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EDITORIAL COMMENT

Atrial Myocardial Infarction

A Neglected Stalker in Coronary Patients*

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In this issue of the *Journal*, the paper by Aguero et al. (1) shakes up the traditional understanding of the causative factors of atrial enlargement and atrial arrhythmias in patients with atherosclerotic coronary artery disease.

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In patients with myocardial infarction involving a sufficient amount of the left ventricle, increased left ventricular myocardial dysfunction causes elevated filling pressure, causing left atrial (LA) hypertension, which begets LA enlargement (LAE), which begets atrial fibrillation (AF), as shown across the top portion of **Figure 1**.

A somewhat different causative pathway (**Figure 1**) entails ischemic mitral regurgitation (MR) resulting from apical tethering of mitral leaflets (2,3). This also leads to LA hypertension and LA enlargement, and to the same cascade, leading to AF. Obviously, coronary patients may have both of these pathways (e.g., in ischemic MR with left ventricular dysfunction). This second causative pathway is a commonly assumed mechanism wherein AF results from nonischemic MR. Maybe a patient cannot have pathway 2 without also having pathway 1.

DEVELOPMENT OF AF AND ATRIAL DYSFUNCTION IN ATRIAL MYOCARDIAL INFARCTION

The production of LAE and AF in coronary artery disease is sometimes more complicated, and may involve a third mechanism (pathway 3 in **Figure 1**) that is not so familiar, but which has similar consequences. Aguero et al. (1) highlight the atrial changes in swine undergoing experimental circumflex occlusion. In this elegant animal model, occlusion of the atrial branch of the circumflex coronary artery caused LAE and AF (and exacerbated the MR). Atrial myocardial infarction (AtMI) is not as well recognized as a cause of some of the previously mentioned complications.

AtMI is not a new discovery; having been reported a century ago (4). Cushing et al. (5) elaborated on AtMI 75 years ago. Additional authors (4,6-10) also have studied infarction of the atrial myocardium, finding varying types of atrial dysfunction, which can result in various types of unsuspected complications, often subtle but still dangerous.

DIAGNOSIS OF AtMI IS DIFFICULT

Although AtMI is not a new discovery; it has mainly been ignored clinically, partly because of our limited capacity for diagnosis (4). Stenosis or occlusion in the arteries that feed the atria has not been on our radar screen. In fact, the atrial coronary branch is unmentioned in most angiographic reports. Our indifference about AtMI reflects the dominance of ventricular infarction, the effects of which are much more obvious by electrical, imaging, or clinical assessments than concomitant atrial infarction.

The currently available methods to diagnose atrial infarction are not specific, accurate, or commonly used. But apparently, AtMI "stalks" in the background, striking some of our patients without our recognition. Although myocardial infarction causes atrial arrhythmias in many patients, so do many other acute cardiac problems. When the patient is in sinus rhythm, AtMI often causes abnormal P waves and PR segments (4,6), but this has been easier to ignore than to clarify.

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Magnetic resonance imaging may have some value in this discussion, as shown by Aguero et al. (1), especially for differentiation of LA reservoir function, conduit function, and booster function. Others have used magnetic resonance imaging to detect LA fibrosis, which was associated with recurrence of atrial arrhythmias after radiofrequency ablation of AF (11).

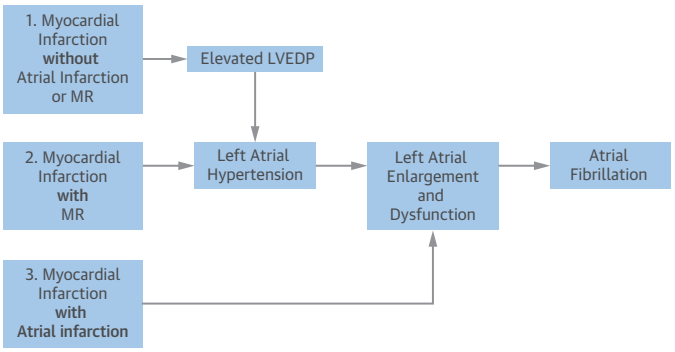
DIFFERENCES IN THE ORIGIN OF ATRIAL BRANCHES OF CORONARY ARTERIES BETWEEN SPECIES

I consulted several of my many friends who frequently do coronary angiography in pigs; none knew whether the coronary artery branches that perfuse the atria are different in humans than in other animals. Of the swine in which Aguero et al. (1) produced AtMI, all resulted from occlusion of the atrial branch of the circumflex artery. Circumflex origin of the atrial coronary supply was also found in a previous study of AtMI in sheep (7). Perfusion of the sinoatrial node in humans originated from the right coronary artery in 57% and from the circumflex in 54% (8).

Presumably, Aguero et al. (1) did not know before the experiment which of the swine would develop an occluded atrial artery and AtMI as a result of circumflex occlusion. It is interesting that they studied a larger number of animals with induced left anterior descending infarction than circumflex infarction. Perhaps the diagnosis of AtMI was not their original aim in developing this experimental model. If that is true, this accidental focus mimics the clinical dilemma; AtMI is not easily diagnosed. To their credit, the authors elegantly demonstrated atrial infarction using histopathologic evaluation, and correlated that with angiographic atrial branch occlusion and with atrial dysfunction by magnetic resonance imaging.

It is feasible to visualize the atrial coronary branches angiographically in humans (**Figure 2**). Álvarez-García et al. (8,12) was able to visualize the atrial coronary braches in all patients studied. Acute, accidental atrial branch occlusion occurred in 16% to 21% of patients undergoing elective stents to the proximal right or circumflex coronary arteries. Compared with patients without atrial branch occlusion, those with occlusion had a higher plasma high-sensitivity cardiac troponin T level, and more frequent periprocedural myocardial infarctions, interatrial conduction disturbances, PR-segment deviations, and episodes of atrial tachycardia and AF (8).

FIGURE 1 In Coronary Disease, 3 Potential Pathways May Lead to Atrial Dysfunction and Arrhythmias

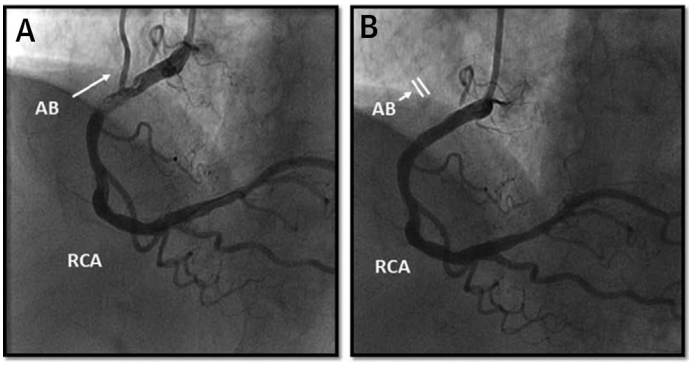


The first potential pathway involves left ventricular ischemia or infarction with elevation of left ventricular end diastolic pressure (LVEDP), which causes elevation in left atrial (LA) pressure, LA enlargement, and LA dysfunction, and therefore atrial fibrillation or other arrhythmias. In the second pathway, left ventricular infarction and dilation causes "functional" mitral regurgitation (MR), with apical tethering of mitral leaflets due to the apical displacement of papillary muscles, and the MR increases LA pressure with the same results. The third pathway, as documented by Aguero et al. (1), is less well proven and difficult to diagnose outside the experimental animal laboratory; it implies that atrial infarction causes LA dysfunction, enlargement, and subsequently atrial fibrillation.

IMPORTANCE OF AtMI

One autopsy study of post-infarction patients reported that the incidence of atrial infarction was 17% (6). A few authors have reported resolution of AF with percutaneous (13) or surgical revascularization (14) of

FIGURE 2 Angiographic Still Frames in a Case Exemplifying That the Atrial Branch of the Coronary Arteries Can Be Seen Well in Ideal Circumstances



(A) Before placement of a stent, the atrial branch (AB) of the proximal right coronary artery (RCA) is patent; and there is a moderate stenosis of the RCA at the origin of the AB. **(B)** After the stent is placed in the RCA, there is total occlusion of the AB, the location of which is shown by **hash marks**. Reprinted with permission from Álvarez-García et al. (8).

occluded atrial coronary branches, with improved clinical effects. However, it is uncertain how to determine if AtMI is really a clinically important finding in a given patient, or whether diagnosing AtMI will allow effective treatment through available therapeutic options.

CAN AtMI CAUSE MR?

Because AtMI causes AF and LAE, it is worth our attention. However, the issue of causality in this landscape is difficult and teleological. The current paper by Aguero et al. (1) also found that MR was more frequent in the animals with AtMI than those without AtMI. Their hypothesis that AtMI causes MR is more difficult for me to understand or justify. Several of these pathways may coexist; ischemic MR may occur whether or not there is LA remodeling as a result of AtMI.

ATRIAL INFARCTION IS SUBTLE AND POORLY IDENTIFIED

The clinical and electrical signs of AtMI are subtle, which makes diagnosis difficult. Its clinical manifestations are not always attributed to their true origin. It has been known for decades that MR of any cause, ischemic or nonischemic, can challenge the myocardial and electrical function of the atria. Aguero et al. (1) have reminded us that atrial dysfunction and fibrillation can also result from AtMI. AtMI is dangerous. It can sneak unrecognized into the lives of patients having acute coronary syndromes.

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