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CASE REPORT

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Giant left main stem aneurysm in a patient with non-sustained ventricular tachycardia: A case report

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Key Clinical Message

It is important to maintain a vigilant approach towards incidental, rare findings like coronary artery aneurysms (CAAs) presenting with arrhythmia, particularly in patients with a history of myocardial infarction and cardiovascular risk factors. Diagnosis needs thorough evaluation. Tailoring management strategies to individual patient characteristics is crucial for optimizing outcomes.

KEYWORDS

atherosclerosis, coronary artery disease, giant coronary artery aneurysms, ischemic heart disease, non-sustained ventricular tachycardia

1 | INTRODUCTION

CAAs are uncommon angiographic findings with an incidence of less than 5% and are characterized by focal dilation of the coronary artery more than 1.5-fold the diameter compared to an adjacent patent segment.[1] CAAs are termed giant if their diameter is greater than the reference vessel diameter by four times or if they are larger than 8 mm in diameter.[2] They most commonly involve the proximal and middle segments of the right coronary artery (RCA), followed by the proximal left anterior descending (LAD) and the left circumflex arteries.[3] It is rare to see aneurysms in the left main stem.[3] Although the causative factor is mainly atherosclerosis, it can also be caused by Kawasaki disease, Takayasu arteritis, congenital abnormalities, and percutaneous coronary intervention.[1,3]

The clinical presentation varies from asymptomatic cases to sudden cardiac death.[4] Although giant CAAs are often incidental findings, many complications, such as local thrombosis, distal embolization, rupture, vasospasm associated with ischemia, heart failure, and arrhythmias have been reported. The optimal medical, interventional, or surgical management still needs to be clarified.[5] For small asymptomatic coronary aneurysms, conservative, or medical management is preferred. Symptomatic or large aneurysms, however, may require surgical or percutaneous intervention. We present the case of a left main stem CAA that was detected after episode of NSVT, with the patient being asymptomatic. To the best of our knowledge, this is the first case describing a main stem aneurysm that presented as an NSVT.

2 | CASE PRESENTATION

2.1 | History

The patient is an 85-year-old male with a past medical history of coronary artery disease, peripheral artery disease, type 2 diabetes mellitus, hypertension, and a history of infrarenal abdominal aortic aneurysm status post graft repair in 2008 who presented to our hospital for a screening

colonoscopy. His pre-operative electrocardiogram (ECG) showed a wide QRS complex tachycardia, leading to postpone his procedure. The patient, who was asymptomatic, denied any history of arrhythmias. He is a 20-pack-year smoker and denied drinking alcohol or having any past or current drug abuse.

2.2 | Physical examination

On physical examination, there were no murmurs or gallops. The point of maximal impulse was lateral to the midclavicular line.

The patient was hemodynamically stable, afebrile, with a blood pressure of 104/69 mmHg, heart rate of 121 beats/min, and respiratory rate of 18 breaths/min.

2.3 | Investigations

A 12-lead ECG showed NSVT with a heart rate of 86 along with a right bundle branch block (Figure 1). Initial laboratory testing revealed a troponin of 34 ng/L (normal range 3–17 ng/L) and no electrolyte abnormalities. A bedside echocardiography showed inferior lateral wall motion abnormalities. Because of this, the patient was loaded with 325 mg of aspirin and was placed on a heparin drip for possible acute coronary syndrome (ACS). His thrombolysis in myocardial infarction (TIMI) score was 4. A formal transthoracic echocardiogram (TTE) was obtained and showed decreased left ventricular systolic function with an ejection fraction of 15%–20%, global hypokinesis with akinesis of the basal to mid anterolateral and inferolateral segments indicative of prior infarct or scarring. Frequent ventricular ectopy was noted during the image acquisition, but no left ventricular thrombi or masses were found.

On the second day of hospital admission, the patient was taken for cardiac catheterization to investigate the etiology of the newly detected NSVT. The coronary angiogram revealed complete occlusion of RCA with collateral vessel formation and a 1.5 cm aneurysm at the origin of the left main coronary artery into the LAD and circumflex artery (Figure 2).

2.4 | Treatment, outcome, and follow-up

Despite adequate flow through the aneurysm without evidence of thrombus formation, cardiothoracic surgery was consulted for consideration of surgical repair. However, due to the patient's advanced age, significant comorbidities and high risk surgery, a consensus decision was made to pursue medical management with dual antiplatelet therapy (DAPT). Guideline-directed medical therapy for heart failure with reduced ejection fraction was initiated, consisting of carvedilol 6.25 mg twice daily, empagliflozin 10 mg daily, spironolactone 12.5 mg daily, and sacubitril/valsartan 49/51 mg twice daily. Additionally, the patient received a 10-day course of amiodarone therapy. Before discharge, an automatic implantable cardioverter defibrillator (AICD) was implanted. Furthermore, the patient was advised to undergo outpatient computed tomography angiography (CTA) of the thorax, abdomen, and pelvis to evaluate for additional aneurysmal dilations.

3 | DISCUSSION

CAAs represent an uncommon diagnostic finding, often incidentally identified during imaging studies or coronary angiography. While the RCA is frequently involved, the occurrence of giant CAAs in the left main stem is exceedingly rare.[3,6] Our case, with a diameter of 1.5 cm, is a giant CAA by definition. Although the underlying pathophysiology is not fully elucidated, atherosclerosis remains a predominant etiological factor in

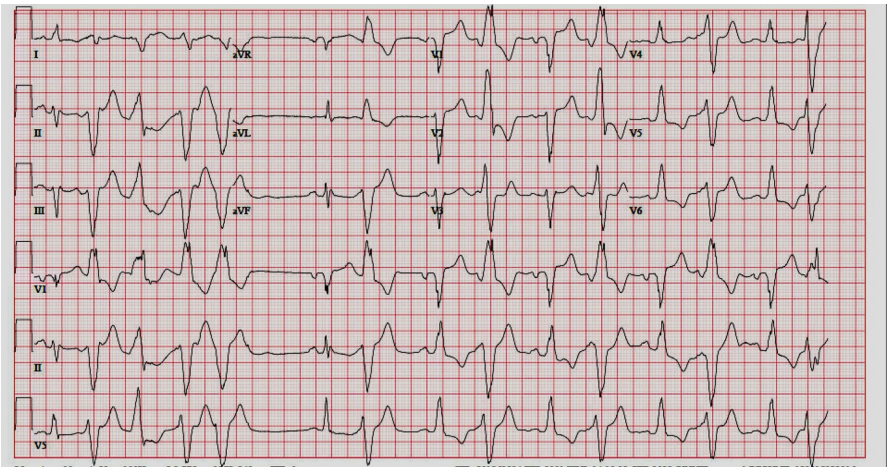

FIGURE 1 12-lead ECG showing non-sustained ventricular tachycardia with right bundle branch block.

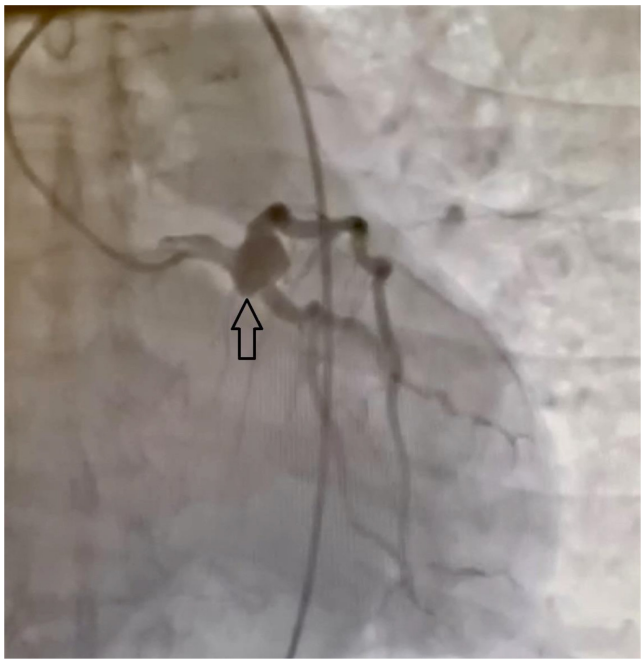

FIGURE 2 Arrow in coronary angiogram demonstrating aneurysm at the origin of left main coronary artery.

adults, while Kawasaki disease accounts for a significant proportion of cases in children.[7] Other causes of CAAs include congenital abnormalities, vasculitic disorders such as Takayasu arteritis, and iatrogenic interventions such as percutaneous coronary intervention.[7,8] CAAs can be identified noninvasively using computed tomography (CT) and magnetic resonance imaging (MRI). CTA angiography and magnetic resonance coronary angiography (MRCA) provide detailed images of the aneurysm's anatomy, size, and location, which are useful for planning surgical interventions. Nevertheless, x-ray coronary angiography remains the definitive method for diagnosing coronary aneurysms.[9]

In our patient, the presence of atherosclerotic risk factors and prior coronary artery disease suggested atherosclerotic etiology, although direct visualization of atherosclerosis was challenging due to the aneurysmal nature of the vessel observed on angiography. However, the TTE findings consistent with prior myocardial infarction/scarring support this. Additionally, our patient has many risk factors for atherosclerosis. Moreover, atherosclerosis can sometimes be extraluminal and may not be defined in cardiac catheterization, coronary angiography can only provide a two-dimensional outline of the coronary lumen and cannot thoroughly demonstrate the complex nature of atherosclerotic plaques. In contrast, coronary CT angiography enables generation of 3D intraluminal views of coronary wall and plaque.[10]

CAAs are generally asymptomatic; however, they can present with stable angina or ACS if symptomatic or during rupture. Complications may involve local thrombus formation, cardiac tamponade, fistula formation, sudden cardiac death, and myocardial infarction due to subsequent distal embolization. In our case, the patient was asymptomatic; however, he was incidentally found to have NSVT during preoperative preparation for screening colonoscopy. His NSVT was likely attributable to a prior myocardial scar, given his history of right coronary artery disease with collaterals, indicative of a chronic pathological process.

Managing patients with CAAs poses significant challenges due to several factors. Firstly, the natural history of CAAs remains largely uncertain. Consequently, determining the best course of action for incidentally discovered CAAs or coronary ectasia without accompanying stenosis or occlusion is unclear. Secondly, for patients experiencing angina or acute myocardial infarction caused by an aneurysmal culprit, where intervention is necessary, both percutaneous and surgical revascularization methods present technical difficulties. Moreover, there is a considerable knowledge gap in both symptomatic and asymptomatic CAAs cases, as randomized trials and comprehensive data are lacking. Current recommendations are predominantly derived from small case studies or anecdotal evidence. Acknowledging these limitations, treatment strategies should be individualized based on the location and morphology of the CAAs, the patient's characteristics, and the clinical presentation.[11]

There is limited large-scale outcome data on the management of CAAs, and the data are controversial. Small, asymptomatic coronary aneurysms are generally managed conservatively with aggressive cardiovascular risk factor modification, antiplatelet therapy, and anticoagulation, along with recommended monitoring every 3 months. Symptomatic CAAs and giant CAAs require surgical intervention, which may involve CABG, resection with end-to-end anastomosis, or interposition vein graft through median sternotomy. For patients at high surgical risk, non-surgical options like coil embolization and percutaneous stent placement are available.[12] Negro et al. analyzed 180 studies and reported 209 left main coronary aneurysms (LMCAs), with 53% of cases treated surgically and only 7% undergoing percutaneous coronary intervention (PCI). However, data regarding antithrombotic therapy were scarce.[13] Conversely, the international coronary artery aneurysm registry trial (CAAR) included 1565 patients with CAAs, with 69% undergoing revascularization with PCI and 53% undergoing coronary artery bypass graft (CABG). After a median follow-up of 37.2 months, there were no differences in aneurysm complications, unstable angina, infarction, embolism, stroke, bleeding, or major adverse cardiovascular events (MACE)

between the CABG and PCI groups. As for antithrombotic therapy, 90.2% of patients received aspirin, 64.8% received DAPT, and 13.4% were on anticoagulation.[14] Our patient, an 85-year-old with multiple medical comorbidities, including coronary artery disease with total right coronary artery occlusion and heart failure with reduced ejection fraction, was at elevated risk for CABG. However, the vessels were deemed ineligible for the bypass procedure by the surgical team, so he was managed with DAPT at discharge.

4 | CONCLUSION

This case report underscores the diagnostic challenge posed by giant CAAs, particularly when involving the left main stem. The incidental discovery of a giant left main stem CAA following an episode of NSVT highlights the importance of comprehensive cardiovascular evaluation in patients with risk factors. Individuals diagnosed with giant CAAs face elevated risks of complications, necessitating diligent monitoring and individualized management approaches. Optimal management strategies for giant CAAs remain uncertain, as current recommendations based on few case studies and lack clinical trials, necessitating individualized approaches based on patient characteristics and clinical presentation.

AUTHOR CONTRIBUTIONS

Saliha Erdem: Conceptualization; writing – original draft. **Ahmad Damlakhy:** Conceptualization; methodology; writing – original draft; writing – review and editing. **Amrit Kanwar:** Conceptualization; writing – original draft. **John Dayco:** Conceptualization; methodology; writing – original draft; writing – review and editing. **Randy Lieberman:** Conceptualization; data curation; investigation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT

The data used to support this study are included within the article. Further inquires can be directed to the corresponding author.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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