Annals of Clinical Case Reports

Case Report
Published: 02 May, 2022

An Unexpected Cause of Chest Pain in a Patient with Closed Atrial Septal Defect

*Bermudez-Gonzalez JL[1,2], Villa-Ramírez CA[1,2], Bautista-Pérez-Gavilán A[1,2], Saenz-Ancira S[1,3], Garcia-Cardenas M[1], Gonzalez-Hernandez MA[1], Ibarra-Moreno A[1], Perez-Partida AM[1], Arce-Sandoval CR[2], Cuéllar-Vargas J[2], Espinola-Zavaleta N[1] and Alexanderson-Rosas E[1,2]**

[1]Department of Nuclear Cardiology, National Institute of Cardiology Ignacio Chavez, Mexico

[2]National Autonomous University of Mexico, Mexico

[3]La Salle University, Mexico

Abstract

Atrial Septal Defects (ASD) represent the most common Congenital Heart Diseases (CHD) in adults and are usually treated with the septal amplatzer occluders. Complications usually arise due to mechanical problems of the device and are traditionally investigated, nevertheless, other chest pain etiologies should be considered, as data regarding late complications like angina, are scarce and insufficient. We present the case of a 70 year-old woman with a closed ASD and coronary microvascular disease diagnosed during follow-up with gated Positron Emission Tomography (PET). Late complications associated with Amplatzer occluder devices are generally derived from a mechanical impairment of the device per se. The case presented opens the door for further research questions regarding microvascular complications arising from occluding devices.

Keywords: Atrial septal disease; Congenital heart disease; Amplatzer septal occlude; Angina; Chest pain; Microvascular disease

OPEN ACCESS

Correspondence:
Erick Alexanderson-Rosas, Department of Nuclear Cardiology, National Institute of Cardiology Ignacio Chavez, Juan Badiano N°1, Colonia Seccion XVI, Tlalpan, PC 14030, Mexico City, Mexico, Tel: +52-5554-529760; E-mail: eamedicinanuclear@gmail.com

Received Date: *18 Mar 2022*
Accepted Date: *27 Apr 2022*
Published Date: *02 May 2022*

Citation:
Bermudez-Gonzalez JL, Villa-Ramírez CA, Bautista-Pérez-Gavilan A, Saenz-Ancira S, Garcia-Cardenas M, Gonzalez-Hernandez MA, et al. An Unexpected Cause of Chest Pain in a Patient with Closed Atrial Septal Defect. Ann Clin Case Rep. 2022; 7: 2175.

ISSN: 2474-1655

Copyright *© 2022 Alexanderson-Rosas E. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.*

Learning Objectives

- To understand the benefits of percutaneous approach versus a traditional surgical closure of atrial septal defects.
- To properly assess chest pain during follow-up after an atrial septal defect closure.
- To consider microvascular coronary disease as a common cause of angina in aged patients with diabetes and hypertension.

History of Presentation

A 70-year-old woman presented to our Nuclear Cardiology Department for myocardial perfusion evaluation due to occasional chest pain from two months of onset and functional dyspnea class deterioration (NYHA II-III). She has a medical history of an ASD corrected four years ago with an Amplatzer device; Systemic Arterial Hypertension (SAH) and diabetes mellitus type II (DM II). She denied previous episodes of syncope of minimal effort dyspnea (NYHA III).

Physical Examination

Vital signs presented a heart rate of 75 bpm, a blood pressure of 114/78 mmHg, a respiratory rate of 15 rpm and an oxygen saturation of 93%. She did not present ankle edema, orthopnea or other clinical signs of heart failure. Thoracic auscultation was unremarkable.

Differential Diagnosis

In adult patients with a corrected ASD, angina may present due to mechanical or functional alterations of the device. First, we aimed to rule out cardiac erosion or perforation, due to the high mortality it confers. Nevertheless, coronary artery disease ought to be considered in a patient this age and with a history of SAH and DM II.

Investigations

The patient had a resting ECG at admission with a sinus rhythm, heart rate of 75 bpm, QRS axis at 0°, and an ST depression in V6; without further relevant findings. A PET study with

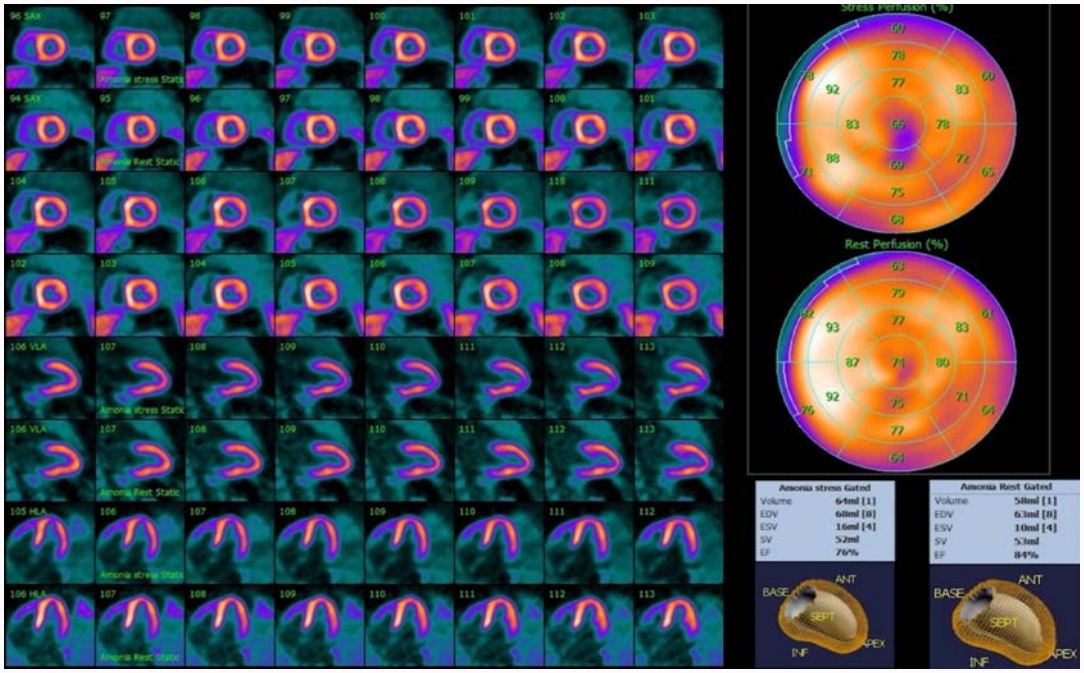


Figure 1: PET myocardial perfusion study with 13N-ammonia exhibits mild reversible ischemia at the apex.

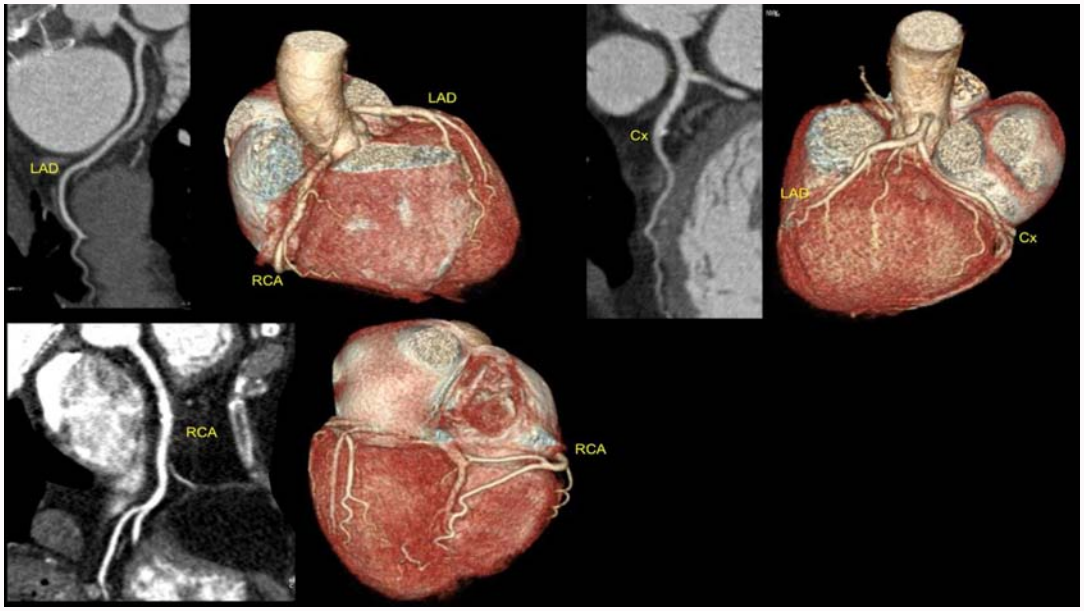


Figure 2: Coronary computed tomography angiography with coronary calcium score of 29.8U. Left anterior descending artery at its mid portion shows a mild obstruction (CAD-RADS2), and the right coronary artery exhibits two mild obstructive lesions (CAD-RADS2).
LAD: Left Anterior Descending Artery; RCA: Right Coronary Artery; Cx: Circumflex Artery

N13-Ammonia was performed revealing adequate perfusion in most segments (Figure 1), only mild ischemia in the apex region, and a Left Ventricular Ejection Fraction (LVEF) reduction of 7% at stress. Afterward, coronary calcium score was calculated at 29.8 (CAC-RDS 1), with calcium mainly in the Left Anterior Descending (LAD) artery and Right Coronary Artery (RCA). Moreover, extra coronary calcium was observed, notably in the thoracic aorta (1029.8 U) and the aortic valve (60.5 U). Coronary angiotomography revealed the presence of atheromatous lesions in the epicardial coronary arteries (Figure 2), one in the midsegment of the LAD artery, with a non-significant obstruction of 25% to 49% and two lesions in the union of the proximal and vertical segments of the RCA, both with an estimated obstruction of 25% to 49%. Additionally, the Amplatzer device was adequately observed in the interauricular septum (Figure 3). However, coronary blood flow quantification by PET demonstrated reduced myocardial blood flows and impaired myocardial flow reserve in all territories, with a global myocardial flow reserve of 1.57 (Figure 4). Thus, the patient was diagnosed with coronary microvascular disease, also known as microvascular angina.

Management

The patient was discharged with aspirin 100 mg qd, atorvastatin

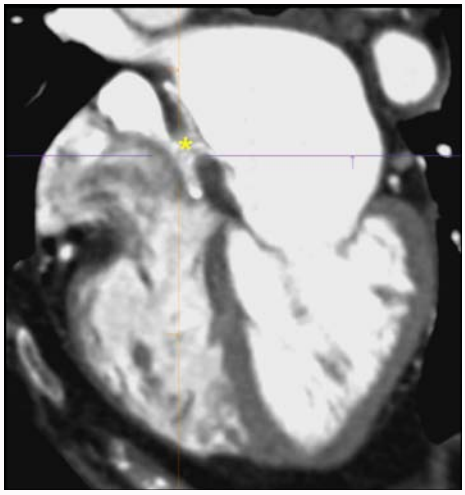


Figure 3: Computed cardiac tomography shows the Amplatzer device in adequate position (*).

60 mg daily and losartan 50 mg qd.

Discussion

Atrial Septal Defects (ASD) represent the most common Congenital Heart Disease (CHD) in adults, with a prevalence of 1 in every 1,500 live births, accounting for approximately 7% of all CHD [1-3].

Numerous adults are asymptomatic [1,4]; nonetheless, most patients will eventually develop symptoms [1], such as exercise intolerance [1,4], palpitations, and a soft systolic crescendo-decrescendo outflow tract murmur with a fixed split in S2 [4].

In the last decades, the percutaneous closure approach has gained popularity due to several advantages in comparison to the traditional surgical approach, such as the avoidance of a cardiopulmonary bypass, shorter hospitalization time, lower incidence of post-procedural complications and a lower cost [3,4]. This has resulted in the manufacture of several devices, of which the Amplatzer Septal Occluder (ASO) is the most commonly used [4-6].

The percutaneous approach has a post-procedural complication risk of 7.2%, approximately a third of postoperative complications risk (24%) [4]. Device thrombosis and cardiac erosion are the most important late complications, whereas atrial arrhythmias remain the most prevalent [3].

In the appearance of chest pain during follow-up after a transcatheter ASD closure, one of the main objectives is to discard cardiac erosion, which occurs with an incidence of 0.1% and is commonly related to a deficient aortic rim. [5,7]. Dibardino et al. demonstrated that cardiac erosion/perforation/rupture was the second most common adverse event (22.9%), only after device embolization (51%). The importance of this phenomenon relies on a relatively high mortality, being the most frequent lethal complication [3].

Another uncommon cause of angina in patients with CHD is due to the compression of the left coronary artery between a dilated pulmonary artery and the aorta, in the context of Pulmonary Hypertension (PH). This phenomenon occurs in 5% to 10% of patients with CHD [8].

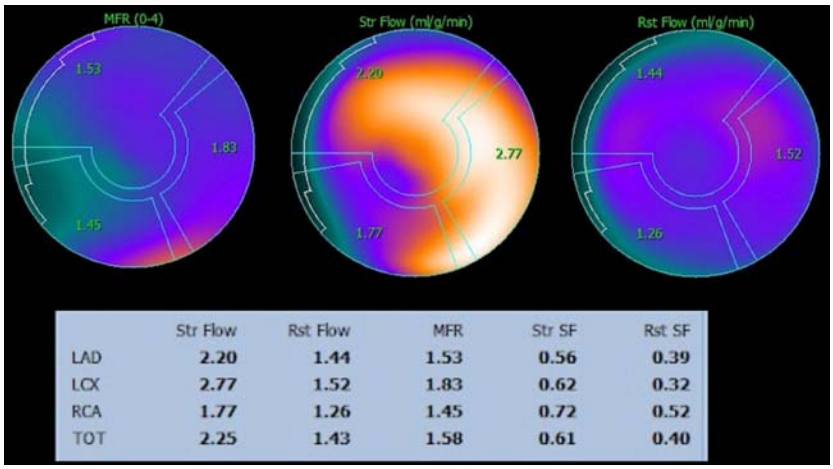
In this report, we describe the case of a patient who presented with angina 4 years after the percutaneous closure of an ASD. Device malposition was excluded through echocardiography. The absence of significant atheromatous plaques in the coronary arteries and a diminished coronary flow reserve at 1.57 pointed to the diagnosis of Coronary Microvascular Disease (CMD) related to an abnormality in microcirculation, thus resulting in an inadequate vasodilatory response and a deficient augmentation of blood flow by the microvasculature in response to stress [9,10]. The prognostic significance of this association is still unclear. Hereupon, the consideration of CMD must be taken into account when evaluating patients with similar clinical characteristics.

Follow-up

The patient is yet to present at our clinic for a follow-up appointment.

Conclusion

Percutaneous closure of an ASD is a safe and effective procedure, reducing the burden of the traditional surgical approach. Despite its widespread acceptance, few data regarding the complications during follow-up are available. The presence of chest pain after the implantation of an ASO device has traditionally merited the



	Str Flow	Rst Flow	MFR	Str SF	Rst SF
LAD	2.20	1.44	1.53	0.56	0.39
LCX	2.77	1.52	1.83	0.62	0.32
RCA	1.77	1.26	1.45	0.72	0.52
TOT	2.25	1.43	1.58	0.61	0.40

Figure 4: Quantified myocardial blood flows determined by PET demonstrate abnormally reduced myocardial flow reserve at the three coronary territories, a characteristic finding of microvascular disease.

investigation of the device.

Embolization and cardiac erosion. Nevertheless, it is important to consider additional causes, such as coronary microvascular disease, which has gained the attention of cardiologists in recent decades. The outcome of this association remains to be elucidated.

Acknowledgement

This manuscript would not have been written without the Nuclear Medicine Investigation Unit of the National Institute of Cardiology Ignacio Chavez. The data compilation and redaction of the article is the result of a conjoint effort of all the authors. We want to send a special acknowledgement to Dr. Mauricio Garcia Cardenas, resident of the National Cardiology Institute for providing the interpretation of the PET and computed tomography images.

References

1. Akagi T. Current concept of transcatheter closure of atrial septal defect in adults. J Cardiol. 2015;65(1):17-25.
2. Alizadehasl A, PirBorj NM, Sadeghpour A, Firouzi A, Sanati H, Movassaghi M. The frequency and importance of chest pain in midterm follow up of transcatheter closure of interatrial septal defect. J Cardiovasc Thorac Res. 2017;9(4):215-20.
3. Jalal Z, Hascoet S, Baruteau A, Iriart X, Kreitmann B, Boudjemline Y, et al. Long-term complications after transcatheter atrial septal defect closure: A review of the medical literature. Can J Cardiol. 2016;32(11):1315.e11-8.
4. Bradley EA, Zaidi AN. Atrial septal defect. Cardiol Clin. 2020;38(3):317-24.
5. Figueras-Coll M, Martí-Aguasca G, Betrián-Blasco P. Chronic chest pain after percutaneous atrial septal defect closure. Cardiol Young. 2021;31(4):671-2.
6. Stout KK, Daniels CJ, Aboulhosn JA, Bozkurt B, Broberg CS, Jack M, et al. 2018 AHA/ACC Guideline for the management of adults with congenital heart disease: A report of the American College of Cardiology/American Heart Association Task Force on clinical practice guidelines. Circulation. 2019;139(14):726-9.
7. O'Byrne ML, Levi DS. State-of-the-art atrial septal defect closure devices for congenital heart. Interv Cardiol Clin. 2019;8(1):11-21.
8. Pascall E, Tulloh RM. Pulmonary hypertension in congenital heart disease. Future cardiol. 2018;14(4):343-53.
9. Sinha A, Rahman H, Perera D. Coronary microvascular disease: Current concepts of pathophysiology, diagnosis and management. Cardiovasc Endocrinol Metab. 2020;10(1):22-30.
10. Taqueti VR, Carli MFD. Coronary microvascular disease pathogenic mechanisms and therapeutic options: JACC State-of-the-Art Review. J Am Coll Cardiol. 2018;72(21):2625-41.