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Cocaine Induced Aneurysmal Dilation of Left Main Coronary Artery

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INTRODUCTION

Coronary artery aneurysms (CAA) are a rare but clinically significant condition that occurs when there is a localized dilation of a coronary artery. These aneurysms are defined as a 1.5-fold increase in diameter compared to the surrounding normal vessel segments. They tend to be in the left anterior descending artery, followed by the right coronary artery and left circumflex artery.[1] The underlying etiology of CAA can be classified as congenital or acquired. Congenital diseases tend to result in singular lesions, and include connective tissue diseases (i.e., Marfans or Ehlers Danlos) and congenital coronary aneurysms. Acquired aneurysms tend to be found in multiple locations and are associated with various risk factors such as hypertension, atherosclerosis, and inflammation.[2,3]

In children, CAA is most frequently caused by Kawasaki disease, while in adults, it is most associated with coronary artery disease and second most by Kawasaki disease.[4] Autoimmune vasculitis that involves coronary vessels include Takayasu arteritis, systemic lupus erythematosus, polyarteritis nodosa, and rheumatoid arthritis.[5] Other etiologies include infections via direct invasion and drug use causing local vasoconstriction and ischemia.[6] The overall reported incidence of coronary artery aneurysms varies from 0.2% to 10%, where up to 30% of cocaine users were found to have aneurysms.[1,4,6,7] The clinical presentation of CAA can vary, with some patients being asymptomatic, while others may present with chest pain, shortness of breath, or even sudden cardiac death.[6] Some patients may develop serious complications such as coronary thrombosis, dissection, and rupture that may increase mortality risk.[4] There is limited evidence on management of CAAs, though an algorithm has been proposed to individualize treatment strategy based on patient characteristics, risk factors, symptomatology, presence of coronary stenosis, and morphology of the aneurysm.[3]

In this case study, we describe a young female patient who presented with non-ST elevation myocardial infarction and was found to have multivessel disease and a coronary artery aneurysm. The patient ultimately underwent coronary artery bypass grafting (CABG) as a result. This case highlights a potential complication associated with drug use, specifically cocaine, and the importance of considering CAA in patients with multivessel disease.

CASE REPORT

A young adult female with a history of substance abuse, specifically cocaine use within the past two days, presented to the emergency department with acute chest pain and shortness of breath upon awakening. The patient had no other significant past medical history. Initial evaluation, including an EKG and high sensitivity Troponin (hs-T) level, revealed non-specific ST-T wave changes in the inferior leads and an elevated hs-T level that peaked at 34,060 ng/L. She was then admitted to the ICU and started on a heparin drip per acute coronary syndrome (ACS) protocol.

The patient had an elevated D-dimer level of 1216 ng/mL, a CT angiography of the chest was performed and was negative for a pulmonary embolism. A transthoracic 2D echocardiogram was performed and showed a normal left ventricular ejection fraction of 55%-60%. Coronary angiography revealed an obtuse marginal artery with 90% stenosis, left circumflex artery with 90% stenosis, and mid-left anterior descending artery (LAD) with 50%-60% stenosis with aneurysmal dilatation. Given the significant stenosis in multiple coronary arteries, a Coronary Artery Bypass Graft (CABG) procedure was performed.

Given her young age, autoimmune testing was conducted. The erythrocyte sedimentation rate was elevated at 36 mm/hr and anti-U1-ribonucleoprotein antibody elevated at 1.1 AI. Other inflammatory and autoimmune markers were negative. In addition, CT angiography of the head, neck, chest, and abdomen did not show evidence of vasculitis or other aneurysms. An artery biopsy could not be obtained during surgery, and ultimately further workup was deferred to an outpatient setting with cocaine use as the plausible etiology.

DISCUSSION

This case report describes a young female patient who presented with non-ST elevation myocardial infarction and was found to have multivessel disease and a coronary artery aneurysm. The patient had a history of substance abuse, specifically cocaine use within the past two days. This case highlights the potential complications associated with drug use, specifically cocaine, as a rare cause of coronary artery aneurysm and the importance of considering CAA in patients with multivessel disease.

The underlying mechanisms of how cocaine can cause coronary artery aneurysms are not fully understood. However, it is believed that cocaine's use can lead to episodic vasoconstriction and episodic hypertension, leading to the subsequent dilation of the coronary artery through repeated ischemia and endothelial weakening.[7] Additionally, cocaine has been shown to increase the formation of reactive oxygen species, which can lead to inflammation and damage to the arterial wall. Other literature has also reported cases of cocaine-associated coronary artery aneurysms, showing a prevalence of coronary artery aneurysms in up to 30.4% of young cocaine users.[7] Therefore, it is important to consider cocaine as a risk factor for CAA.

CAAs can have serious complications if left untreated, including thrombosis, rupture, and sudden cardiac death. In rare cases, the aneurysm can dilate to four times that of a normal coronary artery, leading to a higher risk of angina and myocardial infarction. This is called a Giant CAA.[8] Additionally, cocaine also places users at risk of other cardiac sequela, such as infarction, aortic dissection, myocarditis,

dilated cardiomyopathy, and arrhythmias.[9] Therefore, early diagnosis and management are crucial to prevent these complications. In this case, the patient ultimately underwent CABG because of multivessel disease.

Limitations. Limitations of this report include the lack of detailed information on the patient's substance abuse history. Despite the strong association between cocaine use and the presence of a CAA in our patient, a definitive causal relationship cannot be established without further histological examination. Unfortunately, in this case, a biopsy was not performed. Further research is needed to better understand the underlying mechanisms of how cocaine use leads to the development of CAAs and to determine the best course of treatment for these patients.

CONCLUSIONS

CAA is a known complication of several etiologies, ranging from congenital to acquired diseases. While seen most often due to Kawasaki Disease and atherosclerosis, additional causes include substance use and other autoimmune vasculitides. While aneurysms can remain asymptomatic, they can also manifest as life-threatening acute coronary syndrome and even rupture if large enough. It is thus important to keep this complication in mind while caring for patients with conditions that place them at greater risk.

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