Ventricular Tachycardia due to Anomalous Origin of Right Coronary Artery
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Citation

Abstract
Coronary anomalies are observed in 0.8 to 1% of patients on angiography and about 8% of these occur in Right coronary artery (RCA). Anomalous origin of RCA from left coronary sinus (AORCALS) can cause myocardial ischemia, arrhythmia, syncope and sudden cardiac death (SCD). We report a case of AORCALS presenting as exercise induced ventricular tachycardia (VT). A 48 year old Afro-American female with no known cardiac history presented with exertional palpitations and associated atypical exertional chest pain for couple of months. Dobutamine stress echocardiogram showed normal left ventricular structure and systolic function with mild reversible inferior wall ischemia. An event monitor revealed non sustained monomorphic ventricular tachycardia (VT). Coronary angiogram revealed RCA originating from the left coronary cusp without any significant luminal stenosis. Further evaluation of RCA course with cardiac CT angiography or MRI was recommended. She declined any further invasive testing or treatment. Age less than 30 is more frequently associated with SCD. Sudden cardiac death is more common when aberrant coronary artery is dominant. We report the first case where ANRCA presents as exercises induced ventricular tachycardia. In our patient, compression of RCA between the great vessels was suspected. Any young patient presenting with ventricular arrhythmias or angina without cardiac risk factors should be considered for coronary anomalies. Young symptomatic patients should be considered for definitive treatment to prevent SCD.

INTRODUCTION
Congenital coronary anomalies are not very common but are often associated with myocardial ischemia. RCA anomaly accounts for around 8% of total coronary anomalies\(^1\). Origin of RCA from left coronary sinus has an incidence of 0.03 to 0.11%\(^1\). Roberts et al reported first case of Anomalous RCA from left coronary sinus in 1982\(^3\). It is associated with increased risk of sudden cardiac death during exercises among young adults\(^1\). Age less than 30 is more frequently associated with SCD\(^2\). The risk of sudden death is attributed to myocardial ischemia and electrical instability because of a slit-like lumen, narrow ostium with acute angled takeoff of the anomalous artery or by compression between the pulmonary trunk and aorta\(^4\). Anomalous origin of RCA from left coronary sinus (AORCALS) can cause myocardial ischemia, arrhythmia, syncope, sudden cardiac death (SCD) and Heart Failure\(^1\). AORCALS is the second most common cause of athletic field deaths & accounts for one third of all sudden cardiac death (SCD) in young patients\(^2\). Sudden cardiac death from AORCA is very well described in the literature. Ventricular Tachycardia triggered by myocardial ischemia is certainly the basic mechanism leading to Sudden Cardiac Death\(^4\). However, it is infrequently described in the literature. We report a first case of AORCALS presenting as exercise induced ventricular tachycardia (VT). In our case, exercises induced Ventricular Tachycardia was recorded via Event Monitor. Such documented myocardial ischemia suggests collapse or compression of anomalous vessel during exercises, or vasospasm of the anomalous vessel. This mechanism has been described but scarcely documented in the literature.

CASE
A 48 year old Afro-American female with no known cardiac history presented with atypical exertional chest pain for couple of months. She also complained of exertional palpitations with dizziness lasting few minutes without syncope. Electrocardiogram suggested t wave inversion in III and AvF which were old. An event monitor revealed non sustained monomorphic ventricular tachycardia (VT). Dobutamine stress and resting Technetium Sestamibi Myocardial Imaging study was suggestive of mild reversible ischemia involving the inferior wall of the left ventricle. Trans Thoracic Echocardiogram showed normal left ventricular structure and systolic function. Coronary
angiogram was done to exclude coronary artery disease as an etiology for VT and it revealed RCA originating from the left coronary cusp without any significant luminal stenosis. The likely course of RCA was assumed to be between aorta and pulmonary artery. Further evaluation of RCA course with cardiac CT angiography or MRI was recommended. Patient refused further evaluation. Two years into follow up, patient remains alive with similar symptoms.

**Figure 1**

![Figure 1](image1.png)

**Figure 2**

![Figure 2](image2.png)

**DISCUSSION**

AORCALS is the second most common cause of athletic field deaths & accounts for one third of all sudden cardiac death (SCD) in young patients. Patients with AORCALS can present from teenage to late 40s with angina, myocardial infarction, syncope, arrhythmia or SCD. Age less than 30 is more frequently associated with SCD. Anomalous RCA usually originates from the anterior part of Left coronary Cusp or Aortic wall and lies anterior to Left Main Coronary Artery. It has an acute angle take off, travels across Aortic root and resumes its original track in AV groove. Anomalous origin of RCA from Left coronary Cusp causes vessel compression leading to transient ischemia due to distinct anatomical derangements. Several mechanisms of myocardial ischemia have been postulated. First, Acute angle take off of the Anomalous Artery produces both slit
like orifice and slit like lumen at Proximal RCA. Second, the Proximal RCA segment is intramural and is contained within the Aortic wall. The Initial segment and Aorta share tunica media. Both mechanisms, together or separately during physical exercises, lead to compression and collapse of the proximal segment as Aorta and Pulmonary trunk dilates. Finally, chronic external compression causes endothelial injury. It leads to vessel spasm and modification of coronary blood flow pattern. Endothelial dysfunction causes alteration of NO production and physiologic vasodilatation during exercises. In our patient, Dobutamine stress Echocardiogram was suggestive of mild reversible ischemia of inferior wall. Angiography confirmed the origin of RCA from left coronary sinus. CT scan or MRI was recommended to delineate anatomy and course of the proximal RCA. However, patient refused and was not done. The exact Mechanism of ischemia is impossible to prove as ischemia can be intermittent. Surgery is recommended for young symptomatic patients to prevent SCD. Single vessel Coronary artery bypass grafting with right Internal Mammary artery to RCA is the standard treatment strategy. However, Fedoruk et al have reported high occlusion rate of RIMA bypass graft dye to competitive flow. Associated atherosclerosis of RCA must be excluded to decrease chances of late re-occlusion. Unroofing technique, where proximal intra-mural segment of RCA is longitudinally excised, can be technically challenging with potential risk of Aortic Insufficiency. Implantation into correct coronary sinus, transaortic modification of the proximal RCA portion & unroofing of the intramural segment of the proximal RCA are other approaches. Reimplantation of RCA into right coronary sinus provides most physiological and anatomical correction and should be the preferred method of repair of this abnormality.

CONCLUSION

Any young patient presenting with ventricular arrhythmias or angina without cardiac risk factors should be considered for coronary anomalies. Coronary Dominance has been demonstrated to have a significant impact on the clinical outcome among patients with Coronary Anomaly. No major clinical consequences have been observed in patients with Anomalous RCA, who has dominant left coronary artery. Risk of SCD is high if Anomalous RCA is dominant, anomalous artery has prominent intramural course or Age is less than 35. Coronary CT or Cardiac MRI is recommended to delineate the course of the Anomalous RCA.

References

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