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Malignant Arrhythmia in Acute Conus Artery Occlusion

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ABSTRACT

We present the case of a 74 y.o. woman with multiple cardiovascular risk factors, admitted for worsening angina over the past three weeks. On admission she had no significant electrocardiographic and echocardiographic changes and a negative Troponin test.

Coronary angiography revealed single vessel disease: severe stenosis of the right coronary artery (RCA) ostium (difficult to assess visually), 50% mid-vessel and 60% distal segment. The left anterior descending artery and circumflex artery had non-significant stenoses. Fractional flow reserve technique (FFR) was used to evaluate the RCA ostial lesion which proved to be significant, therefore angioplasty with three drug-eluting stents was performed for all three lesions of the right coronary artery, starting from the ostium. Due to its location, minimal aortic protrusion of the first stent occluded a small ostial branch which proved to be the conus artery and the patient developed mild transient angina during the procedure, but with good outcome regarding the intracoronary flow. After the angioplasty the patient presented anterior leads ST-elevation and developed mild chest pain with an increase in cardiac enzymes (CK-MB peak 39 U/L).

Later on, she had two episodes of ventricular fibrillation with rapid defibrillation to sinus rhythm, with no further events or echocardiographic changes and no recurrent angina. The patient was started on amiodarone to prevent ventricular arrhythmias and continued double antiplatelet therapy with aspirin and clopidogrel. She was discharged six days later.

In conclusion, although the conus branch is a small artery, its acute occlusion can have significant life-threatening complications.

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Introduction

The right coronary artery (RCA) can give three small but extremely important branches: the conus artery, the sinoatrial nodal artery and the atrioventricular nodal artery. The conus artery is the first small branch of the RCA. In most cases it supplies the conus of the main pulmonary artery and the basal anterior interventricular septum [1]. It supplies the right ventricular outflow tract (RVOT), therefore its damaged blood flow is a potential cause for ventricular arrhythmias. Even if it is a small branch, its occlusion is associated with significant ST-elevation infarctions [2].

There are many anatomical variants. In a proportion of 30% to 50% of the population, this artery originates in an independent ostium from the right sinus of Valsalva, near the ostium of the RCA [3]. Sometimes this artery is described as the "third coronary" [4]. It often serves as the principal source of collateral circulation between the right and left coronaries, forming an anastomosis with the left anterior descending artery (ADA) - left conus artery, forming the "arterial circle of Vieussens" [5].

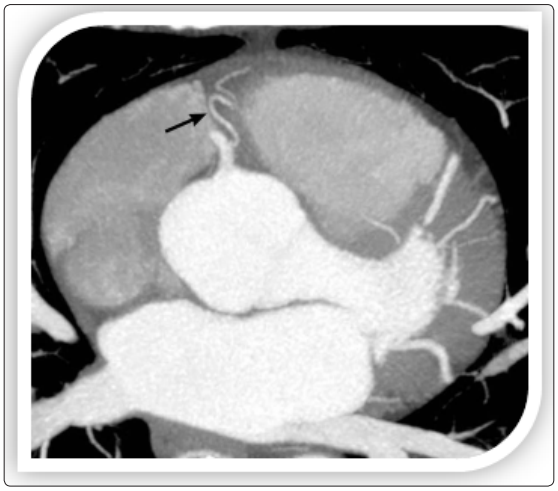

Figure 1: Image from: Computed tomography angiography of the right coronary artery showing the conus artery - source: Medscape, "What are the named arteries that can arise from the right coronary artery (RCA) on coronary computed tomography angiography (CCTA)?" [6].

The main role of this small conus artery in adults with coronary artery disease is that it is a major source of collateral circulation when the ADA becomes obstructed. It can also collateralize the distal segment of the RCA [3]. Since in about half of the patients the conus artery arises straight from the aorta, catheterization of the RCA might fail to opacify the conus artery.

Case presentation

We present the case of a 74 years old hypertensive, dyslipidemic, overweight Caucasian woman, with history of femoral aortic bypass, who was admitted for worsening angina and shortness of breath over the past three weeks. At admission she was clinically stable with no other significant signs or symptoms.

Laboratory tests revealed dyslipidemia with hypertriglyceridemia (170 mg/dL), hyperglycemia (131 mg/dL) and no increase in cardiac enzymes.

The initial electrocardiogram (ECG) showed sinus rhythm, a heart rate of 50 beats per minute (bpm) and a negative T wave in V1 lead (Figure2).

The transthoracic echocardiography revealed left ventricular hypertrophy (the interventricular septum measured 13 mm and the inferolateral wall 13 mm), normal systolic function with a left ventricular ejection fraction (LVEF) of 55%, without regional wall motion abnormalities, diastolic dysfunction grade 1 with normal filling pressure, mild mitral regurgitation, moderate tricuspid regurgitation.

Coronary angiography revealed the following lesions: the RCA had a severe ostial stenosis, which caused the cannulation of the ostial stenosis with the guiding catheter quite difficult, calcified stenosis of 50% in the second segment and a stenosis of 60% in the third segment (Figure 4); the ADA had a non-obstructive stenosis of 40-50% in segment I-II; the circumflex artery (CA) had also a non-obstructive proximal stenosis of 40%. Due to the difficulties in evaluating the severity of the RCA ostial stenosis, we decided to measure the fractional flow reserve (FFR). The value of the FFR was 0.42 in the second segment of the RCA and we decided that revascularization was recommended. The ostial stenosis of the RCA was pre-dilated with a non-compliant 3.0/15 mm balloon and a drug-eluting stent of 3.5/11 mm was placed, which was expanded at 14 atm. Then, the proximal part of the stent was post-dilated with its own balloon at 18 atm. After the stent implantation, we observed the occlusion of the conus branch associated with a short episode of mild chest pain (Figure 5). We continued the revascularization procedure of RCA with two additional drug-eluting stents in the second and third segment of the artery, which needed guided catheter extension with a good final result.

The ECG post-PCI showed ST-elevation in V1-V2 leads (Figure 3). The cardiac enzymes increased after the coronary angiography at about 3 times the normal value: creatine kinase (CK) 415 U/L and CK-myocardial band (CK-MB) 39 U/L. The transthoracic echocardiography revealed wall motion abnormality in the LAD territory.

The patient had two episodes of ventricular fibrillation, 3 hours and respectively 6 hours after the angioplasty, which were both successfully defibrillated (one external electric shock of 150 Joules for each episode of ventricular fibrillation). Intravenous amiodarone was initiated for secondary prevention of arrhythmias. The following day, there was ST-segment resolution and the echocardiography showed no wall motion anomaly.

Taking into account that the patient presented favorable clinical and paraclinical evolution, with the decrease of cardiac enzymes, resolution of the ST-segment elevation in V1-V2 leads, remission of the regional wall motion abnormality and no further arrhythmias occurred, it was decided that no secondary percutaneous coronary intervention was necessary.

The patient was discharged with the following treatment: double antiplatelet therapy (DAPT) (acetylsalicylic acid 75 mg daily and clopidogrel 75 mg daily) with the recommendation to maintain DAPT for at least one year after the angioplasty, atorvastatin 80 mg daily, bisoprolol 5 mg daily, ramipril 5 mg daily, pantoprazole 40 mg daily.

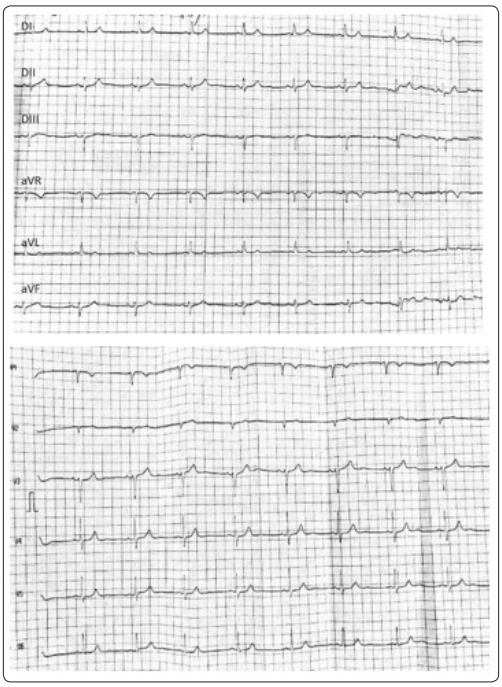

Figure 2: ECG at admission without ST-segment elevation

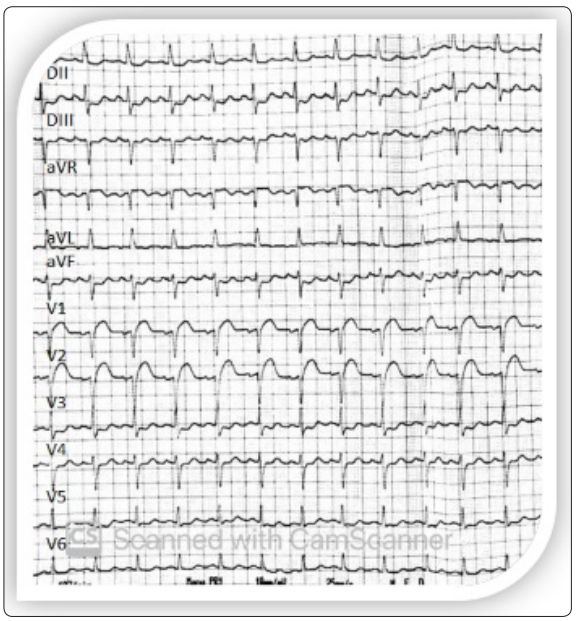

Figure 3: ECG after percutaneous coronary intervention: ST-segment elevation in V1-V2 leads

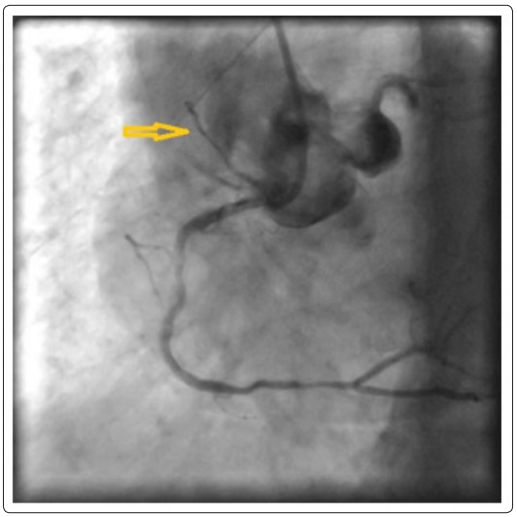

Figure 4: Initial coronary angiography of RCA in which we can observe the conus artery (yellow arrow).

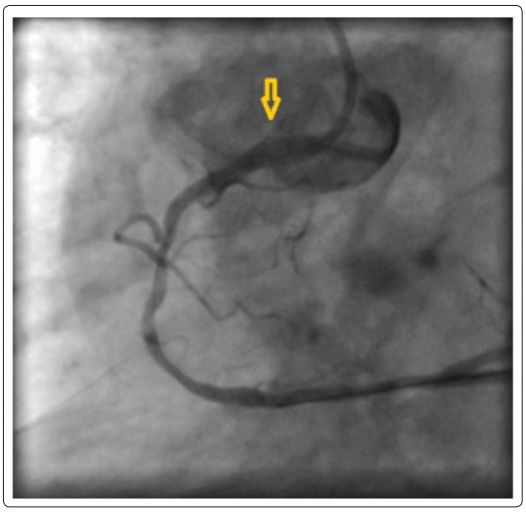

Figure 5: Coronary angiography after stenting the ostial lesion of the RCA in which we can observe the occlusion of the conus branch

1 month follow-up

The patient had no recurring angina, no arrhythmias on repeated Holter monitor and nothing was changed in her treatment so far.

Discussion

Spontaneous atherothrombotic conus branch occlusion can be the cause of acute myocardial infarction. There are a few reported cases of spontaneous acute myocardial infarction due to occlusion of the conus artery and can present with ST elevation in precordial leads [17]. Isolated RV infarction is seen in approximately 3% of all patients with myocardial infarction [9]. In spite of this, there are cases in which RV infarction leads to ST-segment elevation in precordial leads, mimicking anteroseptal or anterior infarction [10-12, 17].

In other cases, the cause of this kind of infarction is iatrogenic – plaque shift following PCI on RCA ostium, the mechanism which cause the acute coronary syndrome in our patient. Conus artery occlusion may be a complication of proximal RCA-PCI (percutaneous coronary intervention) and it may induce severe intraprocedural complications, such as chest pain, ventricular tachycardia, ventricular fibrillation) as previously presented.

The occlusion of the conus artery can have multiple causes, such as atherosclerosis, vasospasm or may be an intraprocedural complication during the angioplasty of RCA ostial lesions by plaque shift or caused by a guiding catheter [8]. The occlusion of the conus artery leads to myocardial ischemia of the RVOT and possibly of the lateral wall of the RV and/or of the anterior interventricular septum if it is a branch with significant dimensions. Usually, RV infarction is associated with left ventricular inferior wall infarction and it should manifest as ST-segment elevation in inferior leads DII, DIII, AVF and in right precordial leads V3R-V6R. Geft et al. also reported 5 cases of RCA occlusion with RV myocardial infarction with by ST elevation in leads V1-V5 [13]. A possible explanation for precordial ST-segment elevation in V1-V2 leads can be that the conus branch supplies blood to the anterior interventricular septum. The ECG changes in conus branch occlusion are similar to those that appear in Brugada syndrome [14], probably the ST elevation is caused by different mechanisms, but there is an electrical unevenness in both entities that carries a high arrhythmic risk.

The RVOT area is a potential origin for ventricular arrhythmias [2]. The phase 1 of the action potential (AP) is mediated by a transient outward potassium current which is stronger in the ventricular epicardium than in the endocardium [15]. This causes a transmural voltage gradient in the ventricular walls. The AP plateau is lost in the epicardium only, but not in the endocardium, leading to a voltage gradient during repolarization and to ST-segment elevation similar to that observed in Brugada syndrome [15]. The loss of AP plateau is caused by the currents that are active at the end of phase 1 of AP, such as the increase of the outward currents – adenosine triphosphate (ATP)-dependent potassium channel and arachidonic acid-sensitive potassium channel and the decrease of the inward currents - calcium channel and sodium channels [15].

Drugs as calcium channel blockers or sodium channel blockers, which are class IC antiarrhythmic agents, give rise to the loss of AP dome and may facilitate the occurrence of ventricular arrhythmias. The loss of AP plateau is predisposing also to premature ventricular contractions through re-entry mechanism, which precipitates ventricular fibrillation and/or ventricular tachycardia [15]. Consequently, the conus branch occlusion causes ischemia of the RVOT myocardium and that's why it may lead to electrical instability and predispose to ventricular fibrillation and/or ventricular tachycardia.

Amiodarone is a potassium channel blocker and is the antiarrhythmic agent of choice in cases of ventricular arrhythmias in order to prevent their recurrence [16]. Amiodarone prolongs the effective refractory period blocking the re-entry circuits and terminates the ventricular arrhythmias produced through re-entry mechanism such as those developed in the case of myocardial ischemia [16]. This is why we prescribed amiodarone to our patient for secondary prevention.

In this case, the occlusion of the conus branch occurred as a complication during stent angioplasty of the RCA ostial lesion and led to RVOT acute ischemia manifested through ST-segment elevation in V1-V2 leads and two episodes of ventricular fibrillation with cardiac arrest successfully defibrillated.

Conclusions

In conclusion, the occlusion of the conus artery by different mechanisms such as atherosclerosis, vasospasm, plaque shift during percutaneous coronary intervention of the RCA ostial lesions, may lead to myocardial ischemia in the area of the

pulmonary artery trunk, that manifests through ST-segment elevation in precordial leads. These patients have a high risk of developing ventricular arrhythmias. Therefore, although the conus branch is a small artery, its occlusion can have significant life-threatening complications.

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