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Review

Atrial myocardial infarction: A tale of the forgotten chamber

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ABSTRACT

It has been almost a century since atrial infarction was first described, yet data describing its significance remain limited. To date, there are still no universally accepted criteria for the diagnosis of atrial infarction. Atherosclerosis is the leading cause of atrial infarction but it has also been described in cor pulmonale and pulmonary hypertension. Atrial infarction almost always occurs concomitantly with ventricular infarction. Its clinical presentation depends largely on the extent and site of ventricular involvement. Atrial infarction can present with supraventricular tachyarrhythmias. Electrocardiographic (ECG) criteria for diagnosing atrial infarction have been described but none have yet to be validated by prospective studies. Atrial ECG patterns include abnormal P-wave morphologies, PR-segment deviations, as well as transient rhythm abnormalities, including atrial fibrillation, atrial flutter, atrial tachycardia, wandering atrial pacemaker (WAP) and atrioventricular (AV) blocks. Complications of atrial infarction include thromboembolic events and cardiogenic shock. There are no specific additional recommendations in the management of myocardial infarction with suspected involvement of the atria. The primary goal remains coronary reperfusion and maintenance of, or conversion to, sinus rhythm.

1. Introduction

Myocardial infarction (MI), an entity referring to irreversible damage or death of cardiac muscle, is the leading causes of morbidity and mortality worldwide. As the bulk of myocardium is predominantly located in the ventricles, most MI present clinically due to symptoms of ventricular myocyte ischemia, or the consequences of ventricular myocardial dysfunction. Atrial infarction, usually concurrent with ventricular infarction, is poorly recognized and understudied. Atrial infarction was first described in 1925 by Clerc and colleagues [1]. The first case series was published in 1942 by Cushing and associates [2]. Its incidence among patients with MI has been reported to range from 0.7% to 42% [2–7], mostly based on autopsy findings. The overall incidence is likely higher if MI survivors are also taken into account. To date, there are still no universally accepted criteria for the diagnosis of atrial infarction. Little attention has been given to this chamber and its clinical implications.

In this article we will review the etiology, pathophysiology, clinical presentation, diagnosis, and management of this frequently overlooked disease entity.

2. Etiology, pathophysiology and anatomic correlates of atrial infarction

The majority of atrial infarctions occur as a consequence of atherosclerotic heart disease. However, one case series found that chronic obstructive pulmonary disease with cor pulmonale can cause atrial infarction, likely secondary to a combination of hypoxia from the pulmonary disease and increased atrial pressure [8]. Another case report found an isolated sinus node infarction in a 31 year-old otherwise healthy female with primary pulmonary hypertension presenting with syncope [9].

In contrast to ventricular infarction, the majority of atrial infarctions involve the right atrium versus the left [10,16]. A review of case series showed that the right atrium is involved in 81% to 98% of cases, the left in only 2 to 19% [10]. Biatrial infarction occurred in 19 to 24% [10]. Interestingly, atrial infarction is more frequently found in the atrial appendages, rather than the lateral or posterior walls of the atria [10]. The considerably higher oxygen content of left atrial blood may explain the difference in incidence between right and left atrial infarction. Of note, there is one case series that found a greater incidence of left atrial involvement, hypothesizing it to be secondary to the greater incidence of left ventricular infarction [11].

The blood supply of the atria arises mainly from the ramus ostii cava superioris (ROCS). The ROCS originates from the proximal right coronary artery (RCA) in 60% of the general population and from the proximal left circumflex artery (LCx) in 40% [12]. Its course and termination however, remain constant regardless of its origin. The ROCS runs cephalad along

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the atria and posterior to the aorta. It then extends to the interatrial groove anteriorly and gives off interatrial branches, before encircling the base of the superior vena cava and terminating near its opening. The ROCS supplies the sinoatrial (SA) node. The right and left intermediate and posterior atrial arteries arise from the RCA and LCx, respectively, and often anastomose with the ROCS over the body of the atria or in the interatrial groove. The artery to the atrioventricular (AV) node originates posteriorly from the RCA in 83% of cases, from the LCx in 7%, and from both in 10% [12]. It courses through the crux cordis, deep into the coronary sinus to the base of the interatrial septum. There are other small atrial arteries that are inconsistently found and will not be mentioned in this review. Because the walls of the atria are so thin (2–3 mm), atrial infarction almost always results in transmural injury.

These variations in blood supply to atrial chambers, SA and AV node explain in part why the clinical presentation and electrocardiographic (ECG) abnormalities of atrial infarction are unpredictable and inconsistent. Manifestations of SA nodal abnormalities such as SA blocks and atrial fibrillation will depend on factors such as the location of the occlusion (proximal or distal to the ROCS origin) and the presence/absence of collateral vessels. Manifestations of AV nodal involvement such as AV nodal blocks are less frequent in left coronary artery lesions as it supplies the AV node in only 7–10% of patients.

3. Making the diagnosis

Atrial infarctions are almost always described concomitantly with ventricular infarction in the literature. Isolated atrial infarctions are rare [13,26]. Atrial infarction presentation depends largely on the extent and site of ventricular involvement. In addition, they may initially present with supraventricular tachyarrhythmias. These arrhythmias have been found to occur more frequently in combined atrial and ventricular infarctions compared to the ventricular infarction alone (61–74% vs 8%) [2]. They may also present as cardiogenic shock secondary to atrial rupture or loss of atrial "kick", as well as with stroke or other thromboembolic events [4,10]. The diagnosis of atrial infarction should be strongly considered in the presence of ischemic symptoms of chest pain together with elevated cardiac biomarkers, the presence of atrial arrhythmias, and ECG abnormalities as detailed below.

3.1. Atrial ECG patterns

The sensitivity and specificity of the ECG in atrial infarction is limited by the variations in coronary anatomy as presented above. Consistent ECG patterns (Figs. 1, 2a and b) are generally subtle because of the thinner atrial walls and their inability to generate a high enough voltage to be appreciated on the ECG. This atrial voltage is also often overshadowed by the depolarization of the larger ventricles. As a result, it can create inter-observer variations in interpretation. One study found considerable variations in interpreting PR-segment deviations among clinicians with different years of experience, although they had fair to moderate overall agreement rates [14]. In addition, as with ventricular infarction, the PR-segment is also affected by the presence of a previous myocardial infarction, pre-existing arrhythmias, or previous interventions, such as placement of a pacemaker or coronary-artery bypass surgery.

The diagnosis of atrial infarction rests largely on ECG findings. Though, several atrial infarction ECG patterns have been described, none have been validated by prospective studies. The first ECG description of atrial infarction was in 1939 which reported deviations of the PQ segment from the isoelectric line in leads II and III [15]. In 1942, experimental atrial infarction in 18 dogs, through ligation and cauterization of one or more atrial arteries, showed morphologic P wave changes such as notching, increase in amplitude and transient changes in contour; depression of the PR segment; or transient rhythm abnormalities, such as premature atrial contractions (PACs), atrial flutter, atrial tachycardia, wandering atrial pacemaker (WAP) and AV blocks [2]. However, there was no constant ECG pattern noted [2] and supraventricular dysrhythmias were the most consistent finding [2,5]. In 1944, Young and colleagues associated PR segment deviation with atrial infarction where an elevation of > 0.5 mm or depression of > 0.8 mm was considered abnormal [16]. Hellerstein described a case of biatrial posterior wall infarction that showed PR segment elevations in leads II and III with episodes of atrial fibrillation and high degree AV block [17].

There are other mechanisms that have been shown to produce P wave morphologic abnormalities and PR-segment changes besides atrial ischemia/infarction. Sympathetic overstimulation, pericarditis, atrial dilatation or hypertrophy, and intra-atrial blocks have been described [11,17–20]. Hypersympathetic activity produces descending PR segment, depressed J point and ascending ST segment with the P-R and S-T segments having concordant deviations. Pericarditis can cause ECG changes if the inflammation involves the epicardium or the visceral pericardium as the parietal pericardium is electrically inert. The most common ECG abnormality is a widespread saddle-shaped or upward concave ST elevation and PR depression with the reciprocal changes in aVR and V1 [21,22].

The most widely accepted ECG criteria of atrial infarction to date are those proposed by Liu et al. in 1961 [11]. They proposed major criteria as follows: (a) PTa (PR) segment elevation of more than 0.5 mm in leads V3 and V6 with reciprocal depression of PTa segments in leads V1 and V2; (b) PTa (PR) segment elevation of more than 0.5 mm in lead I with reciprocal depressions in leads II and III; and (c) PTa (PR) segment depression of more than 1.5 mm in the precordial leads with 1.2 mm depressions in leads I, II and II, associated with any atrial arrhythmia. Morphologic changes in the P wave, such as an irregular or notched shape, either in a "M" or "W" pattern, were considered minor criteria. They also suggested that atrial infarction should always be suspected in patients having an acute myocardial infarction with any form of supraventricular dysrhythmia.

However, two other studies do not completely agree with Liu and colleagues [18,23]. Burch stated that a fraction of a millimeter of PR segment depression was associated with atrial infarction in all cases based on his 30-year autopsy study [23]. A more recent study studied atrial ECG changes of 666 subjects patients with ST-elevation myocardial infarction did not find a single patient who fulfilled any of the Liu major criteria [18].

Localizing an atrial infarction (right, left, or biatrial) using ECG patterns is equally challenging and the evidence is limited and often conflicting

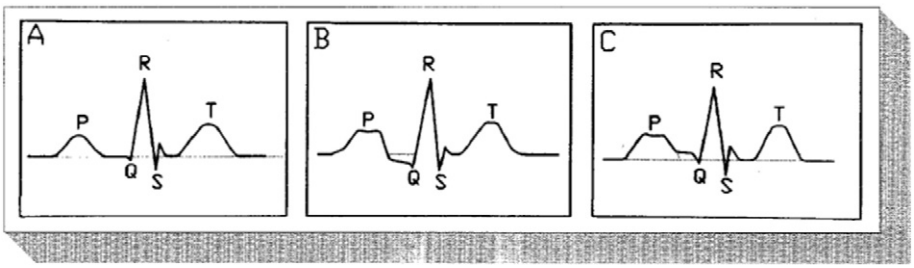

Fig. 1. Various ECG abnormalities of atrial infarction [31]. A — Normal P wave and PR segment. B — Notched P wave and PR segment elevation. C — Notched P wave and PR segment elevation.