $V_5$ , aVL and ST depression in II, III, and aVF (Figure 2). The patient was discharged home 6 d later on dual anti-platelet therapy (aspirin 81 mg and clopidogrel 75 mg) and warfarin (5 mg), aiming to attain a target international normalized ratio of 2–3.

## 2. Discussion

Kawasaki disease is an acute vasculitis of undefined etiology that principally affects children <5 years of age and is the foremost cause of acquired pediatric heart disease

in North America and Japan[1]. Cardiac sequels, such as coronary artery aneurysms and myocardial infarction, are the main causes of morbidity and mortality related to Kawasaki disease[2]. Although Kawasaki disease is usually a self-limiting disorder, 15–25% of affected children may have significant cardiovascular sequels, presenting acutely or late in adulthood[3]. The most known late complication is the persistence of coronary artery aneurysms which may lead to myocardial ischemia and even infarction[4]. The natural history of coronary artery aneurysms in patients with Kawasaki disease may consist of any of the followings: regression, rupture, thrombosis and recanalization, stenosis or myocardial infarction[5].

Acute myocardial infarction in young adults is very infrequent[6]. Age above 40 years, smoking, diabetes, obesity, hypertension, male gender, dyslipidemia and family history of atherosclerosis are the known risk factors for coronary artery disease[7]. In our patient, aside from male gender as a risk factor, the most probable cause of myocardial infarction was the coronary artery aneurysm due to Kawasaki disease. Patients with a history of this disease are at augmented risk for premature atherosclerosis, which consecutively sets them at risk for young-onset acute myocardial infarction.

In Kawasaki disease, aneurysms develop principally in the proximal part of coronary arteries. Size of the aneurysm is the most crucial predictor of myocardial infarction. Giant aneurysms have poor prospects, do not regress and usually result in ischemic heart disease[8]. Our patient had not been treated in childhood and was not being monitored thereafter; so it seems that the aneurysm may have developed as a cardiovascular consequence of Kawasaki disease. We conclude that cardiologists should be familiar with coronary artery sequels of Kawasaki disease and consider it as one of the underlying causes of acute myocardial infarction in young adults.

## Conflict of interest statement

We declare that we have no conflict of interest.

## Acknowledgements

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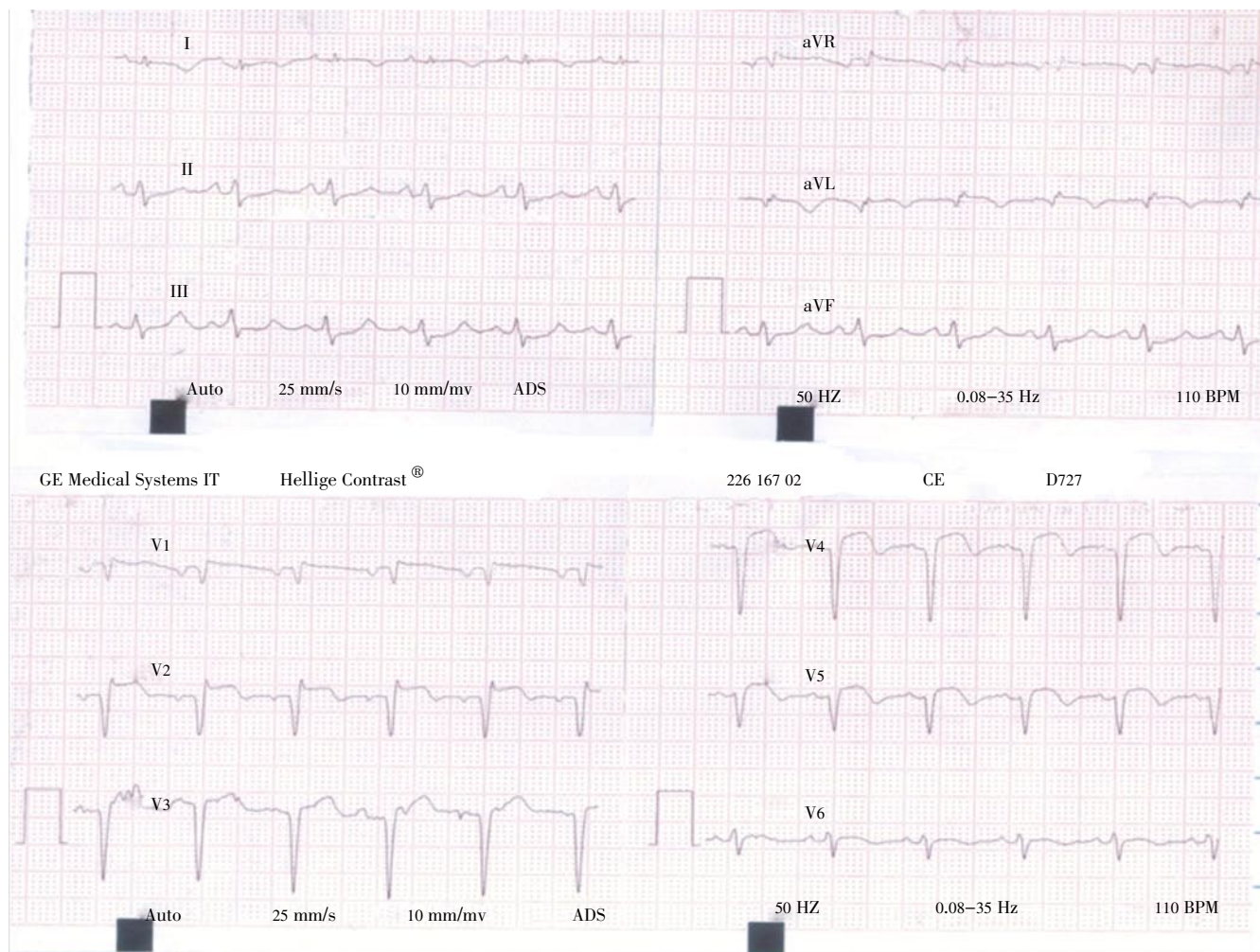
## Comments

### Background

Kawasaki disease is an acute, self-limited vasculitis that is characterized by the subsequent development of arterial aneurysms in certain circulations, in particular, coronary system. The disease is mostly self-limited, but possessing cardiac sequel that may result in the incidence of acute coronary syndrome and rarely, sudden death in young adults.

### Research frontiers

Although the late cardiac sequel of Kawasaki disease have been previously established, accurate data on the possible



**Figure 2.** ECG of the patient 7 d later, which shows Q waves in the precordial leads V1–V5, AVL and ST depression in leads II, III, and aVF leads.

role of close patient monitoring and the administered therapies such as human IVIG remains unidentified.

#### Related reports

With regard to the reports indicating the role of Kawasaki disease late sequela, it seems that the frequency of such history among patients who presented to the ED with acute coronary syndrome is quite rare.

#### Innovations and breakthroughs

This report represents a quite rare cause of acute myocardial infarction in an adult. Hence, it is necessary for cardiologist to keep in mind that Kawasaki disease might be the underlying reason for the development of coronary artery lesions.

#### Applications

Physicians should be aware of the differential diagnosis of the underlying causes of acute myocardial infarction in young adults in the absence of other risk factors.

#### Peer review

In this case report, the clinical course of the disease has been well illustrated. The contents seem rather concise and thorough, and the discussion is thoughtfully relevant. It is also well drafted, and the scientific picture regarding the clinical significance of late onset cardiac complications of Kawasaki disease is clearly described.

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