Right atrial infarction, atrial arrhythmia and inferior myocardial infarction form a missed triad: A case report and review of the literature

Douraid K Shakir FICMS (Card) FICMS (Med) MRCP (UK) DM (Med) MBChB^{1,2}, Salah OE Arafa MRCP (UK)¹

DK Shakir, SOE Arafa. Right atrial infarction, atrial arrhythmia and inferior myocardial infarction form a missed triad: A case report and review of the literature. *Can J Cardiol* 2007;23(12):995-997.

Atrial infarction is rarely diagnosed before death because of its characteristically subtle and nonspecific electrocardiographic findings. These findings may be overshadowed by changes associated with concomitant ventricular infarction. A case of right atrial infarction accompanied by inferior myocardial infarction with rapid decompensated atrial fibrillation is reported. To increase awareness and knowledge of a complicated diagnosis, the present case is described in the context of a review of the relevant literature.

Key Words: Atrial fibrillation; Inferior myocardial infarction; PR segment; P_{Ta} segment; Right atrial infarction

A 53-year-old man with known diabetes mellitus and hypertension and no reported history of ischemic heart disease presented to the emergency department complaining of severe retrosternal chest pain. He described his symptoms as a stabbing but not radiating pain that persisted for 2 h, accompanied by sweating and nausea. Sublingual nitrates failed to alleviate the symptoms. The patient had no prior history of similar symptoms. His electrocardiogram (ECG) showed ST segment elevation in the inferior leads with new-onset atrial fibrillation (AF). He was admitted to the coronary care unit, where he received thrombolysis (streptokinase) and amiodarone. Because of the continuous chest pain associated with a rapid rate of AF, electrical cardioversion to sinus rhythm was carried out with 50 J monophasic synchronized cardioversion. On admission, the patient had an echocardiogram that showed normal area of the right atrium (17 cm²), with a transverse diameter of 4.1 cm. An akinetic inferior wall with low left ventricular ejection fraction (LVEF) (25%) was also documented at that time.

During grand rounds on the third day after admission, a cardiologist identified the ECG criteria (Figure 1) for right atrial infarction in a series of ECGs performed on the patient. The criteria included:

- ST segment elevation in the inferior leads;
- PR segment elevation in aVR and V1;
- PR segment depression in leads II, III and aVF; and
- P wave axis deviation (67°).

Un infarctus de l'oreillette droite, une arythmie auriculaire et un infarctus du myocarde inférieur forment une triade non repérée : Rapport de cas et analyse bibliographique

L'infarctus de l'oreillette est rarement diagnostiqué avant le décès en raison de ses caractéristiques électrocardiographiques discrètes et non spécifiques. Les modifications associées à un infarctus ventriculaire concomitant peuvent éclipser ces observations. On présente le cas d'un infarctus de l'oreillette droite accompagnée d'un infarctus du myocarde inférieur et d'une fibrillation auriculaire à décompensation rapide. Pour mieux faire connaître ce diagnostic complexe, le présent cas est décrit dans le contexte d'une analyse bibliographique pertinente.

All of these changes are consistent with right atrial infarction in association with an inferior myocardial infarction (MI).

Following four days of observation, the patient was discharged with follow-up appointments scheduled for the outpatient clinic. One month later, coronary angiography was performed, which documented critical lesions in the mid-right coronary artery (intact sinus node branch), the mid-left anterior descending artery and the proximal first obtuse marginal artery. Percutaneous coronary intervention was performed using stents in the right coronary artery, the left anterior descending artery and the first obtuse marginal artery, with good results. A second echocardiogram performed six months later documented a right atrial diameter of 22 cm² (normal right atrial area is 20 cm²), with a transverse diameter of 4.3 cm.

DISCUSSION

Atrial infarction is a rare event that occurs in ischemic heart disease and is usually diagnosed on autopsy. The first case of atrial infarction was described in 1925 (1), and this was 17 years before Langendorf (2) made the first ECG demonstration of atrial infarction. In 1938, Bean (3) reported a pathological series of 287 cases of MI that included two cases of atrial infarction (a 0.7% incidence). In 1942, Cushing et al (4) reported the largest series of autopsy-proven atrial infarctions, including 31 of 182 cases of fatal MIs (a 17% incidence). The first antemortem diagnosis of atrial infarction was made by Hellerstein (5) in 1948. The diagnosis was

¹Cardiology Department, Hamad Medical Corporation; ²Department of Medicine, Weill Cornell Medical College, Doha, Qatar

Correspondence: Dr Douraid K Shakir, PO Box 39046, Doha, Qatar. Telephone 00-974-523-9848, fax 00-974-439-2454,

e-mail dshakir@hotmail.com

Received for publication September 6, 2006. Accepted October 16, 2006

Figure 1) Electrocardiogram showing ST segment elevation in the inferior leads (II, III and aVF), PR segment elevation in aVR and V1 (double-headed arrows), PR segment depression in leads II, III and aVF (single-headed arrows), and P wave axis deviation (67°)

TABLE 1
Atrial infarction complications

Complication	Incidence
Rhythm disturbances	61%–74%
Supraventricular tachycardia	
Atrial fibrillation	
Wandering atrial pacemaker	
Sinus tachycardia with or without atrial premature contractions	
Sinus arrest	
Thromboembolic phenomena	84%
Pulmonary emboli	
Systemic emboli	
Atrial rupture	4.5%
Hemodynamic consequences	
Loss of atrial 'kick'	

based on PTa segment changes in the presence of complete heart block using a standard surface ECG. The highest reported incidence by autopsy was 42% by Wartman et al (6) in 1950. This wide variation in incidence of atrial infarction may be a reflection of whether the atria were specifically examined for necrosis. Atrial infarction may be seen in up to 10% of patients with acute MI if PR segment displacement is used as the criterion for atrial infarction (7).

An atrial infarction is more common on the right side of the heart than the left, and is more frequently found in the atrial appendages than the lateral or posterior walls of the atrium. The considerably higher oxygen content of left atrial blood may explain the difference in incidence between right and left atrial infarction. Gardin and Singer (8) reviewed several larger series and found the right atrium involved in 81% to 98% of infarctions, the left atrium involved in 2% to 19% of infarctions and biatrial involvement in 19% to 24% of infarctions.

PTa segment changes, when they occur, theoretically depend on the location of the infarction, analogous to the ST segment deviation in ventricular infarction. For example, injury of the subepicardial myocardium of the posterior basal wall (ie, left atrium) should show positive deviation in lead III, positive deviation in lead II and negative deviation in lead I. Anterior or anterolateral lesions (ie, right atrium), as in the case of our patient, should show depression in lead III and elevation in lead I.

Atrial infarction is frequently accompanied by a variety of complications (Table 1). These include arrhythmia, rupture, loss of atrial 'kick' and thromboembolic phenomena. Arrhythmias are quite common in the setting of atrial infarctions and include paroxysmal AF, wandering atrial pacemaker, premature atrial contractions and runs of atrial tachycardia (9). Reduced secretion of atrial natriuretic peptide (10), as well as low cardiac output syndrome, has been reported when a right ventricular infarction is coincident with an atrial infarction.

ECG diagnostic criteria for atrial infarction

Criteria proposed by Liu et al (11):

Major criteria:

- PTa segment elevation of more than 0.5 mm in leads V3 and V6, with reciprocal depression of PTa segments in leads V1 and V2;
- PTa segment elevation of more than 0.5 mm in lead I, with reciprocal depressions in leads II and III; and
- PTa 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 arrhythmia.

Minor criteria:

- abnormal P waves;
- flattening of P wave in M;
- flattening of P wave in W; and
- irregular or notched P wave.

Additional criteria suggested by Sivertssen et al (12):

- PR segment prolongation (normal PR segment duration is less than 200 ms);
- P wave axis changes (normal P wave axis on frontal plane is 30° to 60°); and
- abnormal atrial rhythms, including atrial flutter, AF, wandering atrial pacemaker and atrioventricular nodal rhythm.

Alteration of atrial repolarization (Ta segment), recognized by deviation from the TP segment, can be either secondary or primary. Secondary changes appear in response to, and are obligatory to, atrial depolarization. However, primary Ta segment changes are independent of atrial depolarization and indicate the nonuniformity of atrial repolarization. The usual cardiac pathological causes of secondary Ta segment depression (which may exceed 1 mm [0.1 mV]), include atrial dilation, hypertrophy and intra-atrial block, while noncardiac causes include chronic obstructive lung disease. In all of these conditions, Ta segment depression may be exaggerated and mistaken for ST segment displacement.

The usual causes of primary Ta segment changes are pericarditis, atrial infarction and atrial injury due to penetrating wounds. Pericarditis exaggerates the normally negative Ta segment, and Ta segment depression is recorded in all leads except aVR; an elevated Ta segment is typically found in this lead. Occasionally, a Ta segment abnormality may be the only convincing evidence of acute pericarditis.

Our patient's condition represents a typical case of right atrial infarction: a combination of inferior myocardial infarction complicated by rapid AF. This is a common complication of atrial infarction that necessitates cardioversion due to continuous chest pain. ECGs performed four days after admittance documented a right atrial infarction that was initially overlooked. After cardioversion, the echocardiogram showed a low LVEF (25%). Combined with the loss of atrial contribution during the AF, the actual LVEF might have been much lower. This is one of the complications associated with atrial infarction. After six months, the echocardiogram showed that right atrial size had increased to above normal, probably due to the remodelling process that occurs after infarction.

Our patient's ECG showed PR segment elevation in leads VR and V1. PR segment depression was observed in the inferior leads II, III and aVF, with the P wave axis shifted to the right. All of these findings are consistent with anterolateral atrial infarction (right atrium). While a biopsy would have confirmed the pathological diagnosis, the thinness of the atrial wall made this impossible.

The present case highlights a combination of conditions that complicate efficient diagnosis of an atrial infarction. A ventricular infarction complicated by AF may be due to an atrial infarction. Similarly, ignoring PR segment changes related to atrial infarction criteria and arrhythmia may result in delayed diagnosis of the atrial infarction. This triad of conditions may be indicated by the presence of atrial arrhythmia or by ventricular infarctions accompanied by complications listed in Table 1.

It is worth mentioning that there are a few other modalities for diagnosing atrial infarction, such as high-gain surface 'atriograms', intra-arterial recordings, Lewis lead ECGs and cardiac gated blood pool scans. A few reports describe transesophageal echocardiograms as a modality for diagnosing atrial infarction (9).

CONCLUSION

This case is presented to highlight the possibility of atrial infarction when considering inferior, and/or posterior infarctions with atrial arrhythmias, hypotension and a cardiac output status.

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