MARSHALL JOURNAL OF MEDICINE
Expanding Knowledge to Improve Rural Health.

Volume 4 | Issue 2 Article 4

2018

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Recommended Citation

Aqtash, Obadah; Amro, Ahmed; Alam, Bilal; Sobeih, Amal; Lester, Melissa; and Sayyed, Rameez (2018) "Urgent Coronary Artery Bypass Grafting Due to Multi- Vessel Coronary Aneurysm," *Marshall Journal of Medicine*: Vol. 4: Iss. 2, Article 4.

DOI: http://dx.doi.org/10.18590/mjm.2018.vol4.iss2.4

Available at: https://mds.marshall.edu/mjm/vol4/iss2/4

DOI: http://dx.doi.org/10.18590/mjm.2018.vol4.iss2.4

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This case report is available in Marshall Journal of Medicine: https://mds.marshall.edu/mjm/vol4/iss2/4

Urgent coronary artery bypass grafting due to multi- vessel coronary aneurysm

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The authors have no financial disclosures to declare and no conflicts of interest to report.

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Abstract

Coronary artery aneurysm (CAA) is defined as a dilation of more than 1.5 times normal in a segment of the coronary artery. The incidence of CAA is highest in the right coronary artery (RCA); left main coronary artery or three-vessel involvement is extremely rare. Wide ranges of factors have been implicated in the cause of CAA; atherosclerosis is the most common at 50%. Congenital CAA occurs in 20-30% of cases followed by connective tissue disease at 10%. Blood stagnation and exposure of the underlying collagen make aneurysms prone to thrombosis, dissection, and vasospasm. Depending on the size, symptoms, and etiology of the aneurysms, a surgical, percutaneous, or medical approach may be used. In this paper, we present a patient who presented to the emergency department (ED) with chest pain due to acute coronary syndrome (ACS) that was found to be due to multi-vessel CAAs. The left main as well as RCA, left anterior descending (LAD) artery and left circumflex artery (LCA) were involved and urgent coronary artery bypass grafting (CABG) was required.

Keywords

Coronary artery aneurysm, Non-ST-segment elevation myocardial infarction, Coronary artery bypass grafting

Introduction

Coronary artery aneurysm (CAA) is defined as a dilation of more than 1.5 times normal in a segment of the coronary artery.[1,2] The incidence of CAA is higher in the right coronary artery (RCA) followed by the left anterior descending (LAD) artery and left circumflex artery (LCA).[2] It is agreed that left main coronary artery or three-vessel involvement is extremely rare.[1,3] In this paper, we present a patient who presented to the emergency department (ED) with chest pain due to acute coronary syndrome (ACS) that was found to be due to multi-vessel CAAs involving the left main as well as RCA, LAD, and LCA and required urgent coronary artery bypass grafting (CABG).

Case Presentation

A 37 year old Caucasian male smoker presented to the ED with sub-sternal chest pain. Medical history is significant for hypertension, hyperlipidemia, and ST-segment elevation myocardial infraction three years earlier that was managed with a bare metal stent placement in the LAD with no evidence of aneurysms. In the ED, the patient was hemodynamically stable with a blood pressure of 131/90 mmHg and a heart rate of 90 /min. Physical exam was unremarkable. Electrocardiogram showed normal sinus rhythm with a non-specific intraventricular conduction delay. Initial laboratory work up including erythrocyte sedimentation rate was normal with the exception of an elevated white blood cell count and troponins. The patient was consequently diagnosed as a non-ST-segment elevation myocardial infarction and placed on a heparin drip. An urgent 2-dimensional transthoracic echocardiogram showed an ejection fraction in the range of 55-65%, with no regional wall motion abnormalities. The patient underwent emergent coronary angiography and was found to have severe aneurysmal disease in all of his coronaries (Images 1, 2, 3, 4, and 5) with significant amount of dye stagnation seen at the aneurysmal sites. The patient underwent emergent three vessels CABG to LAD, obtuse marginal and posterior descending

artery. He followed up as an outpatient one month after discharge and was doing well with no new episode of chest pain. The patient did undergo computed tomography scan due to abdominal pain at that time which was negative for any kidney pathology.

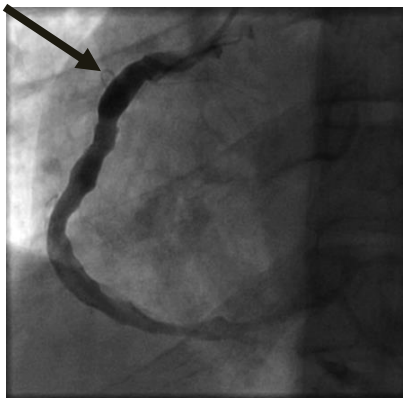

Image 1: Right Coronary Artery dilated as it progresses (arrow)

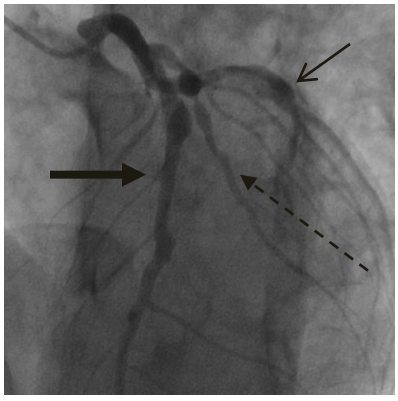

Image 2: Left Anterior Descending (thick arrow), Left Circumflex (thin arrow), Ramus Intermedius (dashed arrow).

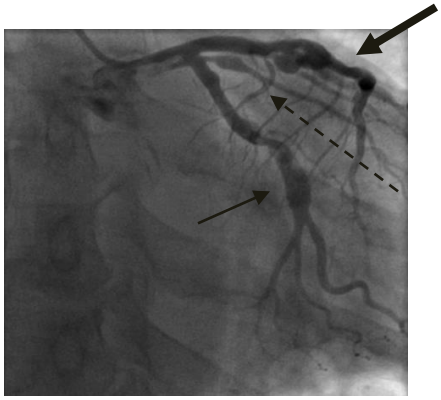

Image 3: Left Anterior Descending (thick arrow), Left Circumflex (thin arrow), Ramus Intermedius (dashed arrow)

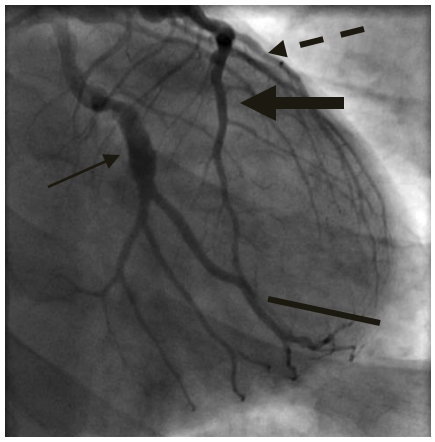

Image 4: Left Circumflex (thin arrow), Left Anterior Descending (thick arrow), Diagonal Branch (dashed arrow), Marginal Branches (straight line)

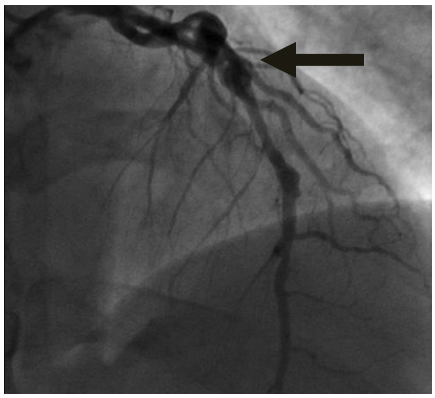

Image 5: Left Anterior Descending (thick arrow)

Discussion

CAA is an uncommon disease with an estimated incidence of 0.3% to 5.3%.[2] It is defined as a coronary artery segment dilation of more than 1.5 time's normal.[1,2] Those that are 4 times the diameter of an adjacent artery are called giant coronary artery aneurysms.[4] Stenosis greater than 70% of the coronary artery is found in the majority of patients.[4] CAAs have a male predominance and are more commonly localized to the RCA.[5] As with aneurysms of larger arteries, CAA can be fusiform or saccular. Fusiform aneurysms have a localized dilation involving the entire circumference and are more common than saccular aneurysms, in which only a portion of the circumference is dilated.[5] A combination of both types of aneurysms can occur, as was the case in our patient.

A wide range of factors have been implicated in the cause of CAA; atherosclerosis is the most common at 50%.[1,2] The process of atherosclerosis is a slow and complex one. Lipid is deposited in the tunica intima with hyalinization eventually occurring.[5] This is followed by an inflammatory process resulting in luminal stenosis with possible occlusion. Turbulent blood flow through this stenosis results in increased wall stress inducing endothelial damage.[6] The tunica

media and adventitia are mainly affected resulting in arterial remodeling, dilatation, and thus CAA formation.[2,5] This leads us to suggest that CAA is a variant of coronary atherosclerosis and not a separate phenomenon.

Another possible mechanism is the compensatory dilation of the atherosclerotic vessel resulting in an overall increased diameter. This allows the luminal diameter to be maintained which can be demonstrated on intravascular ultrasound. If the obstructive occlusion were to regress, it could lead to ectasia.[7] In certain cases, coronary arteries which are not affected with atherosclerosis will see considerable amount of dilation as a result of increased flow through these vessels as a compensatory mechanism. One theory is that the increased shear force (flow) through the vessel triggers the release of endothelium derived relaxing factor causing vasodilation.[8] Chronic overstimulation of nitric oxide can also lead to vascular relaxation and ectasia.[9]

We suspect atherosclerosis to be the cause of CAAs in our patient. This is supported by a left heart catheterization three years earlier, which did not show any aneurysms. Also our patient had the following risk factors for atherosclerosis: hypertension, hyperlipidemia, and chronic smoking.

Congenital CAA occurs in 20-30% of cases followed by connective tissue disease at 10%.[1] The most common of the connective tissue diseases is Kawasaki disease but CAAs can also be seen in patients with Takayasu's arteritis, systemic lupus erythematosus, Marfan syndrome, polycystic kidney disease, and hyper-IgE syndrome.[1] Matrix metalloproteinases are large molecules responsible for tissue turnover by degrading the extracellular matrix proteins. Overexpression of these molecules with elevated levels in blood is thought to play a major role in the CAA formation in Kawasaki disease.[4] Other less common causes of CAAs include infections (bacterial, fungal, HIV, syphilis), drug use (cocaine), iatrogenic conditions, and trauma.[1] Previous history of percutaneous coronary intervention with placement of bare metal stent has been implicated in 4% of cases.[4] This occurs due to mechanical damage caused by the stent with possible dissection and leakage of blood between the layers resulting in a false aneurysm.

Blood stagnation, turbulent flow, and exposure of the underlying collagen make aneurysms prone to thrombosis with possible distal embolization, dissection, and vasospasm.[9,10] The consequence is myocardial ischemia with clinical manifestations that include angina pectoris, dyspnea, edema, myocardial infraction, and sudden death.[11] CAAs are also susceptible to rupture with associated fistula, cardiac tamponade, or hemopericardium.[6]

Depending on the size, symptoms, and etiology of the aneurysms, a surgical, percutaneous, or medical approach may be used. Small asymptomatic aneurysms can be observed with frequent follow ups. Antiplatelet or anticoagulation may be used to reduce the risk of thrombosis. Larger symptomatic aneurysms are usually treated by a surgical approach. This is especially the case with rapidly expanding aneurysms and those involving the left main or bifurcation of larger branches as was seen in our patient.[4] Surgical techniques vary but involve aneurysm resection, ligation, or marsupilization followed by the interposition of graft vessels.[12,13] In regard to our patient, tourniquets were placed in the proximal aspect of the aneurysmal vessels to stop the blood flow followed by graft anastomosis. Patients with higher perioperative risk may be managed with a percutaneous approach.[5] This is limited however by the possibility of restenosis

especially in aneurysms >10mm in diameter.[1] In these situations, the use of covered stents which are produced from polyethylene terephthalate and polytetrafluoroethylene provides a layer between the stent and inflammatory process helping to reduce the incidence of restenosis.[4]

Conclusion

Coronary artery aneurysm is a rare but important disease that can manifest itself as acute coronary syndrome. Treatment can be challenging. Most of the data on this topic is found in case reports with no true randomized studies available.

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