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CASE REPORT

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Coronary Artery Aneurysms Due to Kawasaki Disease – a Rare Cause of Acute Myocardial Infarction

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ABSTRACT

Kawasaki disease, also known as Kawasaki syndrome or mucocutaneous lymph node syndrome, is a pathology that causes inflammation in the walls of medium sized arteries causing symptoms such as fever, lymphadenopathy, rash, and erythema of eyes, lips, nose, palms and feet. The cause is unknown, although clinical features strongly suggest an infectious etiology. We present the case of a 53-year-old woman, known with Kawasaki disease since childhood, with different associated pathologies, that presented with acute inferior ST elevation myocardial infarction.

Keywords: Kawasaki disease, acute myocardial infarction, coronary artery aneurysm

INTRODUCTION

Kawasaki disease, also known as Kawasaki syndrome or mucocutaneous lymph node syndrome is a pathology that causes inflammation in the walls of medium sized arteries, occurring especially in children under 5 years, predominantly in boys rather than girls. The disease has winter-spring seasonality and its symptoms consist in fever, lymphadenopathy, rash, and erythema of eyes, lips, nose, palms and feet. It was first described by Tomisaku Kawasaki in Japan in 1967, and appears in children of all races.[1] The cause is unknown, although some studies present the hypothesis that a viral infection activates some autoimmunological triggers in those patients with genetic predisposition. Kawasaki disease is not curable, but the patients can recover completely after the rapid onset of treatment.[2]

The most important arteries that are affected are the ones that irrigate the heart muscle, the coronary arteries, leading to different cardiovascular complications, such as acute coronary syndromes in children or even a dramatic sudden cardiac death.[3] It can also affect the cardiac tissue, causing myocardi-

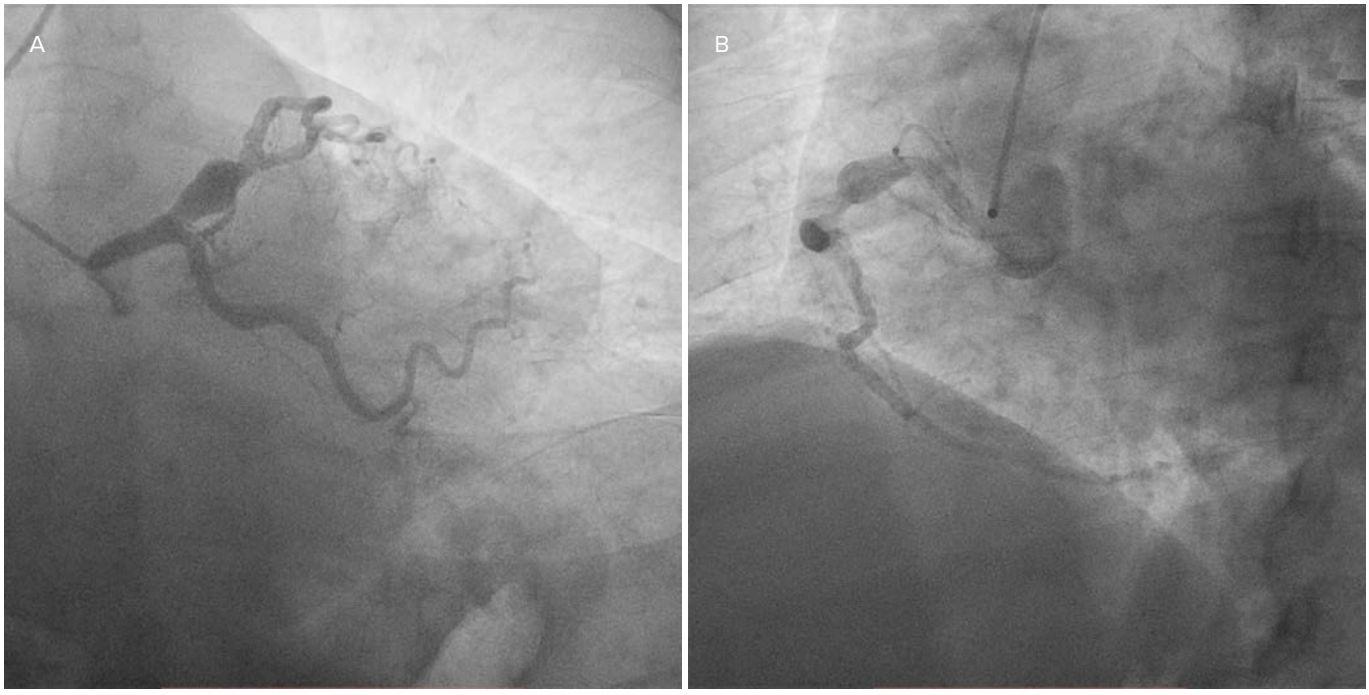

FIGURE 1. Invasive coronary angiography revealing aneurysmal dilations in the left anterior descending artery (**A**) and right coronary artery (**B**), with no significant vascular stenoses

tis, pericarditis and other associated diseases.[4] The time is essential when establishing the diagnosis of Kawasaki disease, meaning that as soon as the treatment is initiated, less damage will occur to the coronary arteries, which most often consists in coronary artery dilation and aneurysm formation. Most frequently, patients present complete recovery, with no related coronary disease after having presented Kawasaki disease. Adult patients with a history of Kawasaki disease may present acute myocardial infarction, with no recollection of the disease that had occurred during childhood, which may cause difficulties in the interventional treatment and further patient management.[5]

CASE PRESENTATION

We present a 53-year-old obese woman known with Kawasaki disease, diabetic, with dyslipidemia, who was hospitalized in emergency conditions, after presenting with constrictive retrosternal chest pain of an extended duration over 30 minutes, with radiation in the left shoulder and arm, associated with dyspnea, nausea and vomiting. The medical history of the patient revealed no significant family history of cardiovascular disease. However, the patient had presented a previous myocardial infarction 6 years prior to the current presentation, and she was also known with type 2 diabetes mellitus, arterial hypertension, mixed dyslipidemia with the predominance of hypertriglyceridemia.

Upon initial physical examination, the pacient was obese, with normal coloured skin, without superficial palpable adenopathies, normal breathing motions of the thoracic wall, cardiac and pulmonar sounds were normal upon auscultation. Blood pressure was 140/90 mmHg and heart rate was 85 bpm, with no sings of peripheral artery disease or vascular murmurs.

On admission, the white cell count was 12,750, the platelets count 348,600, low-density lipoprotein cholesterol 123 mg/dL, total cholesterol 239 mg/dL, triglyceride 504 mg/dL, serum high sensitivity troponin-I 0.13 ng/mL , creatine kinase myocardial brain (CK-MB) 148 ng/mL.

The 12-lead ECG revealed sinus rhythm, 85 bpm, intermediate QRS axis, ST-segment elevation in inferior leads, with the presence of Q wave, indicating an inferior wall myocardial infarction. The patient underwent emergency invasive coronary angiography, after receiving dual antiplatelet therapy with loading doses, and anticoagulation with unfractionated heparin. The coronary angiogram revealed an 11 mm aneurysmal dilation of the proximal segment of the left anterior descending artery (LAD), with no significant coronary stenoses at this level. The left circumflex artery was normal, with no atherosclerotic lesions, while the right coronary artery (RCA) presented a sinusoidal trajectory, with multiple aneurysmal dilations,

with the diameter up to 9 mm, without any significant coronary stenoses (Figure 1). No interventional percutaneous coronary revascularization procedure was employed since there were no significant coronary artery stenoses.

The transthoracic echocardiographic examination performed 2 days after the coronarography revealed: Left ventricle diameter 45/36 mm, posterior wall 10 mm, interventricular septum 12 mm, ascending aorta 34 mm, aortic valve opening 19 mm, E/A ratio 58/81, DT 160 msec; mitral valve – supple, mobile, no pathological regurgitation; tricuspid valve – supple, mobile, with mild to moderate regurgitation with right ventricle/right atrium gradient 17 mmHg and Vmax 2.05 m/sec; aortic valve – supple, mobile, no pathological regurgitation. Ejection fraction 50%. Efficient global contractility of left ventricle, discreet hypokinesia of inferior wall and diastolic dysfunction.

The established treatment was with antiplatelets, statins, antihypertensives with favorable evolution, controlled value of vascular tension and relief of symptoms. The patient was discharged after 6 days, on clopidogrel 75 mg/day, acetylsalicylic acid 100 mg/day, rosuvastatine 10 mg/day, ezetimibum 10 mg/day, metoprolol 50 mg/day, ramipril 10 mg/day, and acenocumarolum 4 mg/day.

The patient signed a written informed consent, in which she approved the publication of her medical information, and the study procedures were performed according to the ethical principles stated in the Declaration of Helsinki.

DISCUSSION

The course of patients with Kawasaki disease and persistent coronary artery abnormalities is not well known. Even though the aneurysmal dilatation diminishes in a period of time, the secondary stenotic lesions are usually progressive.[6] The larger the aneurysms, the higher the rate of stenosis. The main causes of death in Kawasaki disease are myocardial infarction caused by thrombotic occlusion, stenotic occlusion or both.[7]

In this case the particularity was that the revascularization was not necessary, even though the coronary artery aneurysms were massive (over 8 mm in diameter),[8] the patient was stable under conservative treatment and released stable both electrically and hemodynamic, with a favorable evolution.

CONCLUSION

Kawasaki disease is an acute vasculitis of unknown etiology that affects the medium sized arteries and produces aneurysmal dilatation. Without specific treatment and long-term management, it may cause ischemic heart disease and even the most severe form – acute coronary syndrome, which was the particularity of this case.

CONFLICT OF INTEREST

Nothing to declare.

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