

IMAGING VIGNETTE

BEGINNER

CLINICAL VIGNETTE

# A Case of Father–Son Juvenile Acute Myocardial Infarction



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## ABSTRACT

Coronary artery ectasia (CAE) is a cause of juvenile myocardial infarction. The causes of CAE include arteriosclerosis, vasculitis such as Kawasaki disease, and genetic contribution. There are few reports about familial aggregation of CAE-related juvenile myocardial infarction. We report an unusual case of father-son juvenile myocardial infarction owing to CAE. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2023;18:101910) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

## CASE PRESENTATION

A 17-year-old boy visited the emergency department with chest discomfort. The vital signs were within normal limits. Although an electrocardiogram revealed no significant ST-segment changes ([Supplemental Figure 1](#)) and transthoracic echocardiography showed normal systolic function, the troponin-I levels were elevated (troponin-I, 3987 pg/mL; reference value, <46.46 pg/mL). He had been treated for Kawasaki disease (KD) at 3 years of age. Considering the possibility of acute myocardial infarction (AMI), we urgently performed coronary angiography (CAG). There were no coronary aneurysms in the coronary arteries; however, the right coronary artery was tortuous, ectatic, and occluded at the distal site ([Figure 1A](#), [Video 1](#)). Restoration of coronary flow was achieved following percutaneous coronary intervention ([Figure 1B](#), [Video 2](#)). Intravascular ultrasound pullback showed mixed images of intimal thickening, atherosclerotic plaque, and thrombus formation at the proximal site of the occluded lesion ([Video 3](#)). The creatinine kinase level rose to 1,620 IU/L (reference value, 57-284 IU/L). Left ventricular wall motion and ejection fraction was almost preserved. He was discharged 2 weeks after rehabilitation.

Because this was a case of juvenile-onset AMI, we investigated the predisposing factors. The triglyceride and high-density lipoprotein cholesterol levels in the fasting state were 308 mg/dL and 19 mg/dL, respectively. Antiplatelet therapy, lipid-lowering therapy, and lifestyle interventions were initiated for secondary prevention. The patient had a strong family history of AMI. The patient's father had an AMI at 25 years of age. CAG showed that the right coronary artery was ectatic and occluded at the distal site, similar to the right coronary artery in his son ([Figure 1C](#), [Video 4](#)). Successful recanalization was achieved by thrombolytic therapy ([Figure 1D](#), [Video 5](#)). Notably, the patient's grandfather also had a history of an AMI at 55 years of age. An ectatic coronary artery was confirmed by CAG and documented in the medical record.

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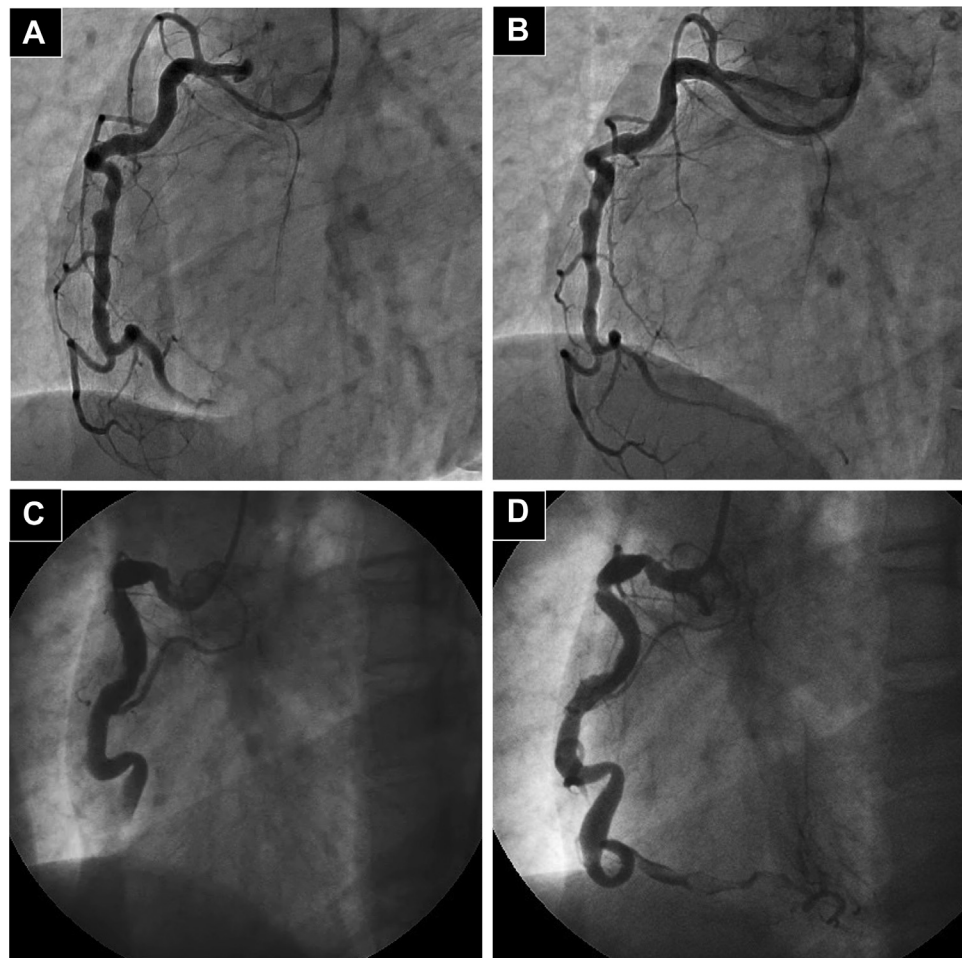
The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the [Author Center](#).

Manuscript received May 2, 2023; accepted May 12, 2023.

**ABBREVIATIONS  
AND ACRONYMS****AMI** = acute myocardial  
infarction**CAE** = coronary artery ectasia**CAG** = coronary angiography**KD** = Kawasaki disease**DISCUSSION**

CAE is a rare finding on CAG. The prevalence of CAE is 0.9% to 5.3%. The causes of CAE include arteriosclerosis, vessel wall injury, vasculitis such as KD, and genetic contribution.<sup>1</sup> CAE owing to KD is among the causes of juvenile myocardial infarction. Several studies have shown that CAE is associated with worse clinical outcomes in patients with an AMI. Dilatation of the coronary arteries disturbs coronary flow, increases blood viscosity, and activates coagulation, which together cause thrombus formation and peripheral embolism. Because of a lack of high-quality evidence, initiation of antithrombotic therapy is at the physician's discretion.<sup>2</sup> Our case suggested familial aggregation of AMI owing to CAE over 3 generations. Inherited predisposition is considered to play a major role in juvenile myocardial infarction, but acquired factors cannot be denied. Familial aggregation in KD has been reported previously. Variants in some genes are associated with an increased risk of aneurysm formation.<sup>3</sup> A definitive diagnosis of KD was not confirmed in the patient's father and grandfather, and genetic factors underlying CAE were not established.

In conclusion, confirmation of the medical and detailed family histories is required in cases of juvenile myocardial infarction. CAE noted during CAG should receive more attention from physicians.

**FIGURE 1** Coronary Angiography of the Son and Father

Right coronary angiography of the son (**A**) before recanalization and (**B**) after recanalization. Right coronary angiography of the father (**C**) before recanalization and (**D**) after recanalization. The right coronary arteries were generally tortuous and ectasic and occluded at the distal site.

## FUNDING SUPPORT AND AUTHOR DISCLOSURES

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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**KEY WORDS** coronary artery ectasia, familial aggregation, juvenile myocardial infarction

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 **APPENDIX** For a supplemental figure and videos, please see the online version of this paper.