Sociedad Cubana de Cardiología

Case reports

Tachycardia with wide QRS complexes during a transesophageal echocardiogram

Taquicardia con complejos QRS anchos durante un ecocardiograma transesofágico

Sandra Alfonso Fernández, MD[1], Margarita Dorantes Sánchez, MD[2]✉, Wilfredo Roque Morán, MD[2], Sheila Hechavarría Pouymiró, MD[2]

[1]Hospital General Universitario Vladimir Ilich Lenin, Holguín, Cuba
[2]Instituto de Cardiología y Cirugía Cardiovascular, La Habana, Cuba

ARTICLE NFORMATION

Recibido: 04/02/2026
Aceptado: 06/03/2026

Competing interests:
The author declare no competing interests.

Corresponding author:
Margarita Dorantes Sánchez, MD
dorantes@infomed.sld.cu

Article category:
Clinical cardiology and risk factors

ISSN: 2078-7170
RNPS: 2235-145

Abstract: We present the case of a female patient admitted to the Arrhythmia and Pacemaker Service of the *Instituto de Cardiología y Cirugía Cardiovascular*, with a diagnosis of atrial flutter with variable atrio-ventricular conduction. During a transesophageal echocardiogram she developed a tachycardia with wide QRS with significant hemodynamic instability. She was initially treated with electrical cardioversion which restored sinus rhythm. Subsequently, radiofrequency ablation of the atrial flutter was performed. This case highlights an unusual and significant complication that may occur during transesophageal echocardiography leading to patient decompensation from the hemodynamic standpoint, which diagnosis is under discussion.
Key words: Atrial flutter; 1:1 Atrio-ventricular conduction; Transesophageal echocardiogram; Ventricular tachycardia
Resumen: Se presenta el caso de una paciente ingresada en el Servicio de Arritmias y Marcapasos del Instituto de Cardiología y Cirugía Cardiovascular, con diagnóstico de flutter auricular con conducción auriculoventricular variable, quien durante la realización de un ecocardiograma transesofágico, desarrolló una taquicardia con QRS ancho con gran inestabilidad hemodinámica. Se trató inicialmente con cardioversión eléctrica, con recuperación del ritmo sinusal, posteriormente se realizó ablación con radiofrecuencia del flutter auricular. Se destaca una complicación inusual e importante que puede presentarse durante la realización de la ecocardiografía transesofágica, cuyo diagnóstico se debate y descompensa a la paciente desde el punto de vista hemodinámico.
Palabras clave: Flutter auricular; Conducción auriculoventricular 1:1; Ecocardiograma transesofágico; Taquicardia ventricular

Introducción:

Transesophageal echocardiography (TEE) is a fundamental diagnostic tool in assessing multiple cardiovascular diseases. It is considered a safe procedure, with a low incidence of major complications; however, it is not without adverse events which can compromise the patient's stability requiring immediate intervention[1].

Among the complications described for TEE those related to oropharyngeal and esophageal trauma are predominant; in contrast, the appearance of significant arrhythmias during the procedure is infrequent and poorly reported in literature[2].

The aim of this report is to present a clinical case that, unusually, manifests a tachycardia with wide QRS complex (TWQRSC) during a TEE, representing an important diagnostic and therapeutic challenge.

CASO CLÍNICO

A 51-years-old white female patient with a past medical history of arterial hypertension, treated with losartan 50 mg every 12 hours and spironolactone 25 mg daily. She had no relevant family history, no substance uses and no known allergies. She was admitted to the Arrhythmia and Pacemaker Service for the evaluation of palpitation episodes lasting two months.

The patient reported that these episodes had a sudden onset, occurred at any time of day, were unrelated to physical exertion and were accompanied by dyspnea and chest tightness that had increased in frequency and duration over the past week.

Physical examination revealed blood pressure of 100/60 mmHg, heart rate of 90 beats per minute, arrhythmic heart sounds on auscultation, normal vesicular respiratory murmur, no rales and oxygen saturation of 98%.

The electrocardiogram (ECG) showed typical counterclockwise atrial flutter, with negative F waves in leads DII, DIII, and aVF and variable 2:1, 3:1 atrio-ventricular conduction (**Figure 1**); as well as intermittent atrio-ventricular dissociation, wide QRS complexes with right bundle branch block morphology and some isolated wide complexes with left bundle branch block morphology.

Complementary blood tests were performed which showed normal values.

The transthoracic echocardiogram showed a left ventricle ejection fraction in 45% by the Simpson`s method, with moderate remodeling of that ventricle, concentric hypertrophy, biventricular systolic dysfunction (moderate of left ventricle and mild of right ventricle), regional segmental contractility abnormalities predominantly in the inferior wall, elevated pulmonary capillary pressures and left atrial pressure, moderate dilation of both atria and no pulmonary hypertension.

The transesophageal echocardiogram was performed after oropharyngeal anesthesia with xylocaine, without difficulty passing the esophageal probe, under cardiac monitoring, with an electrocardiogram and a cardioversion/defibrillation device available. The patient's baseline state showed atrial flutter with variable 2:1, 3:1 conduction. This echocardiogram reported biventricular dilation, a structurally normal mitral valve with moderate regurgitation, a chicken-wing left atrial appendage, without thrombus, normal aortic valve, mild functional tricuspid regurgitation, presence of spontaneous echo contrast in the right atrium, which was dilated and free of thrombus. The conclusion was: biventricular systolic dysfunction, biatrial dilation, functional mitral and tricuspid regurgitations; moderate and mild respectively.

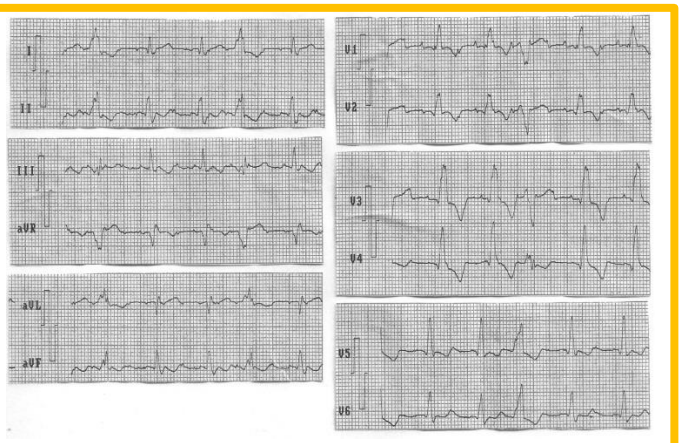

Figura 1- Baseline electrocardiogram:
Legend: atrial flutter with variable 2:1 and 3:1 atrio-ventricular block, counterclock, atrial rate of 300 per minute, intermittent atrio-ventricular dissociation, wide QRS complexes right bundle branch block morphology, and some isolated wide complexes with left bundle branch block morphology.
The F waves of atrial flutter are not always the classic sawtooth pattern. In the figure several waves can be observed in different leads, at a rate of 300 per minute, some of which are blocked. They are clearly seen in leads DIII, V1, V2 and V3.

During this echocardiographic study, exactly when the esophageal probe was being passed, a regular rhythm tachycardia was recorded on the electrocardiogram; with wide QRS of 240 milliseconds, a right bundle branch block pattern of the His bundle, and a heart rate of 280 beats per minute. Although the R-R interval coincided with the F-F interval on the electrocardiogram prior to the event, the electrocardiographic criteria raised the possibility of ventricular tachycardia (**Figure 2**). At that time, blood pressure was 80/40 mmHg, but the event was interpreted as atrial flutter with 1:1 atrio-ventricular conduction and hemodynamic instability. The condition persisted after the removal of the esophageal probe, so electrical cardioversion was performed three times with 200-joule biphasic shocks, after intravenous sedation with propofol. Sinus rhythm was restored as shown in **figure 3**, with no further arrhythmia during follow-up. In a long strip of lead DII, illustrated in **figure 4**, sinus rhythm and an

isolated ventricular premature beat can be seen.

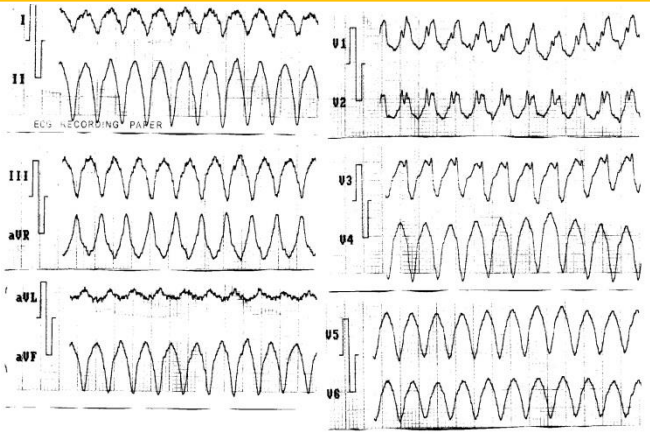

Figura 2- Electrocardiogram during the transesophageal study
Legend: wide-QRS tachycardia with right bundle branch block morphology, rate of 250 beats per minute, QRS duration of 240 ms. Note the QS complex in lead DI, QRS axis –120°, positive aVR, and R/S ratio >100 ms in precordial leads. The QRS axis is similar in the ventricular premature beat and the tachycardia, differing from the baseline ECG.

The definitive diagnosis of the acute event during the transesophageal echocardiogram was interpreted as atrial flutter with 1:1 conduction. However, other diagnoses were considered including ventricular tachycardia, antidromic tachycardia, atrial fibrillation mediated by an accessory pathway, pulmonary embolism and cardiac tamponade. All were ruled out, but ventricular tachycardia could not be rejected from the electrocardiographic standpoint as seen in figure 2, since it meets the criteria[3,4,5].
In favor of the diagnosis of atrial flutter with 1:1 conduction the following points were considered:

- The patient's clinical context with a history of atrial flutter.
- The measurement of the F-F interval before the transesophageal echocardiogram coincided with the R-R interval of the TWQRSC.
- Its onset occurred when passing the probe through the esophagus, where sympathetic plexuses can be stimulated, which also influences cardiac conduction and the left atrium is the closest chamber to the esophagus.

Nevertheless, an electrophysiological study is needed to establish a more definitive diagnosis.
The patient was transferred to the intensive care unit for close monitoring and continuous observation. At 72 hours she was clinically stable, without complications and was readmitted to the arrhythmia and pacemaker ward.
The patient was taken to the operating room for electrophysiological study, in typical atrial flutter, seen during the surface electrocardiogram and corroborated during the intracavitary electrocardiogram, at a F-F rate of 300 per minute. Subsequently, ablation of typical atrial flutter (counterclockwise, cavotricuspid isthmus-dependent) was performed using a radiofrequency catheter, achieving bidirectional isthmus block after 12 applications with controlled temperature between 50 and 60°C. Ventricular stimulation protocol was performed with the aim of ruling out ventricular tachycardia; no arrhythmia of this kind was provoked, but this fact must be carefully interpreted because false negative diagnoses may take place. The procedure lasted 90 minutes and was uneventful.
The following day, a follow-up transthoracic echocardiogram showed a left ventricular ejection fraction of 56%, apical akinesis in its middle and inferoseptal in its middle third. These results were similar to the previous study, except for the evident improvement in the ejection fraction after arrhythmia reversal.
The patient was discharged in sinus rhythm with no recurrence of atrial flutter or any other arrhythmia during four-month of follow-up.
After the study, additional criteria supporting the diagnosis of the 1:1 atrial flutter were added:

- No ventricular tachycardia was induced during ventricular stimulation in the electrophysiological study.
- Ablation of atrial flutter was performed, and the patient never again developed a wide-QRS tachycardia during follow-up.

COMENTARIO

The diagnosis of tachycardia with wide QRS complexes by surface electrocardiogram may be difficult. Diagnosis mistakes are frequent and are commonly associated with higher mortality. On spite of the well-established electrocardiographic criteria that make ventricular tachycardia different from supraventricular tachycardia with aberrancy, a diagnosis mistakes rate between 35 % and 39 % for ventricular tachycardia at the beginning of the treatment has been informed. In addition, with the exclusion of atrio-ventricular dissociation, common criteria used[3-5] are not absolute, so clinical judgment cannot be forgotten.
In the current case, the discussion around the tachycardia with wide QRS and the significant hemodynamic repercussion when passing the probe is precisely to be able to give the definitive diagnosis. The appearance of tachycardia was initially interpreted as an episode of atrial flutter with 1:1 conduction through the atrioventricular node in a patient with no prior history of this conduction, apparently triggered by the transesophageal echocardiogram. It is worth noting that the presence of QS complex-

es during arrhythmia is also seen in other entities such as ventricular tachycardia and tachycardia with anterograde conduction over an accessory pathway, which had to be ruled out.

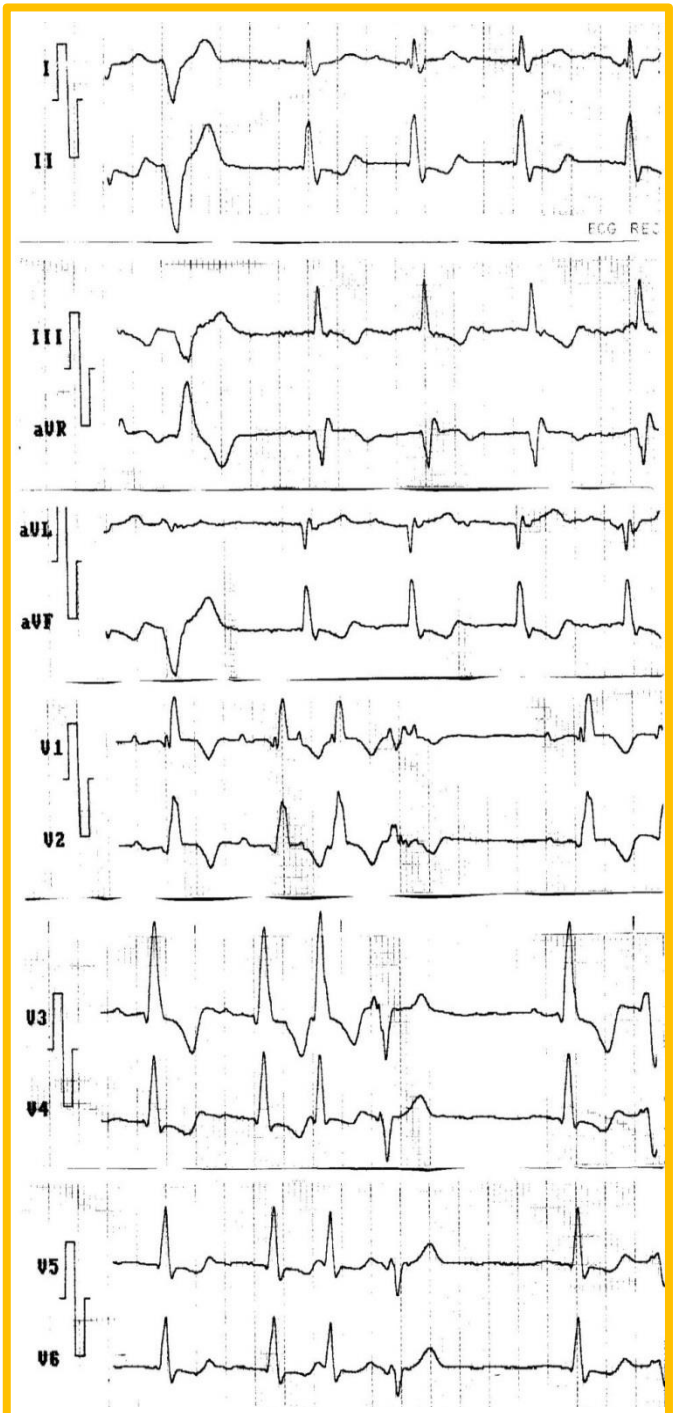

Figura 3- Post-cardioversion electrocardiogram
Legend: Some sinus complexes, different duration QRS. The waves observed and interpreted as P waves in lead V1 are also seen in leads DII, V1, V2 and V3. They are not artifacts and the intervals between these waves and the QRS are consistent. Note in lead V1 an atrial premature beat with a prolonged PR interval, followed by a ventricle premature beat or a junctional beat, dissociated.

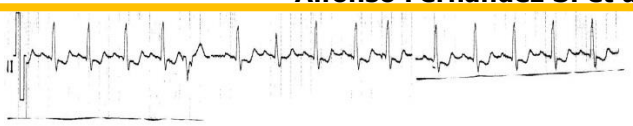

Figura 4- A longer strip of lead DII

After initial diagnosis, it was considered that there were enough elements to think of a ventricular tachycardia according to the surface electrocardiogram. The nosological approach was reassessed as tachycardia with wide QRS complexes and hemodynamic instability as a complication of the transesophageal echocardiogram, which main diagnoses to be established would be typical counterclock atrial flutter with 1:1 conduction or infra-Hisian ventricular tachycardia.

The clinical context favors atrial flutter with 1:1 conduction due to the patient's history and the onset coinciding with manipulation of the echocardiography transducer once inside the esophagus. Other possible triggers, both pharmacological and due to electrolyte imbalances, were ruled out. The patient had never been treated with antiarrhythmic drugs and at no time before the transesophageal echocardiogram did this type of 1:1 conduction exist. Thus, a strict temporal relationship between the procedure and the arrhythmia was established, which suggests that the study could have acted as a trigger.

Certainly, when assessing the tachycardia presented, it meets some electrocardiographic criteria of ventricular tachycardia[3-5] such as: QS complexes in lead DI, axis of the QRS complex: -120^0, positive aVr and R/S ratio >100 ms in precordial leads. The QRS axis is similar in ventricular premature beats and the tachycardia, differing from the electrocardiogram observed in figure 3. In addition to these elements found in the surface electrocardiogram, there are other aspects to be discussed: the absence of history of ventricular tachycardia in the patient, the non-reproducibility of arrhythmias during ventricular stimulation, on which there was no action. After ablation of atrial flutter, no arrhythmia appeared during follow-up.

The occurrence of the arrhythmia during the referred procedure corresponds to the proximity of the esophagus to the cardiac chambers and the manipulation of the esophageal probe transducer. However, why do not reports of this complication appear in the revised series? This could be due to lack of reports of such cases, underreporting, transient events or diagnostic confusions with ventricular tachycardia.

What is interesting about the present case is precisely to establish a diagnostic conclusion, since when faced with a wide-QRS tachycardia, the differential diagnosis is difficult, often clari-

fied by programmed electrical stimulation; which, together with clinical judgment, led us to think of atrial flutter because; as previously explained, the R-R interval of the tachycardia coincided with the F-F interval on the electrocardiogram prior to the event. No recordings of the intracavitary electrocardiogram are available, but it was observed by the working team.

Typical atrial flutter is one of the most common arrhythmias in clinical practice, with an estimated global incidence of 88 cases per 100,000 person-years[7]. Atrio-ventricular conduction is usually 2:1 or lower due to the refractory properties of the atrio-ventricular node; however, 1:1 conduction can produce higher ventricular rates between 200 and 300 beats per minute, with significant hemodynamic compromise. The prevalence of atrial flutter with 1:1 atrio-ventricular conduction is up to 8% in patients undergoing ablation, either at initial presentation or with a prior history; it is more common in young patients and in those on antiarrhythmic treatment[7,8].

The esophagus is intimately related to the heart, the posterior wall of the left atrium is the closest structure. Through autonomous vagal and sympathetic reflexes, mechanical or electrical esophageal stimulation can activate the vagal afferents, and its stimulation or parasympathetic inhibition acts on the sinus node and the atrio-ventricular node, where a reduction in the refractory period could occur, given the complex sympathetic-vagal regulatory mechanisms. This phenomenon is related to recent reports of arrhythmias induced by esophageal stimulation or deglutition, described as either atrial tachyarrhythmias or bradyarrhythmias mediated by vagal reflexes [9.10, 11].

The integrity of the autonomic nervous system is important, maintained by the balance between the sympathetic and the parasympathetic (vagal) nervous systems. Both the right and left vagus nerves enter the mediastinum; in their course, they pass into the posterior mediastinum and form the esophageal plexus, at the level of the esophagus. Mechanical stimulation can cause imbalance, leading to significant variations in heart rate and blood pressure. This stimulation would be more evident in a patient with a pre-existing arrhythmogenic substrate, so manipulation during the passage of an esophageal probe with the transesophageal transducer for echocardiography through this area may cause the imbalance.[12.13]

The findings described in the patient, as well as the mechanical stimulation of the esophagus during the study, highlight the importance of considering the potential risks of a procedure considered routine in clinical practice.

The transesophageal echocardiogram is a fundamental tool to rule out thrombus in the left atrium before performing electrical cardioversion or arrhythmia ablation procedures. Although it is considered a safe technique, it is not without complications. The incidence, though rare, is estimated to be between 0.18 % and 2.8 %, with a mortality of less than 0.01 %.[10.13] Complications described in this diagnostic test may include lung infections, respiratory failure, bronchospasm, syncope or other vasovagal events, arterial hypotension, oropharyngeal trauma, bleeding, esophageal perforation, pulmonary aspiration, and induced arrhythmias, which incidence is extremely low.[14]

Bradyarrhythmias are reported to be between 0.3 to 1%; supraventricular arrhythmias, including atrial fibrillation, atrial flutter and paroxysmal supraventricular tachycardia, together represent between 0,1 to 0.5%; and ventricular tachycardia occurs in less than 0.1% of cases.[8,10,11,13-14]

Normally, the atrio-ventricular node acts as a filter, with a physiological delay due to its decremental properties, which prevents the high frequency of atrial flutter waves from being fully transmitted to the ventricles, because of the impulse delay in the compact atrio-ventricular node, a calcium-dependent tissue. In circumstances such as the presence of edema, inflammation or degeneration, this mechanism is bypassed; allowing 1:1 conduction with extremely high ventricular rates and hemodynamic instability.[15]

Atrial flutter with 1:1 conduction by hyperconductor atrio-ventricular node is not an infrequent entity, the hysiogram is useful for understanding this phenomenon, since it allows to identify the rapid conduction through the node and the absence of the common delay. Short A-H interval and atrial waves at a very high rate (similar to 200–300 beats per minute) are shown. Every atrial wave is conducted into the ventricle and an A:H:V of 1:1:1. ratio is observed.[15]

In general, this arrhythmia is poorly tolerated; it requires urgent treatment with electrical cardioversion. As definitive treatment, ablation of the circuit to achieve cavotricuspid isthmus block, which has success rates above 90% and a low incidence of complications[15]. If it is not possible or the procedure fails, its management with antiarrhythmic drugs is difficult.[16]

Concluding we presented a case of a patient with a baseline atrial flutter who during a transesophageal echocardiogram presented tachycardia with wide QRS with hemodynamic compromise, which diagnosis by surface electrocardiogram could be finally established after electrical stimulation when ventricular tachycardia was not provoked and ablation of the atrial flutter was performed. The patient never

again developed any kind of arrhythmia nor other symptomatology related to it.

REFERENCES:

1. Daniel WG, Erbel R, Kasper W, Visser CA, Engberding R, Sutherland GR, et al. Safety of transesophageal echocardiography. A multicenter survey of 10,419 examinations. Circulation. 1991;83(3):817-21. doi:10.1161/01.CIR.83.3.817.
2. Bunch TJ, Day JD. Examining the risks and benefits of transesophageal echocardiogram imaging during catheter ablation for atrial fibrillation. Circ Arrhythm Electrophysiol. 2012;5(4):621-3. doi: 10.1161/CIRCEP.112.973297.
3. Wellens HJJ, Bar FW, Lie KL. The value of the electrocardiograms in the differential diagnosis of a tachycardia with a widened QRS complex. Am J Med. 1978,64(1):27-33. doi:10.1016/0002-9394(78)90176-6.
4. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation. 1991;83(5):1649-59. doi:10.1161/01.cir.83.5.1649.
5. Vereckei A. Current algorithms for the diagnosis of wide QRS complex tachycardias. Curr Cardiol Rev. 2014;10(3):262-76. doi.10.2174/1573403x10666140514103309.
6. Lopez JA, Lufschanowski R, Massumi A. Transesophageal electrocardiography and adenosine in the diagnosis of wide complex tachycardia. Tex Heart Inst J [Intrnet]. 1994 [cited: 2026 en. 15];21(2):130-3. Available in: https://pmc.ncbi.nlm.nih.gov/articles/PMC325147/pdf/thij00037-0022.pdf
7. Tan S, Zhou J, Veang T, Lin Q, Liu Q. Global, regional, and national burden of atrial fibrillation and atrial flutter from 1990 to 2021: sex differences and global burden projections to 2046-a systematic analysis of the Global Burden of Disease Study 2021. Europace. 2025;27(2):euaf027. doi: 10.1093/europace/euaf027.
8. García-Cosío F, Pastor A, Núñez A, Magalhaes AP, Awamleh P. Flúter auricular: perspectiva clínica actual. Rev Esp Cardiol. 2006;59(8):816-31. doi: 10.1157/13091886.
9. Alyeshmerni D, Pirmohamed A, Barac A, Smirniotopoulos J, Xue E, Goldstein S, et al. Transesophageal echocardiographic screening before atrial flutter ablation: is it necessary for patient safety? J Am Soc Echocardiogr. 2013;26(9):1099-105. doi: 10.1016/j.echo.2013.05.017.
10. Kenawi A, El Masri A, Abdelnasser H. Deglutition-Induced atrial fibrillation in a previously healthy adult male: a rare trigger unmasked after medication discontinuation. Cureus. 2025;17(10):e94395. doi: 10.7759/cureus.94395.
11. Ellington AL, Tarry ST, Ellis MNH, Clayton SB. Situational syncope triggered by swallowing. ACG Case Rep J. 2025;12(1):e01588. doi: 10.14309/crj.0000000000001588.
12. García-Thompson JC, Delgado-Hernández X, Gil-Pérez A, González-Pérez J, Hernández-Xumet JE. Repercusiones de la terapia manual en el sistema nervioso autónomo. Revisión sistemática. Rev Cuidar. 2023:305-331. doi: 10.25145/j.cuidar.2023.03.16.
13. Zhang L, Xie Y, Ren Z, Xie M. Transesophageal echocardiography related complications. Front Cardiovasc Med 2024;11:1410594. doi:10.3389/fcvm.2024.1410594
14. Patel KM, Desai RG, Trivedi K, Neuburger PJ, Krishnan S, Potestio CP. Complications of transesophageal echocardiographic: a review of injuries, risk factors, and management. Vasc Anesth 2022;36(8)partB:3292-3302. doi:10.1053/j.jvca.2022.02.015.
15. Chatterjee D. Atrial flutter with 1:1 AV conduction - a case series. J Cardiol Clin Res. 2024;12(1):1200. doi: 10.47739/2333-6676/1200.