

Case Report

Coincidence or consequence? Single coronary artery in a dilated cardiomyopathy case

Catalina Paraschiv^{1,2}, Livia F. Paduraru^{1,2}, Serban M. Balanescu^{1,2}

- ¹ University of Medicine and Pharmacy Carol Davila, Bucharest 050474, Romania
- ² Elias University Hospital, Bucharest 011461, Romania
- * Corresponding author: Livia F. Paduraru, livia trasca@yahoo.com

CITATION

Paraschiv C, Paduraru LF, Balanescu SM. Coincidence or consequence? Single coronary artery in a dilated cardiomyopathy case. Cardiac and Cardiovascular Research. 2024; 5(1): 2868.

https://doi.org/10.54517/ccr.v5i1.2868

ARTICLE INFO

Received: 2 April 2024 Accepted: 27 May 2024 Available online: 28 June 2024

COPYRIGHT



Copyright © 2024 by author(s). Cardiac and Cardiovascular Research is published by Asia Pacific Academy of Science Pte. Ltd. This work is licensed under the Creative Commons Attribution (CC BY) license.

https://creativecommons.org/licenses/by/4.0/

Abstract: Single coronary artery is a rare congenital condition which entails one artery to supply blood to the entire myocardium. A case of single coronary artery in a patient diagnosed with tachycardia-induced cardiomyopathy is discussed. The focus of this case is to present the clinical implications of this coronary anomaly.

Keywords: coronary anomaly; single coronary artery; tachycardia-induced cardiomyopathy; atrial fibrillation; cardiomyopathy (List five to eight pertinent keywords specific to the article yet reasonably common within the subject discipline.)

1. Introduction

Single coronary artery (SCA) is a rare condition in which a unique artery, originating in the aortic trunk, supplies the myocardium through its branches. Patients with coronary anomalies may be symptomatic, or it can be an incidental finding during coronary angiography or during autopsies [1].

2. Case report

A 73-year-old man was admitted with severe dyspnea and palpitations. The patient had a history of hypertension, dyslipidemia and permanent atrial fibrillation (AF). Upon clinical examination, he had slightly elevated blood pressure (150/100 mmHg), a ventricular rate of 135 beats per min (bpm), basal crackles in both lungs could be heard. The electrocardiogram showed AF with rapid ventricular response, newly diagnosed left bundle branch block. The echocardiography described severe left atrial enlargement, a slightly dilated left ventricle (LV), severe systolic dysfunction with a left ventricular ejection fraction (LVEF) of 25%, due to global hypokinesia. The patient presented no dynamic changes in cardiac enzyme serum levels, nor increased inflammatory markers and the thyroid function was normal. The coronary angiography revealed a single right coronary artery (SCA), divided in two arteries (Figure 1A). The left coronary ostium could not be seen by aortography. The first branch of the single coronary artery runs along the right coronary artery's course. The left artery had an anomalous path, ascendant and parallel to the pulmonary trunk, describing a loop around it. Then, provided a hypoplastic left descending artery, following the anterior interventricular groove and continued towards the posterior wall through the left coronary sulcus it descended towards the anterior interventricular groove. In the medial portion of the anterior descending artery, a 50% stenosis was noticed. The Coronary Computed Tomography Angiography confirmed the anomalous coronary

artery and described a pre pulmonic path of the left branch (Figures 1B–D).

Figure 1. (A) Coronarography image and; **(B–D)** coronary computed tomography angiography images showing a single coronary artery originating in the right sinus of Valsalva and dividing into a right branch (blue arrow) and left branch (yellow arrow). The left artery provides a hypoplastic anterior interventricular artery (green arrow).

On the fourth day of hospitalization, the echocardiography showed LVEF of 40%, and on day 20 the LVEF enhanced to 50%. The symptomatology and LV function improved under standard heart failure and rate control treatment. He was started on angiotensin receptor inhibitor—ramipril 5 mg o.d., later uptitrated to 10 mg o.d., diuretics spironolactone 25 mg and furosemide (initially 80 mg per day i.v. and deescalated to 20 mg p.o. per day). The patient was on low dose beta blocker—25 mg metoprolol daily. The dose was gradually, over a 3-week period, uptitrated to 150 mg metoprolol daily.

3. Discussion

SCA is a congenital condition which entails one artery to supply blood to the entire myocardium. It has been described as a rare occurrence, with a prevalence ranging from 0.014% to 0.044% [1–3]. It has been reported as a single anomaly, or in an association with cardiac congenital abnormalities [2].

Following the Lipton Classification of SCA, in this case it's a RII-A type: the origin of the artery is in the right sinus, it branches into a right artery and a left artery and the left artery passes anterior to the pulmonary trunk. Also, SCA have been classified as having a benign course or a potentially serious one [3]. Consequently, the patients may be asymptomatic or they might present symptoms of ischemia, heart failure, syncope or even sudden cardiac death [1,3]. Myocardial ischemia was documented in a few cases in the absence of coronary artery disease [4,5] but also attributed to atherosclerosis [6,7]. An interarterial course, between the pulmonary trunk and the aorta, could result in coronary compression as the aortic root expands during physical effort [3,8]. A high risk of sudden cardiac death has been described in young people with coronary anomalies, especially exercise-related [9,10]. Other

anatomical features which could lead to ischemia are the following: narrowing of the artery, intramural segments, kinking, acute angle takeoff [8]. In post-mortem studies, some patients with coronary anomalies had areas of myocardial fibrosis due to ischemic episodes, which could be considered an arrhythmic substrate [8]. In our case, as the second artery passes anterior to the main vessels, and not between them, we consider the course as being benign. The coronary anomaly had no high-risk features for ischemia or sudden cardiac death thus allowing the patient to live with this coronary distribution for 73 years without complications.

Tachycardia-induced cardiomyopathy (TIC) is a type of systolic dysfunction which is reversible, partially or completely, once the heart rate is controlled [11,12]. The etiology of this condition is tachyarrhythmic—high ventricular rate, with AF being the most common cause [13,14]. High ventricular rates can lead to dilation of the cardiac chambers and to secondary mitral regurgitation [11,14]. In this case, the patient initially had a dilated LV and a severe mitral regurgitation. After 3 weeks of treatment, the LV returned to normal size, the systolic function improved and mitral regurgitation diminished from severe to moderate. We found the coexistence of a SCA in an elderly patient with TIC as a coincidence, and not as a cause of systolic dysfunction.

4. Conclusion

In conclusion, we discuss a case of what appeared to be, at first, a common association of heart failure with reduced ejection fraction and atrial fibrillation in an elderly patient. As the case developed, a rare coincidence was found: a case of tachycardia-induced cardiomyopathy in a patient with a benign coronary anomaly.

Author contributions: Conceptualization, CP and LFP; methodology, CP; software, LFP; validation, CP, LFP and SMB; formal analysis, CP; investigation, CP; resources, LFP; data curation, LFP; writing—original draft preparation, CP; writing—review and editing, LFP; visualization, CP and LFP; supervision, SMB; project administration, SMB; funding acquisition, SMB. All authors have read and agreed to the published version of the manuscript.

Conflict of interest: The authors declare no conflict of interest.

References

- 1. Akcay A, Tuncer C, Batyraliev T, et al. Isolated Single Coronary Artery A Series of 10 Cases. Circulation Journal. 2008; 72(8): 1254-1258. doi: 10.1253/circj.72.1254
- 2. Lipton MJ, Barry WH, Obrez I, et al. Isolated Single Coronary Artery: Diagnosis, Angiographic Classification, and Clinical Significance. Radiology. 1979; 130(1): 39-47. doi: 10.1148/130.1.39
- 3. Yamanaka O, Hobbs RE. Coronary artery anomalies in 126,595 patients undergoing coronary arteriography. Catheterization and Cardiovascular Diagnosis. 1990; 21(1): 28-40. doi: 10.1002/ccd.1810210110
- 4. Muhyieddeen K, Polsani VR, Chang SM. Single right coronary artery with apical ischaemia. European Heart Journal Cardiovascular Imaging. 2012; 13(6): 533-533. doi: 10.1093/ehjci/jes013
- 5. Melman YF, Cutlip DE, Das S. A 54-Year-Old Woman with a Single Coronary Artery and Watershed Ischemia Treated with Nitrates. JACC: Cardiovascular Interventions. 2015; 8(6): e91-e94. doi: 10.1016/j.jcin.2014.12.248

- 6. Raddino R, Pedrinazzi C, Zanini G, et al. Percutaneous coronary angioplasty in a patient with anomalous single coronary artery arising from the right sinus of Valsalva. International Journal of Cardiology. 2006; 112(3): e60-e62. doi: 10.1016/j.ijcard.2006.02.032
- 7. Jahnke C, Nagel E, Ostendorf PC, et al. Diagnosis of a "Single" Coronary Artery and Determination of Functional Significance of Concomitant Coronary Artery Disease. Circulation. 2006; 113(9). doi: 10.1161/circulationaha.105.564260
- 8. Finocchiaro G, Behr ER, Tanzarella G, et al. Anomalous Coronary Artery Origin and Sudden Cardiac Death. JACC: Clinical Electrophysiology. 2019; 5(4): 516-522. doi: 10.1016/j.jacep.2018.11.015
- 9. Pelliccia A, Sharma S, Gati S, et al. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease. European Heart Journal. 2020; 1–80.
- 10. Basso C, Maron BJ, Corrado D. Clinical Profile of Congenital Coronary Artery Anomalies with Origin from the Wrong Aortic Sinus Leading to Sudden Death in Young Competitive Athletes. National Library of Medicine, 2000; 35(6): 5–9.
- 11. Gupta S, Figueredo VM. Tachycardia mediated cardiomyopathy: Pathophysiology, mechanisms, clinical features and management. International Journal of Cardiology. 2014; 172(1): 40-46. doi: 10.1016/j.ijcard.2013.12.180
- 12. Shoureshi P, Tan AY, Koneru J, et al. Arrhythmia-Induced Cardiomyopathy. Journal of the American College of Cardiology. 2024; 83(22): 2214-2232. doi: 10.1016/j.jacc.2024.03.416
- 13. Raymond-Paquin A, Nattel S, Wakili R, et al. Mechanisms and Clinical Significance of Arrhythmia-Induced Cardiomyopathy. Canadian Journal of Cardiology. 2018; 34(11): 1449-1460. doi: 10.1016/j.cjca.2018.07.475
- 14. Huizar JF, Ellenbogen KA, Tan AY, et al. Arrhythmia-Induced Cardiomyopathy. Journal of the American College of Cardiology. 2019; 73(18): 2328-2344. doi: 10.1016/j.jacc.2019.02.045