Case report

A 64-year-old Caucasian female, with a one-week history of stress angina. She was admitted to the hospital 2 hours after the onset of oppressive retrosternal pain at rest.

Risk factors: hypertension, smoker, dyslipidemia and diabetes.

Figure 1 shows electrocardiography/vectorcardiography (ECG/VCG) at admission.

Echo: Ventricular and atrial chambers of normal size (left atrial (LA) size: 30 mm); mild to moderate reduction of left ventricular ejection fraction = 41% by anterior akinesia.

The percutaneous coronary intervention was indicated, and two drug-eluting stents were implanted.

Introduction

Atrial infarction (AI) is rarely diagnosed before death because of its characteristically subtle and nonspecific ECG findings. AI occurs in 0.7 to 52% of ST-elevation myocardial infarctions. Its incidence in autopsy has been widely variable, from 0.7 to 42%, with a large series of 182 patients demonstrating an incidence of 17%. Ischemic damage to the atrial myocardium is usually associated with infarction of cardiac ventricles, but isolated AI can occur.2

AI ECG patterns

The ECG patterns of AI are generally subtle because of the thinner atrial walls and their inability to generate enough voltage to be appreciated on the ECG. This atrial voltage is also often eclipsed by the depolarization of the larger ventricles. Although several AI ECG patterns have been described, none have been validated by prospective studies. The first description of “infarctus auricularis” was made 93 years ago by Cler.5 Twenty-two years later, Langendorf reported one case of AI found at autopsy that in retrospect could have been recognized antemortem from ECG changes.4 Hellerstein reported the first case with the correct antemortem diagnosis of AI confirmed by necropsy.5

There are other potential causes for P wave morphologic abnormalities and PR-segment displacements besides AI. Sympathetic overstimulation, pericarditis, atrial enlargement, and interatrial blocks have been described.6 Pronounced sympathetic activity produces a descending PR-segment, depressed J point and ascending ST segment with the PR and ST 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.

Accepted ECG criteria of AI are those proposed by Liu et. Al.7

a) Major:
• PR-segment elevation > 0.5 mm in leads V3 and V6 with reciprocal depression in leads V1 and V2 of small amplitude;
• PR-segment elevation > 0.5 mm in lead I with reciprocal depressions in II-III;
• PR segment depression of >1.5 mm in precordial leads with 1.2 mm depressions in I, II and II, associated with atrial arrhythmia.

b) Minor:
• P wave with M-shaped, W-shaped, or notched; depression of the PR segment of small amplitude without elevation of this segment in other leads cannot be regarded by itself as positive evidence of AI.
• Patients having an acute myocardial infarction with any form of supraventricular arrhythmias, such as atrial fibrillation, atrial flutter, atrial tachycardia, wandering atrial pacemaker and atroventricular blocks.8

Regarding the location of AI, the literature evidence is limited and often conflicting. The right atrium (RA) is involved five times as often as the LA.1

Main complications of AI are: supraventricular arrhythmias, atrial rupture, cardiogenic shock and thromboembolic phenomena in the brain or lungs. Diagnosis currently is made in an appropriate clinical setting with characteristic P-wave shape, eventually the Bayés’s syndrome (complete interatrial block in the Bachman region associated with supraventricular arrhythmias).9 Theoretically, PR-segment displacements should correlate to the location of the AI in the same manner as ST-segment displacements in ventricular infarction. Thus, involvement of the laterobasal (formerly dorsal) wall, which corresponds to the LA, will result in PR-segment elevation in leads II and III with reciprocal depression in lead I.9 Likewise, involvement of the anterior

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or anterolateral wall, which corresponds to the LA, will produce PR-segment elevation in lead I with reciprocal depression in leads II, III and the anterior precordial leads V2-V4. However, there are no universally accepted criteria.

Discussion

Detailed ECG analysis revealed PR-segment displacement in several leads. With the aim to clarify this doubt, we isolated the P loop by VCG and enhanced its size 32-fold. We found that the P loop in VCG fulfilled criteria for biatrial enlargement (“Erlenmeyer-like” shape) (Figure 2) with notches in the central portion of the loop; this confirmed the suspicion of associated AI. The apparent contradiction of an atrial abnormality in VCG in conjunction with apparently normal atria on echocardiography could be explained by the fact that the echocardiogram is not an optimal method to evaluate the size of the RA and ventricle, particularly in the absence of concomitant right ventricular enlargement; therefore, enlargement of the RA could go unnoticed.

On the other hand, LA dilatation is not unexpected in an extensive anterior infarction with increase in the end-diastolic pressures. However, in the initial stages post-MI, the atrial chamber size can still be normal, although VCG shows a clearly abnormal P loop. In Figure 2, the comparison with the normal P loop is shown in the 3 planes in this case (AI with biatrial enlargement).

The role of atrial coronary perfusion is incompletely understood. One of the main limitations of our current understanding is that the origin of posterior LA coronary irrigation is unknown.10

Currently, three coronary branches supplying blood to the atria are known:

1. The right anterior atrial artery or sinus node artery, and other small branches arising from the right coronary artery, such as the right intermediate atrial artery.
2. The “ramus ostii cavae superioris” or left anterior atrial artery which arises from the left main coronary artery, the proximal portion of the left circumflex (LCX), obtuse

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**Figure 1** – ECG/VCG correlation. A) ECG diagnosis: Left atrial enlargement (positive Morris index), PR-segment depression in I, II, III and aVF, low QRS voltage in the limb leads (the amplitude of all the QRS complexes in these leads is < 5 mm), QS Pattern from V1 to V5, and low r voltage wave in lead V6, ST-segment elevation convex upward. B) VCG diagnosis: combination of anteroseptal anterior and anterolateral infarction: QRS loop directed to the back and minimally to the left near the orthogonal Z lead. The T-loop directed to the front with broad QRS/T angle (≈170°). Conclusion Acute extensive anterior myocardial infarction. Possible association with atrial infarction.
Figure 2 – Comparison between normal P loops and the present case. Frontal plane: In the present case, the maximal vector voltage is > 0.2 mV, and the morphology is broad with a notch in the middle portion (arrows). Right sagittal plane: The maximal anterior forces are ≥ 0.06 mV and maximal posterior forces are > 0.04 mV. Biaatrial enlargement. Horizontal plane: the normal P loop maximal vector location is located between +50° and -45°, maximal vector voltage is < 0.1 mV, maximal anterior forces are up to 0.06 mV and maximal posterior forces are up to 0.04 mV. In the present case, anterior and posterior forces exceed these values. Conclusion: Biaatrial enlargement and suspicion of AI by notched P loop in the frontal and right sagittal plane. RA: right atrium; LA: left atrium.

marginal, or diagonal coronary arteries. In the present case, the coronary obstruction occurred in the proximal portion of the LAD, consequently also the diagonals that can irrigate the LA causing AI in this structure.

3. The branches of LCX. These branches provide irrigation for the LA.

Conclusion

Though AI was first reported 89 years ago, its recognition remains elusive. AI should be suspected in any patient who presents with typical chest pain, elevated cardiac biomarkers and ECG changes consistent with AI: PR-segment deviations (elevation and depression), the presence of abnormal P-wave shape (M-shaped, W-shaped, irregular or notched) and/or presence of supraventricular tachyarrhythmias. P loop VCG analysis appears to be a valuable diagnostic tool.

Author contributions

Conception and design of the research, Writing of the manuscript and Critical revision of the manuscript for intellectual content: Riera ARP, Barros RB, Silva e Sousa Neto AF, Raimundo RD, Abreu LC, Nikus K; Analysis and interpretation of the data: Riera ARP, Nikus K.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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