https://doi.org/10.35839/repis.4.2.618

The syncope as a debut form of mitral stenosis

Síncope como forma de debut de la estenosis mitral

Rodolfo Vega-Candelario[1,a,*]

Abstract

Introduction: The common mechanism to the syncope is a transient alteration in cerebral blood flux, this fact generate a temporary decrease in the brain metabolism with involvement of the ascending reticular system and cerebral cortex. The syncope due to mitral stenosis does not appear in the reviewed literature.
Objectives: To show a clinical case from a patient with mitral stenosis that debuts with recurrent syncope.
Clinical case: A 45-year-old patient, mestizo without history of disease, a farmer, is admitted to the internal medicine ward for recurrent syncope. He was evaluated by a neurologist and a cardiologist; he was diagnosed with critical mitral stenosis with severe pulmonary hypertension and a right ventricular Systo-diastolic dysfunction. After the mitral valve replacement by cardiovascular surgery, he did not suffer syncopal episodes again and leads a normal biopsychosocial and work life.
Comments: The critical mitral stenosis causes decreased in the ventricular filling and decreased the cardiac output with decreased cerebral blood flux, accentuated by the severe pulmonary hypertension and the right heart failure. This fact caused repetitive syncope.

Keywords: syncope, mitral stenosis, pulmonary hypertension, cardiac output, cerebral blood flux.

Resumen

Introducción: El mecanismo común a todo síncope es una alteración transitoria del flujo sanguíneo cerebral, que provoca una disminución momentánea del metabolismo cerebral, con afectación del sistema reticular ascendente y córtex cerebral. El síncope por estenosis mitral, no aparece en la literatura revisada.
Objetivos: Presentar un paciente con estenosis mitral que debuta con cuadro de síncope recurrente.
Caso clínico: Paciente de 45 años, mestizo, sin antecedentes de enfermedad, trabajador agrícola, ingresa en sala de medicina interna por padecer síncopes recurrentes. Fue valorado por neurólogo y cardiólogo; se diagnosticó estenosis mitral crítica con hipertensión pulmonar grave y disfunción sistodiastólica de ventrículo derecho. Luego de la sustitución valvular mitral por cirugía cardiovascular no volvió a padecer episodios sincopales y lleva una vida normal biopsicosocial y laboral.
Comentarios: La estenosis mitral crítica ocasiona disminución del llenado ventricular y disminución del gasto cardiaco con disminución del flujo sanguíneo cerebral, acentuado por la hipertensión pulmonar grave e insuficiencia cardíaca derecha. Ocasionó síncopes repetitivos.

Palabras clave: síncope, estenosis mitral, hipertensión pulmonar, gasto cardiaco, flujo sanguíneo cerebral.

[1]Assistant Professor at the Faculty of Medical Sciences of Ciego de Avila, Cuba.

[a]Cardiologist

ORCID:
*https://orcid.org/0000-0003-4459-8350

Corresponding author:
Rodolfo Vega Candelario
Address: Provincial Teaching Hospital ¨Roberto Rodríguez Fernández, Morón, Ciego de Ávila, Cuba.

Email: rvc_50@infomed.sld.cu

Reception date: 02 de febrero de 2020

Approval date: 18 de marzo de 2020

Quote as: Vega-Candelario R. Síncope como forma de debut de la estenosis mitral. Rev. Peru. Investig. Salud. [Internet]; 4(2): 83-86. Available from: http://revistas.unheval.edu.pe/index.php/repis/article/view/618

Introduction

The common mechanism to the syncope is a transient alteration in cerebral blood flux, this fact generate a temporary decrease in the brain metabolism with involvement of the ascending reticular system and cerebral cortex, which causes a decrease in the level of consciousness. Based on previous experiences in the tilting - table test, the drop in mean arterial pressure below 60 mmHg causes an alteration of the self-regulating mechanism of the cerebral flux with the consequent appearance of syncope. If this loss of brain flux is maintained above a critical time, around 5 minutes, it can cause irreversible neurological damage. The causes of the syncope are numerous; however, it is necessary to make a differential diagnosis with other causes of decreased consciousness, which are not considered syncope, such as the vertebrobasilar transient ischemic attacks (TIA), metabolic disorders such as hypoglycemia, hypoxia, poisoning, etc. There are two types of syncope: the syncope of cardiovascular origin and the neurally - mediated syncope (1-4).

The treatment of the syncope must be oriented to solving the underlying cause and avoiding the recurrences. The cardiogenic syncope should be treated by the specialists, with specific treatments (pacemakers, defibrillators, drugs, surgery and cardiac intervention). In the cases to the neurally - mediated syncope, the postural and hygienic measures should be the first therapeutic step. Other treatments (drugs, pacemakers) have not shown great benefits (4-9).

There are many causes of syncope to be known, but the mitral stenosis does not appear in any, less as the unique symptom. This debut is frequent in the aortic stenosis, in primary and secondary cardiac tumors, in second and third degree atrioventricular blocks, in the sinus node disease, cardiac arrhythmias (especially ventricular), the cardiac tamponade, the primary pulmonary hypertension, the Brugada syndrome, the long QT syndrome, but not in the mitral stenosis (9-13).

The syncope of cardiovascular origin (14-16) is frequent (from 3 to 5% of emergencies attended; from 1 to 2% of hospital admissions).

The 20% of the population suffers from syncope at least one time in their life. The prognosis is highly variable and it is according with the cause. Those of cardiac origin present an estimated mortality between 18% and 33% annually. The main predictor of mortality in the patients with syncope is the presence of structural heart disease (10).

The clinical case described in this article show a patient to be admitted with clinical characteristics of syncope without family pathological history or personal heart disease, no symptoms to remember it.

The objective of this clinical case is to show a patient with mitral stenosis who debuts with recurrent syncope.

Clinical case

A 45-year-old patient, mestizo without history of disease, no harmful habits, farmer, who during 6 months he was suffered from syncope, with total loss of consciousness.

The family pointed that the generalized tonic - clonic seizures last from 30 to 35 minutes, this including the seizure postictal period of the syncope, then the patient recovers, remembers nothing and continues to work, in most opportunities related to physical effort and rarely unrelated to it.

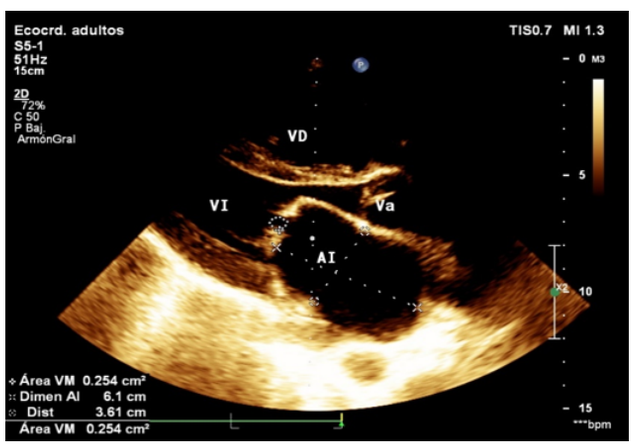
The patient was examined by the doctor, the neurologist and the cardiologist, posteriorly a simple and a contrasted computed tomography (CT) of the skull, electrocardiogram, electroencephalogram, telecardiogram and complete hematological study were performed him. The results were normal. After a careful questioning and a detailed physical examination, a critical mitral stenosis is diagnosed with severe pulmonary systolic hypertension. A color Doppler echocardiogram was performed him and a right ventricular systo-diastolic dysfunction was found. Due to this, it was logical to consider the above as the cause of syncope.

The use of phosphodiesterase - 5 inhibitors in cardiac surgery, with pulmonary arterial hypertension, is being studied due to the need to prevent the postoperative complications and the appearance of right ventricular (RV) failure (15-18); that was already present in the patient. It was started with a dose of 50 mg daily and increased to 50 mg every 8 hours, depending on the echocardiographic result. After 6 months, he underwent to a surgery with open-heart mechanical mitral valve replacement in the Cardiology Institute and Cardiovascular Surgery of Cuba. The postoperative evolution was excellent. After the postoperative period, he was kept under pharmacological treatment with 150 mg of sildenafil for 6 months, he stayed asymptomatic, without syncope, and additionally he received the administration of warfarin, with INR (International Normalized Ratio) between 2 and 3.

Comments

Although the mitral stenosis is not related as a cause of syncope, this patient suffered syncope, with recurrent generalized tonic-clonic seizures.

In this patient, the mitral stenosis (Fig. 1,2 ,3) could have been the cause of syncope, due to the degree of stenosis and its repercussions on the pulmonary arterial pressure, the tricuspid regurgitation and the right heart failure (15-18).



Legend: LA (left atrium); LV (left ventricle); AV (healthy aortic valve); RV (right ventricle).

Fig. 1 Two-dimensional echocardiogram on the left parasternal long axis. It shows a large atrium, with difficulty to open the mitral valve. The mitral valve area is 0.25 cm2, the same as in the short axis.

Pathophysiologically the critical mitral stenosis causes decreased left ventricular filling. This is due to a mechanical difficulty at the level of the mentioned valve to the passage of the blood coming from the left atrium and therefore, a decreased cardiac output. Besides, the secondary severe pulmonary hypertension causes a decrease in the gas exchange,

hypoxemia due to increase in the resistance and fibrosis of the hematoalveolar barrier that causes a vicious circle and as such can cause syncope. The consequences of the right heart failure, the tricuspid insufficiency and the hematoalveolar perfusion difficulty constitute a multifactorial scheme that explains the decrease in the cerebral flux, with dramatic consequences at the level of the reticular substance, the cerebral cortex and the justification of the cardiac syncope due to the critical mitral stenosis (15-18) (Fig. 1,2). The pulmonary arterial systolic hypertension (57 mmHg) contributes to the seizure in the recurrent syncope (Fig. 3)

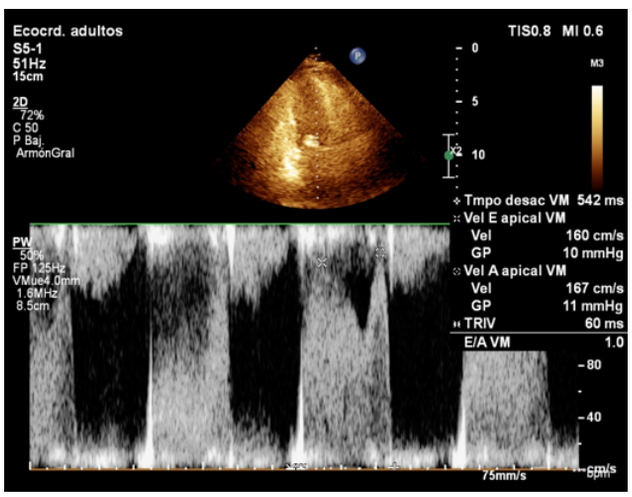


Fig. 2 A Doppler echocardiogram of the mitral valve (MV). It shows the auscultatory elements of cardiac cycle changes in critical mitral stenosis

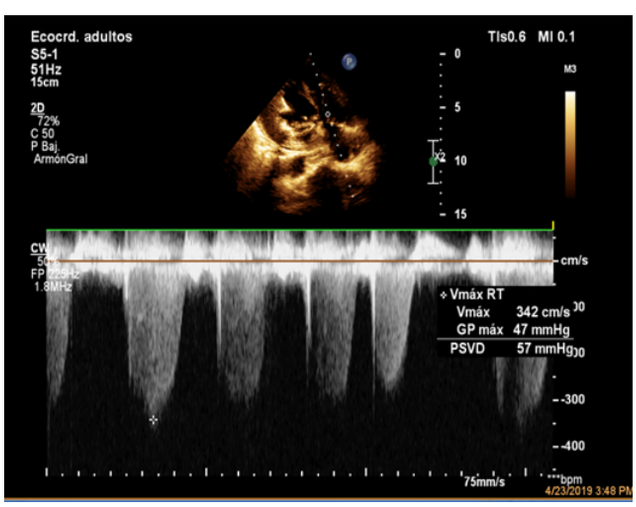


Fig. 3 The axis of 4 cameras from the apex of the Doppler echocardiogram. An elevation of pressures is observed in the right ventricle that infers severe pulmonary hypertension: Maximum pressure gradient of 47 mmHg and Systolic pressure of the Right Ventricle of 57 mm Hg

The Sildenafil was effective on the severe pulmonary arterial hypertension and with effective pre and post-surgical efficacy of the acquired heart disease (critical mitral stenosis) (15-18).

References

1. Lacunza Ruiz FJ, García Alberola JR, Gimeno Blanes M, Valdés M. Síncope. Unidades de Síncope y Arritmias. Medicine. 2005[acceso: 12/11/2019]; 9(37):2447-54. Disponible en: http://www.sciencedirect.com/science/article/pii/S0211344905737425/
2. Moya Mitjans A, Rivas Gandara N, Sarrias Merce A, Pérez Rodón J, Roca Luque I. Puesta al día: Arritmias (VIII), Síncope. Rev. Esp Cardiol. 2012 [acceso: 12/11/2019]; 65(8):755–65. Disponible en: https://www.revespcardiol.org/es-pdf-S0300893212001947
3. Matthew JR, David E, Andrew J, Robin RJ, Prescott KG, Keith GJ, Gray R. The ROSE (risk stratification of syncope in the emergency department) study. Journal of the American College of Cardiology. 2010 [acceso: 15/11/2019]; 55(8) 713-21. Disponible en: http://www.onlinejacc.org/content/accj/55/8/713.full.pdf
4. Tretter JT, Rae Ellen WK. Distinguiendo el síncope cardiaco del síncope. J Pediatr. 2013 [acceso: 2/12/2019]; 163: 1618-23. Disponible en: https://www.jpeds.com/article/S0022-3476(13)00881-0/pdf
5. Azocar D, Ruiz Granell R, Ferrero A, Martínez Brotons A, Izquierdo M, Domínguez E, et al. Síncope y bloqueo de rama. Rendimiento del uso escalonado del estudio electrofisiológico y de la monitorización electrocardiográfica prolongada. Rev. Esp Cardiol. 2011 [acceso: 12/12/2019]; 64(3): 213–9. Disponible en: http://www.revespcardiol.org/es/content/articulo/90000664/
6. Moya A, Sutton R, Ammirati F, Blanc JJ, Brignole M, Johannes BD, et al. Guía de práctica clínica para el diagnóstico y manejo del síncope (versión 2009). Rev Esp Cardiol. 2009 [Acceso: 20/12/2019]; 62(12): 1466. Disponible en: https://www.revespcardiol.org/es-pdf-S0300893210000825
7. Shen WK, Decker WW, Smars PA, Goyal

DG, Walker AE, Hodge DO, et al. Syncope Evaluation in the Emergency Department Study (SEEDS): a multidisciplinary approach to syncope management. Circulation. 2004 [acceso: 25/12/2019]; 110: 3636-45. Disponible en: https://www.ahajournals.org/doi/pdf/10.1161/01.CIR.0000149236.92822.07

8. Farwell DJ, Sulke AN. Does the use of a syncope diagnostic protocol improve the investigation and management of syncope? Heart. 2004 [acceso: 27/12/2019]; 90(1): 52-8. Disponible en: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1768011
9. Ammirati F, Colivicchi F, Santini M. Diagnosing syncope in clinical practice: implementation of a simplified diagnostic algorithm in a multicenter prospective trial. Our Heart J. 2000 [access: 28/12/2019]; 21(11): 935-40. Disponible en: https://www.revespcardiol.org/es-pdf-13145649
10. Brignoles M, Ungar A, Bartoletti A, Ponassi I, Lagi A, Mussi C, et al. Standardized-care pathway vs. usual management of syncope patients presenting as emergencies at general hospitals. Euro pace. 2006 [acceso: 30/12/2019]; 8(8): 644-50. Disponible en: http://asmn.netribe.it/asmnbiblio2015/pubblicazioni/Europace%202006.pdf
11. Rodríguez Entem F, González Enríquez S, Juan J, Olalla A, Cobo Beláustegui M, Expósito García V, et al. Manejo del síncope en el servicio de urgencias sin ingreso hospitalario. Rev. Esp Cardiol. 2008 [acceso: 04/01/2020]; 61(1): 22-8. Disponible en: https://www.revespcardiol.org/es-pdf-13114953
12. Moya A, Rivas N, Pérez Rodón J, Roca I, García Dorado D. El síncope: un problema con mayúsculas. Rev. Esp Cardiol Supl. 2010 [acceso: 6/01/2020]; 10(Supl.1):53A-59ª. Disponible en: https://www.sciencedirect.com/science/article/abs/pii/ S11313587 107 01126
13. Prieto Salcedo ML, Pérez López AM. Protocolos de Cardiología Síncopes. Bol. Pediatr. 2006 [acceso: 13/01/2020]; 46 (Supl 2): 281-5.Disponible en: http://www.sccalp.org/boletin/46_supl2/BolPediatr2006_46_supl2_281-285.pdf
14. Ramakrishna G, Sprung J, Ravi B, Chandrasekaran K, McGoon M. Impact of pulmonary hypertension on the outcomes of noncardiac surgery: Predictors of perioperative morbidity and mortality. J Am Coll Cardiol. 2005[acceso: 15/11/2020]; 45(10):1691-9. : Disponible en: https://www.sciencedirect.com/science/article/pii/S0735109705004961?via%3Dihub
15. Galie N, Olschewski H, Oudiz R, Torres F, Frost A. Ambrisentan for the treatment of pulmonary arterial hypertension. Circulation. 2008 [acceso: 12/01/2019]; 117(23): 3010-9. Available from: https://www.ncbi.nlm.nih.gov/pubmed/18506008
16. Lewis G, Lachmann J, Camuso J, Lepore J, Shin J, Martinovic M, et al. Sildenafil improves exercise hemodynamics and oxygen uptake in patients with systolic heart failure. Circulation. 2007 [acceso: 15/01/2020]; 115(1): 59-66. Disponible en: https://pdfs.semanticscholar.org/2f04/9790063265fb62706efd30573fada2ac0af3.pdf
17. Hemnes A, Champion H. Right heart function and hemodynamics in pulmonary hypertension. Int J Clin Pract 2008 [acceso: 18/01/2020]; 62(s160): 11-9. Disponible en: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1742-1241.2008.01812.x
18. Falk V, Bax JJ, De Bonis M, Hamm C, Holm H, Lung B. Guía ESC/EACTS 2017 sobre el tratamiento de las valvulopatías. Rev Esp Cardiol. 2018; 71(2): 110.e1-e47. Disponible en: https://www.revespcardiol.org/es-pdf-S0300893217308096