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Atrial infarction: a literature review

Infarto atrial: revisión de la literatura

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ABSTRACT

Atrial infarction is an often-missed entity that has been described in association with ventricular infarction or as an isolated disease, which is mainly caused by atherosclerosis. The electrocardiographic diagnostic criteria were proposed more than fifty years ago and have not yet been validated. The diagnosis is based on elevations and depressions of the PTa segment and changes in the P wave morphology. However, supraventricular arrhythmias such as atrial fibrillation are the most common finding and often predominate in the clinical presentation. Early recognition and treatment may prevent serious complications such as mural thrombosis or atrial rupture. Further studies need to be carried out in order to establish unified criteria for the diagnosis and the actual prevalence of this entity.

RESUMEN

El infarto atrial es una entidad frecuentemente olvidada, ha sido descrita en asociación con el infarto ventricular o de manera aislada y es causado principalmente por aterosclerosis. Los criterios diagnósticos electrocardiográficos fueron propuestos hace más de 50 años y aún no han sido validados. El diagnóstico se basa en el hallazgo de elevación o depresión del segmento PTa y de alteraciones en la morfología de la onda P; sin embargo, las arritmias supraventriculares como la fibrilación atrial son las más comunes y con frecuencia predominan en el cuadro clínico. Un rápido reconocimiento y tratamiento pueden ayudar a prevenir complicaciones graves como la trombosis mural o la ruptura auricular. Se necesitan más estudios para establecer criterios diagnósticos unificados y para conocer la prevalencia real de esta entidad.

INTRODUCTION

Ventricular infarction (VI) is a well known pathology that in most of the cases of atrial infarction (AI), covers all the attention of the clinical presentation. A wide variety of presentations can make the diagnosis of this pathology more difficult. Most of the times it is associated with ventricular ischemia, but in cases of hypertrophy, myocarditis, COPD (chronic obstructive pulmonary disease), pulmonary hypertension or muscular dystrophy, AI can be an isolated disease.[1,2] The two atria can be compromised, or only one of them, being the right atrium the most frequent one.[3]

Almost a century ago, Clerc et al. described the first case report documented in literature,[4] and in 1942 a case series was described by Cushing et al.[5] Until today, there are no unified criteria for the diagnosis of AI.

The presence of supraventricular arrhythmias, such as atrial fibrillation, wandering pacemaker, atrial tachycardia, and atrial premature complexes, might suggest the existence of AI in the context of an acute coronary syndrome, as only 20% of cases of isolated VI present supraventricular arrhythmias, differently occurs in AI, in which the incidence increases up to 70%.[6]

Not only arrhythmias are present in these patients, more threatening complications such as thrombosis, atrial wall rupture and heart failure decompensation, can lead to a high mortality.[2,7]

The purpose of this review is to bring attention to a frequently unnoticed disease.

Risk factors and pathophysiology

An exact incidence of AI in admitted patients with VI is unknown, autopsy studies had been

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broadly variable with incidences that range from 0.7% to 42%,[2] a bigger study conducted by Cushing et al, demonstrated that 31 of 182 cases of VI resulted in atrial ischemia, with an incidence of 17%, proven with autopsy examination.[5]

The main cause of AI, as in VI is atherosclerosis,[2,8-11] it has also been associated with other entities like COPD with cor pulmonale, elevated chamber pressure plus hypoxia, that is consequence of the pulmonary disease itself,[2,8,9] primary pulmonary hypertension,[2,8-10] muscular dystrophy and Friedreich's ataxia.[2,9]

Due to the thin atrial wall (2-3 mm), most AI are transmural,[2,8] they occur mainly in the right atrium and are more frequently found on the atrial appendages;[1-3,5,8-10] when the right coronary artery is occluded it does commonly in the first 2-3 cm, therefore compromising the atrial branches; interestingly in the study conducted by Cushing et al. occlusion of left coronary artery and its atrial branches occurred in 65% of cases, but the incidence of AI was still higher in the right atrium. This could be explained by the higher oxygen concentration in the left atrium, suggesting that there may be other mechanisms involved.[5,8]

Nevertheless, mostly of AI occur concurrently with VI,[1,2,5,8,11] in this context, left ventricle infarcts are more prevalent, probably explaining why in some series the left atrium is mostly compromised.[11]

The AI occurs when blood supplying arteries are occluded *(Figure 1)*, and some of its clinical and electrocardiographic (ECG) manifestations, like supraventricular tachycardias,[1,3,8,11,12] are explained by the compromise of structures such as the sinoatrial (SA) node and atrioventricular (AV) node, which are irrigated by branches of the main arteries that nourish the atria.

The ramus ostii cava superioris (ROCS) originates in 60% of people from the proximal right coronary artery (RCA), and in 40% from the proximal left circumflex artery (LCx); irrigating the SA node through its course along the atrium, passing across the interatrial groove forming the interatrial branches, towards its ending near the superior vena cava opening. The right and left intermediate and posterior atrial arteries, branches from the RCA and LCx respectively, anastomosing with the ROCS in the interatrial groove or over the atrium body. The AV node artery arises commonly from the RCA (87%), in 7% of cases from the LCx and in 10% from both. Due to the variability in atrial blood supply, the clinical and ECG findings are inconsistent.[8,13]

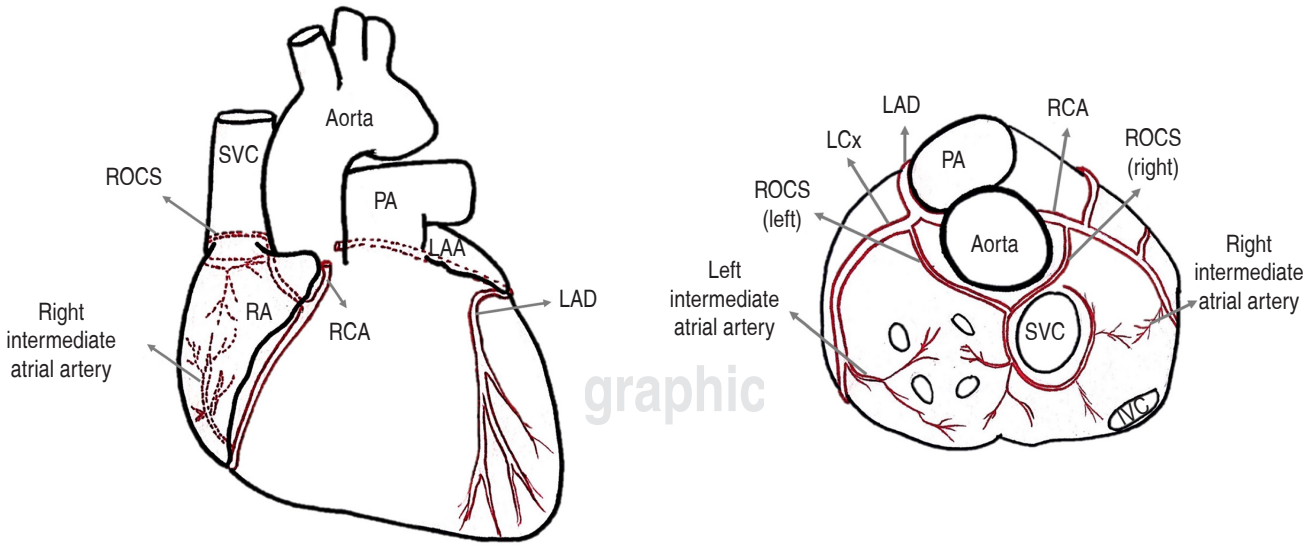

Figure 1: Atrium blood supply (SVC = superior vena cava, IVC = inferior vena cava, PA = pulmonary artery, RA = right atrium, LAA = left atrial appendage, RCA = right coronary artery, LAD = left anterior descending, LCx = left circumflex, ROCS = ramus ostii cava superioris).

Diagnosis

To this day, there are no unified criteria for the diagnosis of AI. The clinical presentation depends mostly on the area and extension of the affected myocardium.[8] In addition, ECG findings are subtle and nonspecific, making the diagnosis difficult.[14]

AI associated with VI is the most common type, especially with acute inferior and right VI;[15,16] however some cases of isolated AI have been described.[17] In 1991, Wong et al. concluded that if a patient presents angina, paroxysmal supraventricular arrhythmias, changes in the PTa segment and elevation of cardiac enzymes, without evidence of VI, an isolated AI is a probable diagnosis.[18]

In 1948, Hellerstein reported the first case of a patient that had an ante-mortem diagnosis of AI based on the ECG.[1] The ante-mortem diagnosis depends on the ECG findings, based on elevations and depressions of the PTa segment (representing atrial repolarization) and changes in the P wave; under normal conditions atrial repolarization in the ECG takes place at the same time as the ventricular depolarization (QRS complex), explaining why it is not usually seen in the ECG, as the QRS complex voltage is higher. Conversely, a diseased atrium has its repolarization (PTa segment) earlier in the ECG, therefore the changes can be identified in the PR segment. However, these changes are not always present in the ECG, this might be due to the low voltage generated by the atria and because these changes are generally masked by the underlying alterations in the ventricular depolarization.[2,11] Also PR segment prolongation and P wave axis changes have been reported.[19]

Supraventricular arrhythmias are the most common finding, ECG must be done especially after these episodes are over and the sinus rhythm is reestablished, in order to look for AI signs.[8,11]

In 1961, Liu et al. reported six cases of patients with AI that also had VI, in which the ante-mortem diagnosis was done and confirmed with an autopsy.

The electrocardiographic criteria proposed by Liu et al.[11] are shown in *figure 2*.

However, these major criteria have not been observed in subsequent studies and have not yet been validated.[20]

Recently, Yildiz et. al conducted a retrospective study that included patients with inferior-wall STEMI, finding PTa segment displacement only in a few patients with AI and not in patients without this entity. In the P-wave parameters analyzed, the P-wave duration was longer, and the amplitude was lower in inferior leads in patients with AI than in the control group. They suggest a P-wave duration of ≥ 95.5 ms in lead II for AI diagnosis.[21]

Changes in the PTa segment usually last between a few hours to a few days. It is believed that these changes improve with infarction treatment. Besides, it is also believed that PTa deviations occur before any other ECG alterations.[6]

Liu et al. suggested that AI must be suspected when a patient presents atrial arrhythmias and an associated VI. In one of the cases described by Liu et al, the VI diagnosis confirmed with an autopsy was not seen in the ante-mortem ECG, but the AI was in fact seen. This is why it is advised that in the presence of ECG changes suggestive of AI, an associated VI must be assumed and treated.[11]

The sensitivity or specificity of the PTa segment deviations for the AI diagnosis are unknown.[6]

The infarction location, in theory, would determine the PTa segment deviation:

- When there is an ischemia of the posterior wall: PTa segment is elevated in lead II and III, with a reciprocal depression in lead I *(Figure 3)*.
- If the ischemia is located on the anterior or anterolateral walls (including the right atrial appendage): PTa segment is elevated in lead I, with a reciprocal depression in lead II and III.[1]

Nevertheless, PTa segment deviations can also be present in pericarditis or sympathetic overstimulation,[22] and P wave abnormalities can also be seen in atrial enlargement and interatrial blocks.

Riera et al. published a case report in which they used vectocardiography as an additional diagnostic tool that helped determine atrial dilatation, showing notches in the P loop suggestive of AI, even though, no alterations were found in the complementary echocardiography done at this time.[17]

Bryce et al. concluded in 2017 that the presence of interatrial block is more common in patients with multi-vessel coronary disease *(Figure 4)*. They also suggested that this block is the result of persistent atrial ischemia.[22]

Echocardiography

There are limitations in the visualization of the atria by conventional echocardiography. Transesophageal echocardiography (TEE) is better for the evaluation of atrial wall motion and presence of thrombi.[2,9] In patients with inferior wall infarction with right ventricle compromise, the TEE might be useful in order to identify atrial ischemia.

In 1993, Vargas-Barron et al. described the following findings in TEE:[23,24]

- Akinesis of the right atrial free wall, despite left atrial contraction.
- Dilatation with spontaneous echo contrast effect in the right atrium.
- Thrombosis at the site of parietal akinesis.
- Lack of Doppler A wave across the tricuspid valve with normal mitral A wave.

Other findings include inversion of the normal interatrial septal convexity in patients with associated right ischemic ventricular dysfunction.[24]

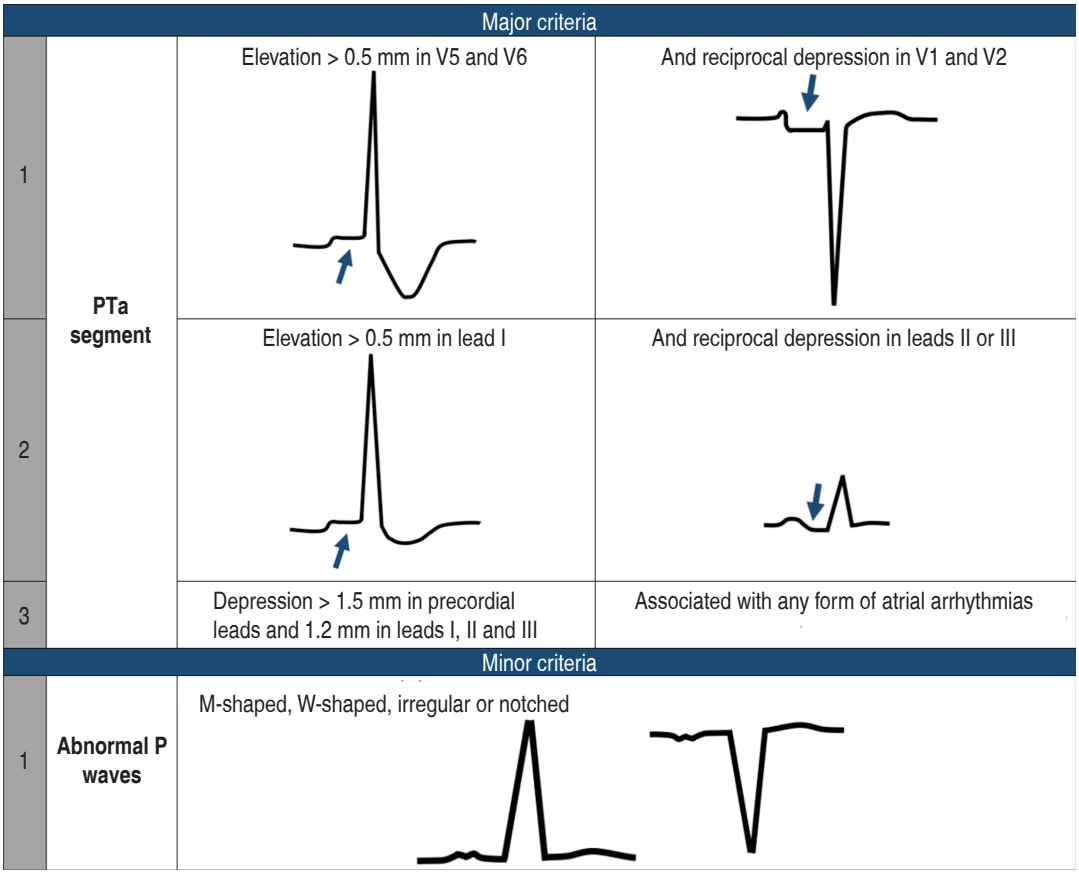

Figure 2: Electrocardiographic criteria proposed by Liu et al.

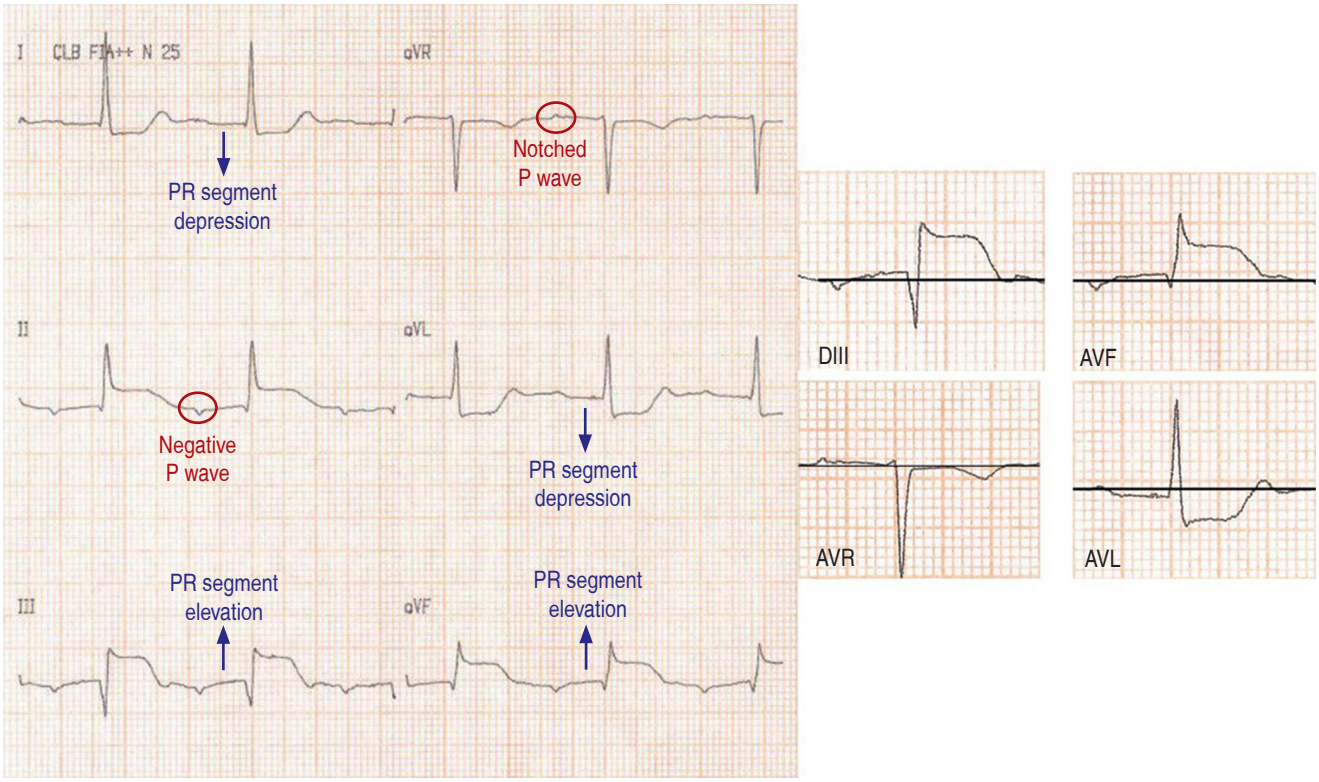

Figure 3: ECG showing PR segment elevation in lead III and aVF, PR segment depression in lead I and aVL, and P wave abnormalities with an associated ST segment elevation in the inferior leads.

Complications

Initially, some tachyarrhythmias might occur causing hemodynamic repercussion and cardiac failure decompensation.[1] Nielsen et al. carried out a study in 1992 finding that the presence of displacements in the PTa segment at the moment of admission in patients with VI helps predict the development of supraventricular arrhythmias the following days.[6]

The recognition of AI is important, due to the severity of its complications when left untreated:

- **Arrhythmias:** atrial fibrillation, atrial flutter, premature atrial complexes, paroxysmal atrial tachycardia, sinus tachycardia, sinus arrest, wandering pacemaker, nodal rhythm, sinus bradycardia and atrioventricular blocks have been described.[25] These typically start and end suddenly. They have an incidence of 61-74%, and are more frequent than in VI alone.[1,2,6,16] The presence of morphological changes in the P wave could be a predictor of new onset atrial fibrillation.[20]
- **Mural thrombosis with thromboembolic episodes:** intramural thrombus has an incidence of 80-84%[26] and might lead to a pulmonary embolism, which is more common because of the higher incidence of right AI, or to a systemic embolism (e.g. towards the brain).[1,2,17] Transmural ischemia usually leads to thrombus formation.[27] However, Lanjewar et al. reported the case of an AI which occurred due to a thrombus in the right atrium appendage in a patient with thyrotoxicosis and atrial fibrillation, with normal coronary arteries.[28]
- **Atrial wall rupture:** signs of cardiac tamponade must always be kept in mind. In 1994, Orcajo et al. reported the case of a female patient who presented sudden death due to a right atrial rupture, with

no electrocardiographic criteria for AI. They described an incidence of atrial wall rupture of 4.5%, with a clinical presentation similar to the ventricular rupture, the role of an early diagnosis and treatment is vital in order to save the patient's life. Other authors suggest that atrial rupture could cause death more slowly than ventricular rupture, citing that some patients can survive more than 24 hours, providing a longer time to perform a surgical repair.[7] In 2007, Rose et al. described the case of a patient with left VI, who deteriorated and later died, and whose autopsy revealed a left atrial wall rupture.[10]

- **Loss of atrial kick:** it generates a decrease in cardiac output with hemodynamic repercussion, ending up in a cardiogenic shock. Nevertheless, it is not believed that an isolated AI can cause acute cardiac failure.[9]
- **Left atrial enlargement:** an experimental study done by Aguero et al. revealed that pigs in which left AI was induced, had higher degree of left atrial dilation in resonance images and ischemic mitral regurgitation than those in which circumflex atrial branch was not occluded.[29]

It is believed that the addition of atrial ischemia to a VI implies a worse prognosis and higher morbimortality.[24]

Treatment

There are no additional treatment recommendations in the management of VI with suspected atrium compromise, the goals of treatment are coronary reperfusion and returning or maintaining sinus rhythm.[1,2,8,9,11,12] Even Liu et al. recommend treating isolated AI findings like VI,[2,11] as it could be ventricular compromise without electrocardiographic changes.[6,11]

If supraventricular tachycardias are present, some recommend rate control with beta blockers,[2,6,8,9] considering cardioversion in case of instability.[8]

Anticoagulation should be considered, taking into account that intramural atrial thrombus are commonly found,[1-3,9,11] and systemic or pulmonary embolism must be prevented.[1,2,11]

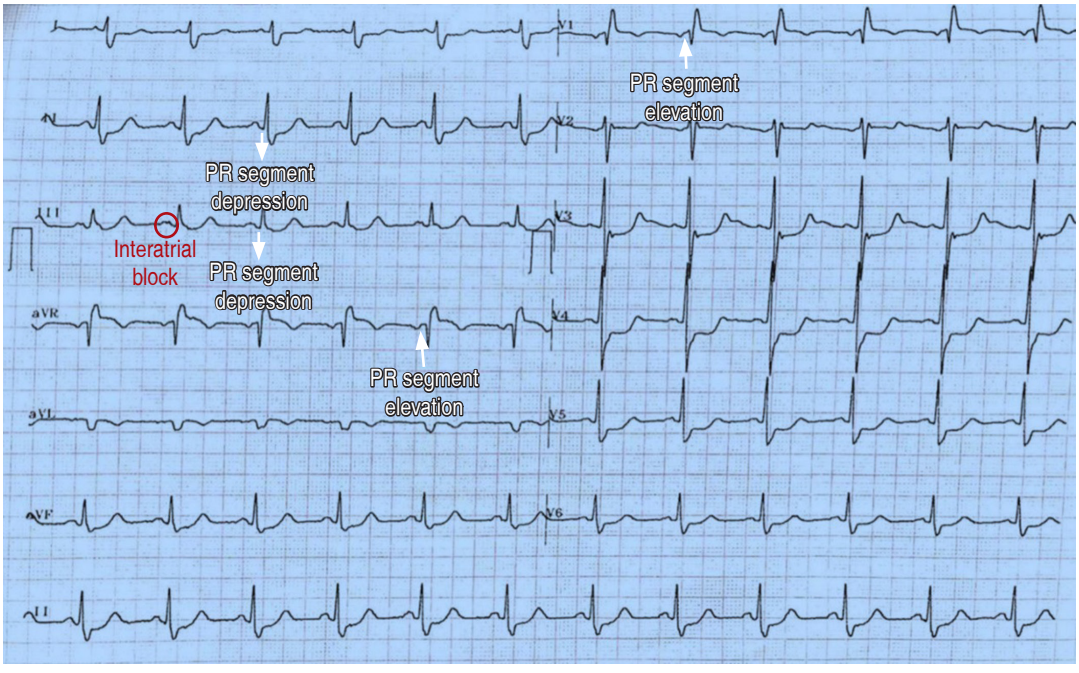

Figure 4: ECG of a patient with ventricular infarction due to left main coronary artery occlusion, also presenting complete bundle branch block, PTa segment alterations in V1 and aVR and interatrial block in lead III.

In case of suspected rupture of the atrial wall (e.g. cardiac tamponade), a prompt surgical repair should be carried out.[2]

CONCLUSIONS

AI is a frequently unnoticed disease because it commonly occurs in the context of VI, nevertheless it can present as an isolated disease with important complications, being a prognostic determinant for patients, thus needing to be recognized.

Its main risk factor is atherosclerosis and it develops when atrium arteries are occluded. Clinical and electrocardiographic findings are inconsistent, making the diagnosis difficult and explaining why there are no yet unified diagnostic criteria. It should be suspected in patients with myocardial ischemia, supraventricular arrhythmias, changes in the P wave and PTa segment displacement. Management is based in achieving coronary reperfusion, maintaining sinus rhythm and preventing or treating complications.

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