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Narrative Review

A Comprehensive Review of Atrial Infarction

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Abstract

Atrial infarction is often undiagnosed in patients with underlying ischemic heart disease and is identified only later, upon autopsy. One of the main challenges in diagnosing the condition is its localization within the affected atria. Treatment of atrial infarction focuses on acute reperfusion therapy, long-term management of cardiovascular disease risk factors, consideration of antiarrhythmia medications, and anticoagulation therapy. This review covers the anatomy of the atrial vasculature, complications associated with atrial infarction, diagnostic criteria for use of electrocardiography and other imaging modalities, and overall prognosis and management.

Keywords: Myocardial infarction; arrhythmias, cardiac; atrial function

Introduction

Atrial infarction is frequently unrecognized and undiagnosed, often identified only incidentally upon autopsy, and it commonly concurs with ventricular myocardial infarction (MI). Its prevalence in the existing literature varies substantially, ranging from 0.7% to 42% among patients with confirmed ventricular MI, although some studies suggest a true prevalence closer to 17%.[1] Major risk factors for atrial infarction include atherosclerotic heart disease, MI, chronic obstructive pulmonary disease, pulmonary hypertension, muscular dystrophy, and Friedreich ataxia. According to Ventura et al,[2] primary pulmonary hypertension is the leading cause of isolated atrial infarctions, likely secondary to increased hypoxia and oxygen demand of the atrial myocytes.

This overview covers the vascular anatomy of the atria, clinical presentation of atrial infarction, current diagnostic strategies, complications, prognosis, and treatment. A key challenge in diagnosing atrial infarction is accurately localizing the infarction. Common complications associated with atrial infarction include supraventricular arrhythmias, thromboembolic phenomenon, and congestive heart failure. Treatment strategies focus on reperfusion therapy, the standard of care for all MIs, along with chronic management of any complications. Increasing awareness of atrial infarction can help improve diagnosis and treatment, ultimately enhancing patient outcomes.

Anatomy

Understanding the diagnostic findings in atrial infarction requires a detailed review of atrial vascular anatomy, as illustrated in Figure 1 through Figure 3.[3]

The primary blood supply of the atria originates from branches of the right coronary artery and left circumflex coronary artery. The 3 main vessels are the anterior, intermediate, and posterior atrial branches. The anterior and

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posterior circulation connects through the anastomosis of the Kugel artery, which can originate from the right coronary artery or the left circumflex artery. Another important source of atrial blood supply is the ramus ostia cava superioris, which travels inferiorly through the interatrial groove on the anterior surface, providing the interatrial branches that terminate at the base of the superior vena cava.

Key Points

- This review describes the prevalence, complications related to, and management strategies for atrial infarction.
- This review discusses the atrial blood supply and common locations of atrial infarction.
- This review discusses possible diagnostic strategies using ECG criteria, along with other imaging modalities.

Abbreviations

CHA_2DS_2-VASc, congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack or thromboembolism (doubled), vascular disease, age 65 to 74 years, sex category

ECG, electrocardiography

MI, myocardial infarction

Diagnosis

Because atrial infarction most often presents concurrently with ventricular MI, distinguishing its presentation can be difficult. Signs suggestive of atrial infarction include typical anginal chest pain, elevated cardiac biomarkers, supraventricular arrhythmias, and related changes on electrocardiography (ECG).

Many studies have attempted to identify substantial ECG changes for diagnosing atrial infarction. Electrocardiographic changes associated with atrial infarction were first described in 1939 by Langendorf et al[4] and later refined by Liu et al[5] in 1961. Diagnosing atrial infarction by ECG can be challenging, however, because these changes are often subtle and may be overshadowed by changes related to concurrent ventricular depolarization or infarction.

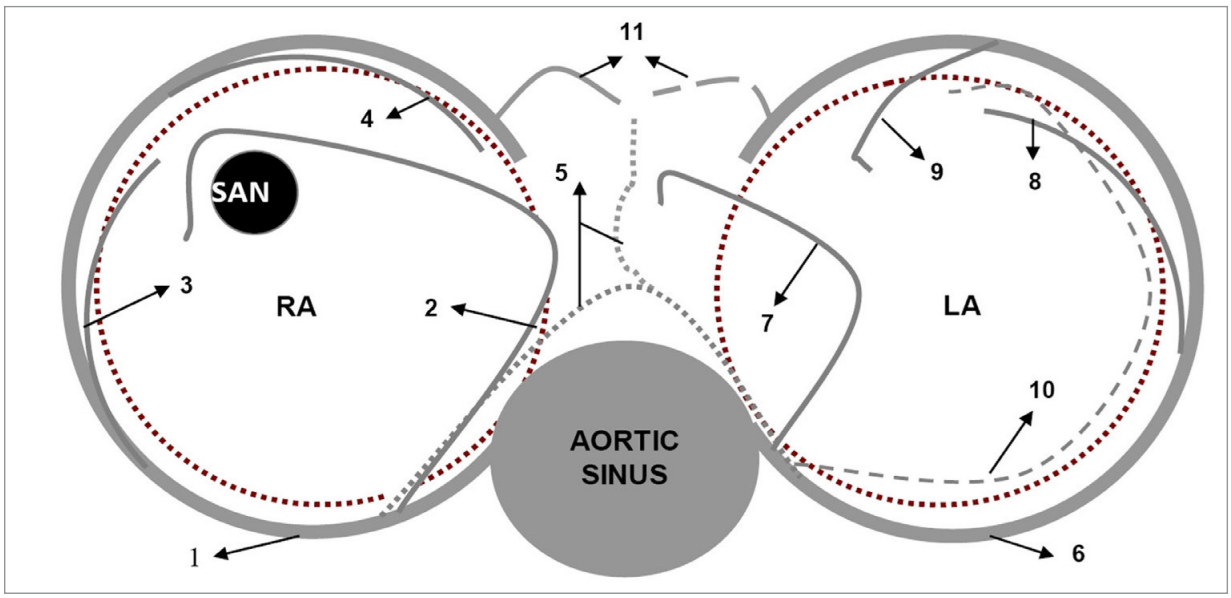


Fig. 1 The dotted circles represent the outline of the atrial walls. Black arrows detail perfusion territory. The coronary artery branches are numbered as follows: (1) right coronary artery, (2) right anterior atrial branch, (3) right intermediate atrial branch, (4) right posterior atrial branch, (5) Kugel arterial anastomotic branch, (6) left circumflex artery, (7) left anterior atrial branch, (8) left intermediate atrial branch, (9) left posterior atrial branch, (10) left atrial circumflex branch, and (11) atrioventricular nodal branch.

LA, left atrium; RA, right atrium; SAN, sinoatrial node.

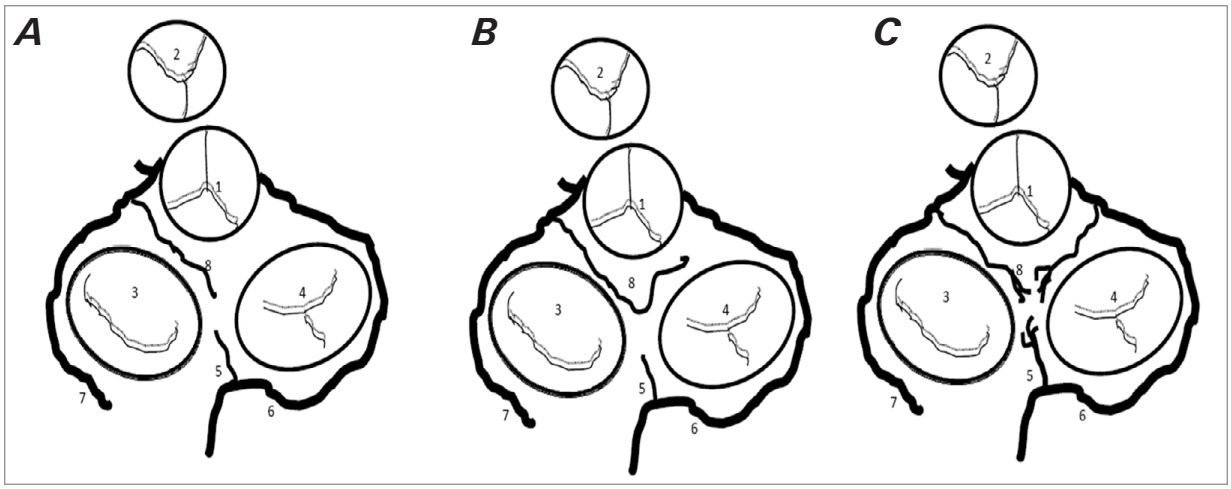


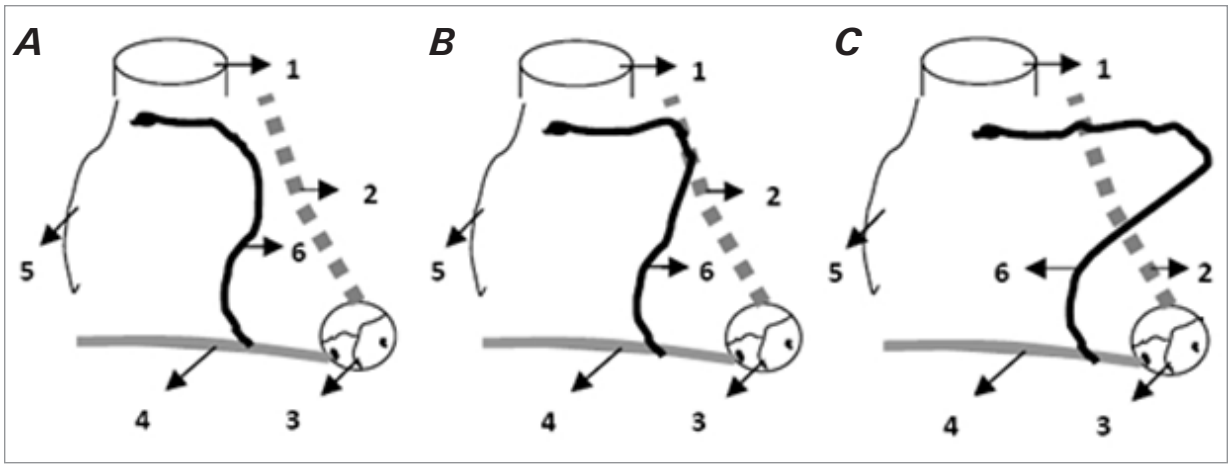
Fig. 2 Schematic representation of the heart shows the 3 anatomic variations of the Kugel artery, arteria anastomotica auricularis magna, and superior view: (**A**) The arterial vessel arises from left circumflex artery, meeting with the vessels at the crux; (**B**) the vessel arises from the left circumflex artery or its branches and takes an abrupt turn to meet the vessels at the right atrium anterior wall; (**C**) the vessel arises from the left circumflex artery and fans out to meet with vessels from the right coronary artery.

(1) Aorta, (2) pulmonary trunk, (3) mitral valve, (4) tricuspid leaflet, (5) atrioventricular nodal artery, (6) right coronary artery, (7) left circumflex artery, and (8), anastomotica auricularis magna.

Electrocardiographic criteria for atrial infarction are furthermore subdivided into major and minor criteria. Major criteria include PR-segment elevation greater than 0.5 mm in lead V_5 and lead V_6, with reciprocal PR-segment depression in lead V_1 and lead V_2; PR-segment elevation greater than 0.5 mm in lead I, with reciprocal depression in lead II and lead III; and PR-segment depression of more than 1.5 mm in precordial leads and 1.2 mm in leads I, II, and III associated with any atrial arrhythmias.[5] Minor criteria are abnormalities in the P-wave morphology, flattening to an M or a W shape, or an irregularly notched *P* wave.[5] These ECG criteria are illustrated in Figure 4. A 30-year autopsy study highlighted an association between "a fraction of a millimeter" PR-segment depression and atrial infarction.[6] PR-segment changes have been proposed as an early indicator of acute atrial infarction, but they often normalize within hours.[7] Although these criteria provide a basic guide for identification of atrial infarction, they lack sufficient sensitivity and specificity. A study performed by Van Diepen et al[8] found that none of the 666 study patients experiencing ST-segment elevation MI met any major ECG criteria for atrial infarction. This finding is consistent with the transient nature of PR-segment changes, which can be masked by ST-segment elevation as a result of ventricular infarction.

Important indicators of atrial repolarization on ECG are the P wave and the PT segment. The P wave represents atrial depolarization, while the PT segment encompasses the electrical conduction system from the end of atrial depolarization to the beginning of ventricular depolarization. Underlying ischemia causes an imbalance in the depolarization and repolarization of atrial tissue, leading to characteristic elevation or depression in these segments. These changes are more specific for atrial infarction when reciprocal changes are present in perpendicular leads.

Visualizing atrial repolarization (PR segment), however, is challenging because it is disrupted by more prominent ventricular depolarization and repolarization (QRS-T segments). Ventricular depolarization, which has greater amplitude and electrical activity, often conceals the ST segment, which represents atrial repolarization. No diagnostic criteria currently exist for the localization of atrial infarction, but the a basic understanding of ECG can help predict potential electrophysiologic changes. Left atrial (posterobasal) infarction can be visualized by PT-segment elevation in lead II and lead III and reciprocal depression in lead I,[5] while right atrial (anterior/anterolateral) infarction can be visualized by PT-segment elevation in lead I and reciprocal depression in leads II, lead III, and leads V_2 through V_4.[5]



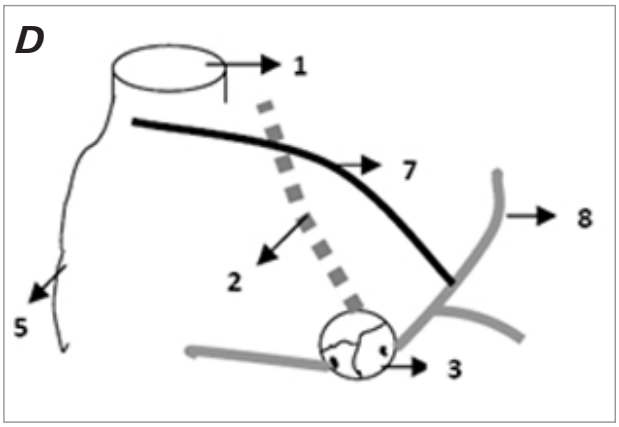


Fig. 3 (**A**) The sinus nodal artery does not reach the anterior interatrial groove; (**B**) the sinus nodal artery reaches the anterior interatrial groove but does not go beyond it; (**C**) the sinus nodal artery crosses the anterior interatrial groove; and (**D**) the left sinus nodal artery crosses the anterior interatrial groove.

(1) Superior vena cava, (2) anterior interatrial groove, (3) aortic sinus, (4) right coronary artery, (5) appendage of the right atrium, (6) right sinus nodal artery, (7) left sinus nodal artery, and (8) left circumflex artery.

In addition to PT-segment and P-wave morphology changes, studies of selective acute atrial branch occlusion in animal models suggest that specific R-wave and ST-segment changes can also be observed in atrial infarction.[9] In a study by Amorós-Figueras et al,[9] progressive R-wave enlargement and ST-segment elevations were noted within the first 30 minutes of infarction, with subsequent indirect changes in P-wave amplitude and duration after 2 hours.

There is increasing use of additional diagnostic tools for atrial infarction, including transesophageal echocardiography, multigated acquisition cardiac blood pool scanning, and cardiac magnetic resonance imaging. Transesophageal echocardiography can identify atrial infarction by demonstrating dilatation of the right atrial cavity, contractile dysfunction, akinesis of the right atrial free wall, and absence of A waves across the tricuspid valve.[10] Multigated acquisition cardiac blood pool scans can characterize atrial dysfunction, atrial kick, and atrial filling, identifying potential areas of atrial infarction.[11] Cardiac magnetic resonance imaging has been used to determine overall atrial function and detect scarring or fibrosis.[12] Future research studies and data collection are required to determine the use of these imaging modalities in the diagnosis of atrial infarction.

Complications

Overall, the complications associated with atrial infarction are not well understood because diagnosis is often made post mortem and can be missed during autopsy.[13] Patients with a history of cardiac disease may have underlying complications attributed to other etiologies until atrial infarction is discovered. The most common complications associated with atrial infarction are supraventricular arrhythmias, thromboembolic events, and diastolic heart failure.[2]

Arrhythmias are the most common complication of atrial infarction, including atrial fibrillation (AF), atrial flutter, wandering atrial pacemaker, and supraventricular tachyarrhythmia. Arrhythmias have been observed in up to 70% of patients with atrial infarction compared with 20% of patients with isolated ventricular

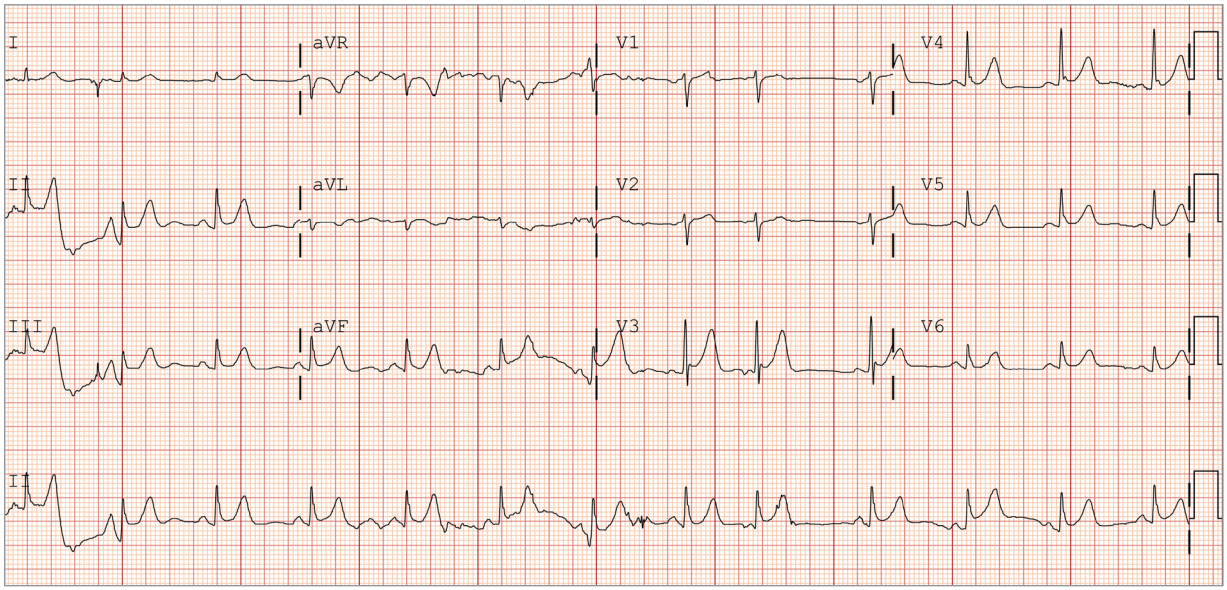


Fig. 4 An electrocardiogram demonstrates criteria for atrial infarction—namely, a PR-segment depression in leads II, III, and aVF and a PR-segment elevation in lead aVR.

infarction.[7,13] Changes in P-wave morphology following atrial infarction are early predictors of the development of AF.[14] Scar formation increases the risk of reentrant circuit formation, leading to a higher incidence of arrhythmias.

Another common complication associated with atrial infarction is a thromboembolic event. An autopsy study of patients with prior MIs revealed that the presence of mural thrombi statistically significantly increased the likelihood of underlying atrial involvement.[1] Mural thrombi have been reported in approximately 85% of atrial infarction cases.[15] Literature on the prevention of mural thrombi formation with anticoagulation is limited; further research is needed to determine whether maintenance anticoagulation is required. Because arrhythmias such as AF and atrial flutter are common in patients with atrial infarction, anticoagulation should be considered based on congestive heart failure, hypertension, age ≥75 years (doubled), diabetes mellitus, prior stroke or transient ischemic attack or thromboembolism (doubled), vascular disease, age 65 to 74 years, sex category (CHA_2DS_2-VASc) score risk assessment.[16] Anticoagulation therapy based on risk assessment should theoretically reduce the frequency of thrombotic events seen in this population.

Another complication associated with atrial infarction is worsening diastolic congestive heart failure. One leading theory is that atrial infarction can result in the loss of atrial contraction, also known as the "atrial kick," which contributes approximately 20% of a person's cardiac output. Although the loss of atrial contraction may not substantially affect healthy individuals with appropriate compensatory pathways, it can be detrimental to individuals with underlying cardiac dysfunction. This loss leads to decreased ventricular filling pressures and cardiac output and may progress to cardiogenic shock and hemodynamic compromise.[5] Decompensation carries a high morbidity and mortality risk in patients with underlying heart disease because they are unable to compensate appropriately for these hemodynamic changes.

Finally, a rare complication associated with atrial infarction is atrial rupture. In a study on right atrial infarctions, atrial rupture was observed in approximately 4.5% of cases, typically resulting in hemodynamic collapse and pericardial tamponade.[15] Patients with atrial rupture have better survival outcomes in the first 24 hours (survival rate, 15%) than do patients with ventricular rupture (survival rate, 2%).[17]

Prognosis and Outcomes

Determining the overall prognosis of atrial infarction alone is challenging because it often coexists with other contributing cardiac or pulmonary etiologies. For instance, atrial infarction that occurs with a ventricular MI carries a much worse prognosis than primary atrial infarction secondary to underlying pulmonary

hypertension. Apart from cases in which patients who have experienced atrial rupture undergo life-sustaining surgery, most instances of atrial infarction are uncovered during postmortem examinations.

Treatment

Treatment of atrial infarction can be categorized as acute or chronic. During the acute phase, the focus should be on reperfusion therapy and on managing the condition like an acute MI. Chronic management should concentrate on reducing cardiac risk factors through lifestyle modifications, lowering lipids, controlling blood pressure, and implementing appropriate guideline-directed medical therapy aimed at positive cardiac remodeling.

Data regarding the use of antiarrhythmia agents in atrial infarction are limited, but early administration of β-blockers, particularly metoprolol, during acute MI has been shown to reduce the progression of supraventricular tachyarrhythmias. Cardioversion can be considered in patients with unstable disease, regardless of their anticoagulation status, if there is reason to believe that the arrhythmia originated from the acute infarction.[5]

In summary, patients who have experienced an atrial infarction should undergo treatment similar to patients who have underlying coronary artery disease and have a previous MI, with considerations for antiarrhythmia and anticoagulation therapy on a case-by-case basis.

Conclusion

Atrial infarction remains largely underdiagnosed, emphasizing the need for additional research to understand its pathophysiology, improve diagnostic capabilities, prevent complications, and establish guideline-directed management strategies. Further exploration of advanced imaging modalities could enhance the diagnosis and early management of atrial infarction. Recognizing this condition is crucial in guiding patient care and ultimately improving clinical outcomes.

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