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# Aleteo auricular: caso clínico

*Atrial flutter: case report*

[1] Evelyn Johanna Banegas Santos https://orcid.org/0000-0002-1919-8390
Catholic University of Cuenca, Cuenca, Ecuador.
jhulye19@hotmail.com

[2] Rodrigo Jose Mendoza Rivas https://orcid.org/0000-0001-6020-9226
Catholic University of Cuenca, Cuenca-Ecuador.
rodrigo.mendoza@ucacue.edu.ec

[3] Aida Marlene Yunga Court https://orcid.org/0000-0001-8334-8480
Catholic University of Cuenca, Cuenca-Ecuador.
aida.yunga@ucacue.edu.ec

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**Palabras claves:**
Atrial flutter; fibrilación auricular; aleteo auricular;

**Resumen**

**Introducción.** El aleteo auricular es una enfermedad que no pone en peligro la vida y puede ser muy sintomática debido a las altas frecuencias ventriculares que normalmente ocurren. El diagnóstico es fácil porque solo se requiere un ECG de 12 derivaciones. En pacientes que no pueden revertir o prevenir el aleteo, se puede intentar con éxito la resección del nódulo aurícula ventricular y la implantación de un marcapasos permanente. Las técnicas de ablación por radiofrecuencia ahora son muy exitosas para revertir el aleteo auricular y prevenir su inducibilidad y recurrencia. **Objetivo:** Determinar el manejo de la patología aleteo auricular para presentar aspectos nuevos o instructivos de la enfermedad. **Metodología:** estudio de caso clínico de tipo descriptiva, retrospectivo. La técnica que se utilizó para la recolección de la información del caso fue mediante la revisión de historia clínica y para la descripción de la patología será mediante la recopilación de artículos extraídos de bases de datos reconocidas como: Scopus, PorQuest, Pubmed, web of science, lilacs. Como criterio de inclusión: artículos publicados en los últimos 5 años, en español e inglés. El caso cuenta con el consentimiento informado del paciente para dar cumplimiento a lo establecido por Bioética. **Resultados:** Se identificaron las principales causas, síntomas y signos, el diagnóstico, tratamiento y prevención del aleteo auricular, como personal de salud es de vital importancia adquirir nuevos conocimientos sobre esta patología que afecta con más frecuencia a adultos. **Conclusiones:** El mayor riesgo de desarrollar aleteo auricular son los hombres, los ancianos y las personas con insuficiencia cardíaca preexistente o enfermedad pulmonar obstructiva crónica es por ello que nuestro estudio fue aplicable el tratamiento con antiarrítmicos, además de anticoagulantes. **Área de estudio general:** medicina. **Área de estudio específica:** cardiología. **Tipo de estudio:** Casos clínicos.

**Keywords:**
Atrial flutter; atrial fibrillation; heart headset.

**Abstract**

**Introduction.** Atrial flutter is a non-life-threatening disease and can be very symptomatic due to the high ventricular rates that normally occur. Diagnosis is easy because only a 12-lead

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ECG is required. In patients who cannot reverse or prevent flutter, resection of the atrioventricular node and implantation of a permanent pacemaker can be successfully attempted. Radiofrequency ablation techniques are now very successful in reversing atrial flutter and preventing its inducibility and recurrence. Objective. Determine the management of atrial flutter pathology to present new or instructive aspects of the disease. Methodology. Descriptive, retrospective clinical case study. The technique used to collect information on the case was through a review of the medical history and to describe the pathology, it will be through a collection of articles extracted from recognized databases such as: Scopus, PorQuest, Pubmed, web of science, lilacs. As inclusion criteria: articles published in the last 5 years, in Spanish and English. The case has the informed consent of the patient to comply with the provisions of Bioethics. Results. The main causes, symptoms and signs, diagnosis, treatment and prevention of atrial flutter were identified. As health personnel, it is of vital importance to acquire new knowledge about this pathology that most frequently affects adults. Conclusion. The highest risk of developing atrial flutter are men, the elderly and people with pre-existing heart failure or chronic obstructive pulmonary disease, which is why our study applied treatment with antiarrhythmics, in addition to anticoagulants.**General area of study:**medicine. Specific area of study: cardiology. Type of study: clinical cases.

## Introduction

Atrial flutter (AFL) is one of the most common arrhythmias present in clinical practice, which after atrial fibrillation (AF), represents the second most frequent supraventricular arrhythmia.(1)The term flutter first appeared in 1887, with Mac William, who described the visual phenomena resulting from faradic stimulation of the atria that puts them into rapid fluttering.(2).

In his study Bun (3) initially proposed a single wave circular motion mechanism, but the possibility of reproducing the AFL morphology on the Electrocardiogram (ECG) with a high pacing frequency or with a focal injection of aconitine supported a focal mechanism as another possible hypothesis. Although both mechanisms are easily observable in

animal models, the circus motion theory has finally been accepted as the most frequent one in man. A macroreentrant mechanism was finally demonstrated by detailed mapping in the operating room as well as in the electrophysiology laboratory.

Atrial flutter is a common supraventricular arrhythmia characterized by a reentrant circuit around a central obstacle, which may be a fixed anatomical structure or a functional electrophysiologic line of block. Depending on the obstacle, AFL is usually classified as "typical" if the reentry is around the tricuspid valve, or "atypical" if the tricuspid valve is not involved. Atypical AFL is often associated with structural heart disease, especially in patients who have undergone cardiac surgery or extensive catheter ablation for the treatment of atrial fibrillation (AF). In these cases, an electrophysiologic (EP) study is the most common way to unravel the mechanisms causing the arrhythmia and plan an appropriate ablation. Although AFL is not directly related to death, it affects quality of life due to the increased rate of ventricular activation and can cause significant complications such as heart failure and stroke. Furthermore, the presence of AFL often suggests an underlying predisposition to AF, which is a more complex arrhythmia.(4).

Atrial fibrillation (AF) and atrial flutter often coexist due to shared risk factors and precipitants. Both rhythms can be a cause or consequence of heart failure and are associated with stroke and increased mortality.(5)Atrial flutter occurs in many of the same situations as AF. Typical atrial flutter, also known as "typical AFL" involves a macroreentrant circuit around the tricuspid annulus passing through the cavotricuspid isthmus on the right side of the heart. This is the arrhythmia associated with the classic electrocardiogram (ECG) finding of sawtooth flutter waves in the inferior leads when the circuit is going counterclockwise. The same clockwise circuit is called "reverse typical AFL." If the flutter involves a circuit other than the tricuspid valve/isthmus, then it is called "atypical" AFL, which is also known as "isthmus-dependent non-cavotricuspid macroreentrant AT." AFL was previously classified as type I or type II. That terminology is no longer used.(6).

It is not uncommon for AFL and AF to be treated as interchangeable diagnoses. However, more than 70% of patients with AFL do not experience AF and less than 10% with AF are also diagnosed with AFL. Furthermore, the distinction between AF, typical and atypical AFL is important, as the risks and success of catheter ablation are markedly different.(7)In this sense, atrial flutter and atrial fibrillation are two distinct arrhythmias whose electrophysiological mechanisms are completely opposite to each other; however, both arrhythmias are often considered similar and, what is more dangerous, managed in a similar way.(8).

Both arrhythmias, often coexisting, share some similarities regarding clinical management and possible complications; however, they differ in the underlying mechanism of the disease. The initially episodic arrhythmia frequently becomes

persistent and eventually permanent, as a result of atrial electrophysiological disorganization and anatomical remodeling.(9)Both rhythms can be a cause or consequence of heart failure and are associated with stroke and increased mortality.(10,11). This pathology can be diagnosed by an ECG(12).

Important clinical implications of AF and AFL include their negative impact on quality of life, increased incidence of heart failure, stroke, systemic thromboembolism, and increased risk of death in this patient population. These patients are 3 times more likely to have heart failure and 5 times more likely to have stroke. Patients with AF and stroke have a markedly increased additional risk of death, while individuals with AF and nonfatal stroke have a higher risk of severe disability than stroke subjects without AF. They are also associated with increased risk of hospital admissions.(13).

Atrial flutter has a low incidence in children. It is characterized by a rapid atrial rate of approximately 300 beats per minute or more and distinctive "sawtooth" P waves, called F waves. Although atrioventricular conduction can be variable, the most common is 2:1 conduction, which is usually well tolerated; however, 20% of cases may present with heart failure. In adults and older children, it is usually associated with structural heart disease, one of the complications that can occur after surgical correction of different congenital heart diseases. A possible etiology should be sought in the first episode, ruling out cardiac dysfunction or associated heart disease.(14).

Flutter waveform morphologies in macroreentrant ATs are highly variable, and there are few invariable features that identify specific reentrant circuits. Atypical right atrial flutters often demonstrate predominantly negative deflections in V1. However, other right atrial tachycardias, such as counterclockwise ITC-dependent atrial flutter, have positive deflections in V1, usually preceded by an isoelectric or negative component. Atypical left atrial flutters often show wide positive deflections in V1 but may show initial negative deflections followed by positive ones. The limb and precordial leads in left atrial flutters often show very low amplitude signals, particularly in patients who have undergone prior ablation. These generalizations are often violated, as the extent and distribution of the atrial scar influences the morphology of the resulting flutter wave.(15).

Atrial flutter can be caused by scarring of the heart as a result of previous heart disease or heart surgery, but it can also occur in some patients without other heart problems.(12)Atrial flutter usually occurs in the right atrium and regularly involves a large circuit that extends from around the tricuspid valve area located between the right atrium and the right ventricle. This type of atrial flutter is known as typical atrial flutter.(12)The mechanism is a large reentry circuit contained in the right atrium (RA) with passive activation of the left atrium (LA).(16)Activation occurs supero inferiorly in the anterior and lateral RA and infero superiorly in the septal RA, with a critical inferior

inflection point between the tricuspid annulus and the inferior vena cava (IVC) known as the cavotricuspid isthmus (CTI).(17).

Less commonly, atrial flutter can result from circuits in other areas of the right or left atrium that cause the heart to beat faster. Atypical atrial flutter is the type of flutter mentioned above, which results from these less common types of circuits.(12)The term atypical has been applied to rapid atrial tachycardias with ECG patterns different from typical flutter and their mechanism can only be determined by mapping and pacing electrocardiography studies.(16)Atypical flutter is often associated with structural heart disease, especially in patients who have undergone cardiac surgery or extensive catheter ablation for the treatment of atrial fibrillation.(18).

The incidence of typical and atypical forms of atrial flutter is age-dependent with 5/100,000 in patients younger than 50 years and approximately 600/100,000 in subjects >80 years of age.(19). About 80% of patients with flutter are men(17); otherwise, fluttering occurs in clinical contexts such as old age, chronic obstructive pulmonary disease, alcohol abuse, or during endurance exercise or sports practice.(18).

AF/AFL-related deaths totaled 287,241 in 2017 and remain one of the leading causes of death worldwide. While the age-standardized mortality rate for AF/AFL has decreased by 2.53% over the past decades, the absolute number of disability-adjusted life years due to AF/AFL increased globally from 1990 to 2019 and has risen to 5.97 million people. AF/AFL has become one of the leading causes of disability worldwide, and post-AF/AFL care and economic costs of treatment are substantial. In the United States, 3–5 million people have AF/AFL, and as the population ages, it is expected to affect more than 8 million people by 2050.(20,21).

Treatment options for both rhythms include pharmacological rate monitoring and rhythm control (antiarrhythmic medications, cardioversion, and catheter ablation).(22,23)In the specific case of atrial flutter, individuals who suffer from this pathology have a greater risk of suffering a cerebrovascular accident and cerebral hemorrhage.(24)Atrial flutter responds to class III antiarrhythmic drugs, whereas class IC agents are not helpful as they almost always fail and may actually create a substrate for flutter.(25). If applied for the first time, it is reasonable to perform pharmacologic cardioversion in the hospital to observe for possible adverse effects. In addition, other agents are used for immediate cardioversion, including even the typical rhythm control agents, amiodarone and sotalol, which primarily control rate rather than rhythm in the first few hours of treatment and are therefore ineffective conversion drugs.(26).

Electrical cardioversion terminates the arrhythmia in more than 90% of cases and is the treatment of choice in patients with severe hemodynamic compromise with new-onset AF or AFL. Compared with AF, electrical cardioversion is more effective in patients with

atrial flutter and also requires less energy. Electrical cardioversion can be safely performed under brief sedation with intravenous midazolam and/or propofol and continuous monitoring of blood pressure and oximetry during the procedure.(27).

Electrical cardioversion is most effective when using a biphasic defibrillator, and about 40% of patients are pretreated with an implantable cardioverter-defibrillator. The anteroposterior position of the electrode restores sinus rhythm better than the anteroapical position. Starting with the maximum available shock energy appears more effective than increasing shock energies. In patients with an implanted pacemaker or implantable cardioverter-defibrillator, damage to the system can be avoided by biphasic cardioversion in the anteroposterior paddle position. Even in patients with implanted defibrillators, it appears preferable to internal cardioversion performed with the implantable cardioverter-defibrillator.(28).

For almost 60 years, vitamin K antagonists (VKAs) have been the mainstay of anticoagulant therapy. In 2008, a new class of drugs was introduced to the European Union and US markets, providing a promising alternative to VKAs in the prevention of embolic complications in non-valvular AF, as well as in the treatment of patients with deep vein thrombosis and pulmonary embolism. These were new-generation oral anticoagulants, originally referred to as new/novel oral anticoagulants (NOACs) and now as direct oral anticoagulants (DOACs).(29).

They act as direct factor Xa inhibitors (rivaroxaban, apixaban, edoxaban, and betrixaban) or direct thrombin inhibitors (dabigatran). Their anticoagulant effect is more predictable and stable (i.e., less dependent on interactions with food, herbal supplements, and other drugs) compared with warfarin and acenocoumarol. The use of DOACs does not require individual dose adjustment or routine monitoring of blood coagulation parameters such as international normalized ratio (INR), activated partial thromboplastin time (APTT), and thrombin time. With VKA treatment, a therapeutic INR range of 2.0 to 3.0 is recommended in the prevention of embolic complications in nonvalvular AF, in the treatment of deep vein thrombosis, and in pulmonary embolism. The recommended time in therapeutic range (TTR) is >70% during VKA treatment, which in the context of significant dietary interactions and individual pharmacokinetic profiles requires frequent INR monitoring. Therefore, the cost-effectiveness and safety of long-term VKA treatment are considerably lower.(30).

Therefore, the development of this study is argued to be based on the adequate management of this pathology to ensure good care, since the prevalence of atrial flutter is more frequent in men than in women. A significant risk factor is advanced age, since other associated disorders in patients with atrial fibrillation include systemic hypertension, diabetes mellitus, and a history of alcohol abuse.

**Methodology**

A review and analysis of a clinical case was carried out. Data were collected through a review of the clinical history of the case of interest. Regarding the writing, the Vancouver style was used to reference the description of the pathology. Likewise, the following structure was applied: definition of the pathology, pathophysiology, risk factors, diagnosis, prognosis, signs and symptoms, consequences, nursing care plan and medical treatment.

**Results**

A 67-year-old male patient was admitted to the health center for cardiovascular evaluation. He had chronic ischemic heart disease, had a pacemaker placed 6 years ago, no specific cause was given, and denied any allergies to medications or foods. He reported an uncomplicated ophthalmologic surgical history.

Refers to habitssmoking in youth who have been inactive for 20 years, reports a pattern of dyspnea and class II angina. On physical examination:

Vital signs: Right upper limb BP 110/70mmHg, left upper limb BP 100/60mmHg, HR60 bpm RR 17 rpm Spo2 95% (room air). Anthropometric measurements: Weight: 82kg height: 1.63 m.

Patient in stable general condition, afebrile, eupneic, tolerating oral route and ambient oxygen, normocephalic, mobile neck, regular non-igurgitated veins, TO 3cm from the angle of Louis, symmetrical carotid pulses of good amplitude without murmurs, thorax: symmetrical with normal configuration, normoexpansible, respiratory sounds present in both lung fields without aggregates. Cardiac apex not visible or palpable, arrhythmic heart sounds of good tone, single r1 silent systole, pathological split r2 silent diastole, soft flat abdomen, symmetrical, soft not painful to superficial or deep palpation, hydro-air sounds present of normal frequency without viceromegaly, symmetrical extremities, eutrophic, without edema with grade II varicose veins, decreased posterior tibial arterial pulses, rest preserved in amplitude. Neurological preserved.

**Complementary exams**

- **Electrocardiogram:**Non-sinus rhythm / HR 100 bpm / PR-ms / QRS 90 MS / QT 420 ms AXIS +30 ° Conclusion: atrial fibrillation-type rhythm disorder with adequate ventricular response.
- **Echocardiogram and transthoracic view:**Preserved systolic function, LVEF 62%, biatrial dilatation, grade II diastolic dysfunction, mild aortic valve insufficiency, and other valve sections morphologically and functionally normal.

**Medical treatment indicated on an ongoing basis:**

1. PLENACOR: 50mg tablet every 12 hours
2. XAROBAN one tablet once a day.
3. FUROSEMIDE: half a 40 mg tablet every 12 hours.

**Diagnosis:**

- Atrial fibrillation-type rhythm disorder with adequate ventricular response

**Suggestions:**

- Evaluation by the electrophysiology service.

**Discussion**

Atrial flutter is an important condition for nurses to understand and manage, as it is a cardiac arrhythmia that can cause palpitations, shortness of breath, chest pain, and fatigue. Treatment focuses on controlling the heart rate, converting the rhythm back to sinus if possible, and decreasing the risk of stroke through the use of anticoagulant therapy. Having a comprehensive nursing care plan is essential to managing atrial flutter, promoting the patient's overall health and well-being, and reducing the occurrence of future episodes. By understanding the condition, identifying the rhythm, controlling the heart rate, preventing blood clots, monitoring and assessing the patient's progress, and providing health teaching and promotion, nurses can play a crucial role in the treatment of atrial flutter.

We present the case of an adult with a history of cardiac disorders, which required the placement of an internal pacemaker several years earlier, which is probably related to cardiac conduction or rhythm disorders; he also reports a history of ischemic heart disease, but it is not specified which one. At the time of consultation, there were no acute symptoms associated with the arrhythmia and his vital parameters were normal, which indicates that this is a case that is probably chronic, not associated with hemodynamic instability and, consequently, it is not a medical emergency.

At this point, it is worth noting that atrial flutter is the second most common cardiac arrhythmia after atrial fibrillation; it is commonly associated with atrial fibrillation, but the incidence and prevalence of atrial flutter are less well known compared to atrial fibrillation. Atrial flutter is common in patients with underlying diseases such as chronic obstructive pulmonary disease, pulmonary hypertension, and heart failure.(31)In the case presented, several of these risk factors are found, since the patient is a former smoker, who probably has associated COPD, with a history of probable heart failure, with

probable dilation of the atrial cavities, which justifies the appearance of flutter and/or fibrillation.

Isolated atrial flutter in the absence of abnormal cardiac anatomy is uncommon and is usually present when abnormalities in atrial size have developed. It is more common in men than in women. Aging is an important risk factor, as other associated disorders in patients with atrial fibrillation include systemic hypertension, diabetes mellitus, and a history of alcohol abuse. Advanced age is associated with an increased risk of atrial fibrillation and atrial flutter.(32).

They are sometimes asymptomatic or may present with a variety of symptoms including palpitations, fatigue, dizziness or reduced functional class, chest pain or dyspnea. The risk of thromboembolism is probably similar to that of atrial fibrillation; therefore, the same antithrombotic prophylaxis is required in patients with atrial flutter. Acutely symptomatic cases may undergo cardioversion or pharmacologic rate control to relieve symptoms.(33).

However, it may be the first presentation of more serious conditions such as acute pulmonary embolism, acute coronary syndrome, or acute pulmonary edema. The severity of symptoms is closely dependent on the initial left ventricular ejection fraction (LVEF), ventricular rate during flutter, and underlying SHD. As a common scenario, patients present with stroke or decompensated heart failure secondary to tachycardia-induced cardiomyopathy. Atrial flutter occurs in approximately 25% of patients with atrial fibrillation.(34).

The general goals of treatment of symptomatic atrial flutter are similar to those of atrial fibrillation and include control of ventricular rate, restoration of sinus rhythm, prevention of recurrent episodes or reduction of their frequency or duration, prevention of thromboembolic complications, minimization of adverse effects of therapy. However, these goals may be modified for each patient. In an acute setting with pending hemodynamic collapse, follow adult advanced cardiac life support (ACLS) algorithms to manage atrial fibrillation and flutter. Consider immediate electrical cardioversion in hemodynamically unstable patients. The main difference between atrial fibrillation and atrial flutter is that most cases of atrial flutter can be cured with radiofrequency ablation (RFA). In all available studies, catheter ablation is superior to rate and rhythm control strategies with antiarrhythmic drugs.(32,35).

The role of atrial flutter treatment involves reducing the risk of stroke, treating the rapid heart rate, and maintaining normal sinus rhythm.(12)In the specific case of atrial flutter, people who suffer from this pathology have a higher risk of suffering a stroke, this is because in this condition the blood does not move as quickly through the atria (the upper chambers of the heart) as it does during normal rhythm. The slower movement of the blood carries the risk of the formation of small blood clots that can cause a stroke.(24).

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If atrial flutter occurs in the setting of an acute pathologic process, medication for long-term rhythm control is usually not required once it is converted and the underlying pathologic process is eliminated. However, if there is a certain substrate for AFL recurrence, such as RA enlargement or scarring, medical suppression of AFL can be extremely difficult. Therefore, an ablation procedure with a high success rate and low risk of complications is the method of choice for typical atrial flutter.(32).

Antiarrhythmic agents should be combined with atrioventricular node (AVN) blockers to avoid the risk of rapid ventricular rates. Indeed, class IC drugs have a vagolytic effect on AVN. Although the atrial flutter rate will be reduced, a greater proportion of these atrial impulses will be conducted through AVN (enhanced conduction), thus increasing the net ventricular rate. As a result, rapid 1:1 AV conduction is mostly observed if Class IC antiarrhythmic medication is not combined with AVN blockers such as beta-blockers. As mentioned, typical AFL is highly susceptible to ablation, but AV junction ablation and pacemaker implantation may be indicated if rhythm and rate control strategies, including ablation, have failed in atypical flutter. The anticoagulant policy will be implemented based on the same guideline for AF.(36).

Rate control should be the first step in treatment in symptomatic patients with rapid ventricular rate. This is often a difficult goal in flutter, and even combinations of AV nodal blocking drugs (digoxin, beta-blockers, and calcium channel blockers) may fail, necessitating cardioversion to sinus rhythm. Dofetilide and ibutilide, pure class III antiarrhythmic drugs, are effective in stopping flutter with a small risk of QT prolongation and kinase ataxia.(37).

Class IA and IC antiarrhythmics are relatively ineffective or have no effect and may be problematic if they cause a slow atrial flutter rate ≤200/min with 1:1 AV conduction and QRS widening mimicking ventricular tachycardia (see Figure 4). Amiodarone may not be very effective in restoring sinus rhythm in acute situations, but it helps control the ventricular rate.(38).

The clinical presentation will dictate an acute therapeutic approach that may include cardioversion or a rate control strategy.(39)Cardioversion (electrical or chemical) is usually the initial treatment of choice in acute cases. Antiarrhythmic drugs such as intravenous amiodarone and sotalol have been reported to be effective.(40)have a high success rate in chemical cardioversion. These class III antiarrhythmics prolong the refractory period, resulting in a slower cycle length that could terminate atrial flutter.

The pharmacokinetics of amiodarone are best represented by a two-compartment model: central/vascular and peripheral/adipose with rapid intercompartmental distribution. These features explain the success of using a single bolus of high-dose amiodarone to produce transient elevations in concentration sufficient for cardioversion while maintaining safety,

even with a single oral dose of 30 mg/kg. This success is due to its distribution half-life between the 2 compartments (15 to 20 hours) being approximately 80 times shorter than its elimination half-life, which causes it to be rapidly distributed outside the vascular compartment.(41).

As a result, the bolus of amiodarone entering the central (vascular) compartment, from an intravenous or gastrointestinal source, only transiently elevates the serum concentration. Without additional doses, the vascular compartment rapidly equilibrates with the peripheral/adipose compartment, returning the vascular compartment to serum concentrations close to predose levels and avoiding prolonged exposure to high concentrations sufficient to cause toxicity.(42).

On the other hand, intravenous ibutilide has been more effective than the above in up to 76% of patients. Low-energy electrical cardioversion of 50 J has a very high success rate. Excessive atrial pacing using an RA catheter or a pre-existing pacemaker/defibrillator is an effective alternative option to terminate typical AFL. Anticoagulation should be considered using the same criteria as for AF, prior to cardioversion. Oral or intravenous AV nodal blockers such as verapamil, diltiazem, beta-blockers, and digoxin can be used for rate control. However, rate control is difficult to achieve unlike in AF.(43).

Ibutilide, a class III antiarrhythmic drug approved for use by the Food and Drug Administration in 1995, is available only for intravenous use because of its extensive first-pass metabolism. It prolongs repolarization time, action potential duration, and refractory period of atrial and ventricular myocardium through its action as a potassium channel blocker, affecting both the fast and slow components of potassium channels. It also increases the refractory period of the accessory pathway, the His-Purkinje system, and the atrioventricular node.(44).

The most common electrocardiographic changes caused by ibutilide are a mild slowing of the sinus rate and prolongation of the QT interval, similar to most other class III antiarrhythmic drugs. The degree of QT prolongation is related to dose, infusion rate, and serum concentration. The QT interval returns to baseline within 2 to 4 hours after discontinuation of the infusion; however, the PR or QRS intervals are not affected.(45).

Current guidelines on the treatment of AF predominantly focus on appropriate anticoagulation, followed by symptom control with rate or rhythm control and treatment of risk factors and comorbidities.(46). Despite significant advances, currently available therapies for the treatment of atrial fibrillation and flutter remain suboptimal. There is increasing evidence that rhythm control (i.e., maintaining normal sinus rhythm) can improve clinical outcomes, but current approaches have limited efficacy, in part due to a one-size-fits-all approach that ignores the diversity of etiology and mechanisms underlying the disease.(47).

Furthermore, currently available rhythm control pharmacological treatments are severely limited by potential proarrhythmic side effects. However, increasing understanding of the complex cellular and molecular mechanisms of these arrhythmias has revealed new potential targets and created opportunities for the development of novel pharmacological therapies. Traditionally, the main focus of pharmacological treatment has been the development of antiarrhythmic drugs. However, due to increasing awareness of the progressive nature of the disease, attention is now shifting towards the development of drugs targeting AF-related atrial electrical and structural remodelling, which play an important role in the maintenance and progression of the arrhythmia. This is relevant in the presented case because it was concluded as atrial fibrillation.(48).

Complications secondary to the use of antiarrhythmic drugs are related to the type of drug and the underlying mechanism of the drug. Regarding ablation complications, right atrial flutter is associated with lower complication rates than left atrial flutter ablation, and the risk of embolic stroke is increased with left-sided atrial flutter ablation compared with right-sided procedures.(31).

Compared with the healthy population, atrial flutter represents a higher mortality and thromboembolic risk, so ablation often represents the first-line treatment for the treatment of typical atrial flutter.(49)In the present case, the administration of antiarrhythmics plus anticoagulant treatment with good therapeutic effect is evident.

The 2018 Canadian Cardiovascular Society Guidelines recommend that all patients with acute atrial fibrillation and atrial flutter undergoing cardioversion receive 4 weeks of oral anticoagulation after cardioversion, including those without risk factors for stroke.(50)Anticoagulant therapy is an important aspect of the management of atrial flutter, as it may help prevent thromboembolic complications. Patients with chronic atrial flutter should receive long-term anticoagulant therapy, with a target INR of 2-3.

Anticoagulant therapy should also be considered for all patients with atrial flutter older than 65 years. Prior to cardioversion, patients with atrial flutter should maintain a therapeutic INR for 3 weeks. Anticoagulants such as warfarin may be prescribed to prevent blood clot formation in patients with atrial flutter. Nurses should closely monitor the patient's anticoagulant therapy and assess their compliance with medication or lifestyle changes. In summary, anticoagulant therapy is an essential component of controlling atrial flutter and preventing thromboembolic complications. Oral anticoagulants (OACs) are recommended in patients with atrial flutter and fibrillation to reduce the risk of stroke and thromboembolic events. In addition, direct oral anticoagulants (DOACs) were noninferior to warfarin (and superior with certain agents) with respect to the risk of thromboembolic events.(51).

Oral anticoagulation has a narrow therapeutic window. When the international normalized ratio (INR) is greater than 2.0, the risk of ischemic stroke is low. However, above the level 4.0, the risk of bleeding complications, especially intracranial hemorrhage, increases significantly. For those who continue to have TIAs or strokes despite a therapeutic INR or those who have a mechanical heart valve, many experts recommend a target INR of 2.5 to 3.5. Risk factors for major bleeding include an INR ≥4.0, older age, history of stroke, and hypertension and unstable INR control. A cohort study identified three risk factors for bleeding in an older population: alcohol abuse, chronic renal failure, and previous gastrointestinal bleeding.(52).

Radiofrequency ablation (RFA) is considered a curative procedure for typical atrial flutter (AFL); however, patients remain at risk of developing new atrial fibrillation (AF)(53).Such treatment often involves electrical cardioversion and/or antiarrhythmic medications.(54)Type I and type III antiarrhythmic drugs are often used to terminate or prevent recurrent episodes, and type II (beta blockers) and type IV (calcium channel blockers) can be used to control the ventricular rate during atrial flutter. However, antiarrhythmic drugs alone control atrial flutter in only 50% to 60% of patients.(55).

Another form of treatment is radiofrequency ablation, which has been used since the 1990s and is acutely successful in over 90% of cases, and avoids the long-term toxicity seen with antiarrhythmic drugs. Advanced mapping techniques and newer methods of delivering radiofrequency lesions are also being used to delineate unusual atrial flutter patterns and minimize fluoroscopic exposure during the procedure.(55).

Radiofrequency (RF) catheter ablation of the cavotricuspid isthmus (CTI) in typical or common atrial flutter is recommended for patients who are symptomatic or refractory to pharmacologic rate control. The CTI is a well-defined anatomical quadrilateral-shaped area bordered by the tricuspid valve (TV). To improve procedural success and prevent recurrence of AFL, bidirectional block in this region should be established as a primary endpoint. The anatomical features of the cavotricuspid isthmus have been associated with prolonged procedure time, a higher total amount of RF energy, and reduced success rates, regardless of RF catheter selection. Stroke, complete heart block, and pericardial effusion are the most frequently reported complications.(56).

Radiofrequency (RF) ablation carries risks similar to those associated with a standard EP study and additional risks specific to the ablation procedure itself. The risks encompass those typically associated with any cardiac catheterization procedure, including hemorrhage, thromboembolism, phlebitis, infection, and cardiac perforation. However, these risks are considerably lower compared to a standard cardiac catheterization; most EP studies do not involve arterial puncture and cause less damage to the arterial tree. The overall risk of these complications is less than 1%. This does not include risks associated

with radiation exposure during the procedure, which may be prolonged in complex cases.(57).

The main risks associated with RF ablation are the possibility of inadvertent complete heart block, which usually occurs when ablation is performed near the normal conduction system, as well as the risk of cardiac perforation and tamponade, which is commonly seen during ablation procedures performed on the atria and coronary sinus, or right ventricle. The incidence of these complications is less than 2%.(58).

Exceptionally rare complications may also arise, including the development of arrhythmogenic foci, damage to the valvular apparatus resulting in the introduction of mitral or tricuspid regurgitation, systemic embolization during manipulation within the left-sided chambers, pulmonary vein stenosis, and formation of stenotic lesions within the coronary arteries, particularly the right coronary artery. This complication warrants considerable attention, particularly when ablation is performed within the right ventricular outflow tract, coronary sinus, or cardiac veins. Serious, life-threatening irreversible complications are extremely rare; the overall risk of death, myocardial infarction, or stroke is usually less than 0.5%.(59).

## Conclusion

- Atrial flutter is an arrhythmia that does not threaten the patient's life but is sometimes very symptomatic due to the high ventricular rate with which it usually occurs.**flutter**Isolated atrial flutter has a risk of stroke at least as high as isolated atrial fibrillation and carries a higher risk of subsequent development of atrial fibrillation than in the general population. Anticoagulation should be considered for all patients with atrial flutter older than 65 years. Those at highest risk of developing atrial flutter are men, the elderly, and those with pre-existing heart failure or chronic obstructive pulmonary disease, which is why our study was applicable to treatment with antiarrhythmics in addition to anticoagulants.The most important decision for the cardiologist remains whether to strive to maintain heart rhythm or simply maintain an adequate heart rate. The first option is more advisable for symptomatic patients without much structural heart disease; the second, for patients who are asymptomatic or mildly symptomatic, elderly, and with associated structural heart disease that makes it likely that sinus rhythm will not be maintained. The combination with non-pharmacological treatments, such as ablation, is currently a reality that will probably become even more noticeable in the near future.

### Conflict of interest

The authors declare that there is no conflict of interest in relation to the submitted article.

**Authors' contribution statement**

Author 1: I actively participated in the planning and design of the literature review. I also carried out a critical evaluation of the selected studies, analyzing both the methodological quality and the validity of the results.

Author 2: Significantly contributed to the interpretation and discussion of the findings obtained in the clinical case. He/she also played an important role in the writing and revision of the content of the manuscript.

Author 3: Provided valuable input by providing comments that improved the clarity and coherence of the work. Actively participated in the development of the results and conclusions of the study.

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