

Atrial Myocardial Infarction

A Case of Acute Coronary Syndrome Featuring a Forgotten and Disguised Intruder

Andreas Y. Andreou

Department of Cardiology, Limassol General Hospital, Limassol, Cyprus; University of Nicosia Medical School, Nicosia, Cyprus

Abstract

An infrequently diagnosed case of atrial infarction which occurred in conjunction with left ventricular myocardial infarction is presented. Atrial infarction was recognised by the presence of PR segment depression in conjunction with premature atrial complexes. Spatial vector analysis of premature atrial complexes, is highlighted as a means to localise atrial infarction.

Keywords: Electrocardiography; acute coronary syndrome; PR-segment deviation; atrial arrhythmias; atrial infarction

Case Presentation

A 52-year-old male patient, a cigarette smoker with a history of type II diabetes mellitus and dyslipidaemia but no history of cardiovascular disease, presented to hospital owing to sudden onset, constrictive retrosternal chest pain of thirty minutes duration. A 12 lead-electrocardiogram (fig. 1) recorded on admission showed a basic sinus rhythm, a heart rate of 94 beats per minute, coving and subtle elevation of the ST segment in leads II, aVF and III, subtle ST segment elevation in lead V6 and at least 0.1 mV ST segment depression in leads V2 to V5; the ST segment depression in lead V2 was horizontal and associated with a negative T wave, whereas in leads V3 through V5 it was up-sloping and associated with a positive T wave.

What other ECG abnormalities do you observe? What do they suggest? What would be your next course of action?

Discussion

This ECG is notable for two additional abnormalities. First, each sinus P wave is associated with an atrial depolarisation (P') wave (arrows in fig. 2) that is superimposed upon and deforms the ST segment. Each P' wave shows

a very short coupling interval (approximately 240 msec) thereby raising suspicion that it may be a manifestation of atrial reciprocal activation. Yet, this was precluded by the absence of lengthening of the preceding PR interval, which is a prerequisite of its development. Consequently, P' waves were interpreted as a

manifestation of premature atrial depolarisation, and because of a fixed coupling interval they were ascribed to reentry in the atrial myocardium. None of the premature P' waves is followed by a pause, suggesting, failure to penetrate the sinus node, that is, sinus nodal entrance block. The PR interval is stable at approximately 160 msec and does not prolong after a premature P' wave, suggesting that the latter fails to penetrate a significant part of the atrioventricular node, that is, atrioventricular nodal entrance block. Premature P' waves are inverted in leads II, aVF, and III, and upright in leads aVR and aVL, indicating a P' wave vector directed superiorly in the frontal plane, thus suggesting an ectopic focus in the posteroinferior atrial wall [1, 2]. Furthermore, premature P' waves are upright in lead V1 and

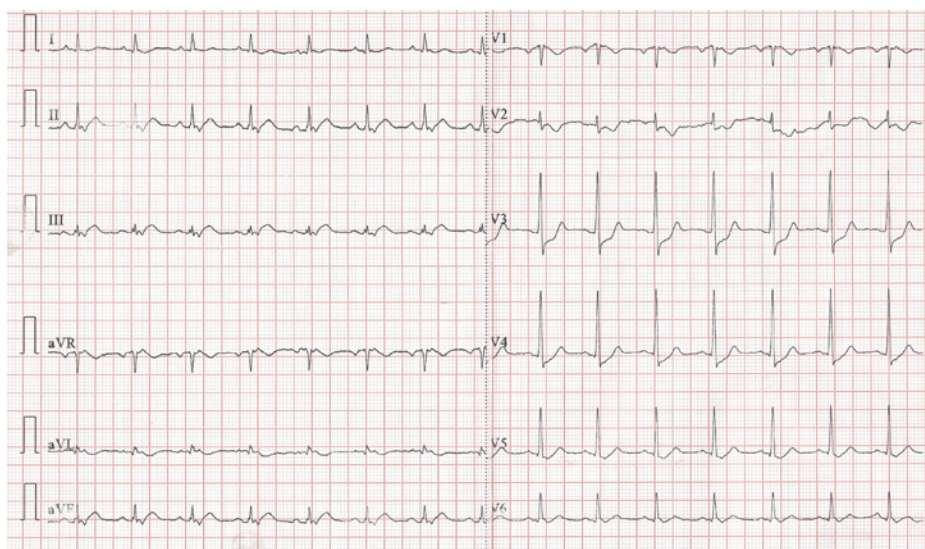


Figure 1: Twelve-lead ECG on admission.

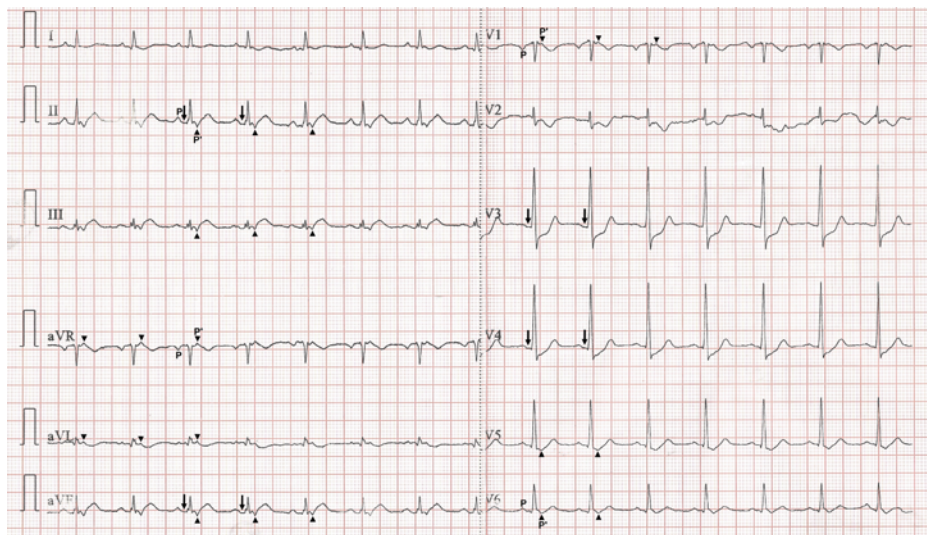


Figure 2: Twelve-lead ECG on admission. ECG depicts atrial bigeminy with non-conducted extremely premature atrial depolarisations (P' waves; arrowheads) whose spatial vector points superiorly, to the right and anteriorly suggesting an ectopic focus in the posteroinferior wall of the left atrium. Note also the PR segment depression in leads II, aVF, V3 and V4 (arrows).

inverted in leads V5 and V6, indicating a P' wave vector directed anteriorly and rightward in the horizontal plane, thus suggesting a left atrial ectopic rhythm. The second abnormality detected in this ECG is PR segment depression with respect to the TP segment in leads II, aVF, V3 and V4 (arrows in fig. 2), which, together with the non-conducted premature atrial complexes (P' waves) were ascribed to posteroinferior left atrial infarction [3]. Scrutiny of the presenting ECG (fig. 1) also shows that the ST segment deviation vector is better seen in the horizontal than in the frontal plane and more in leads V3 to V4 than in leads V1 to V2. It is, therefore, inferred that early in an ischaemic process affecting the inferior and inferior-lateral left ventricular walls, the basal (formerly

posterior) and mid inferior segments which likely bend upwards, were the segments affected the most [4]. Indeed, this was confirmed in a posterior-lead ECG, which disclosed at least 0.05 mV ST segment elevation in leads V7–V9. The patient underwent emergency coronary angiography, which showed a total occlusion in the proximal segment of a co-dominant left circumflex artery (arrow in fig. 3A) tackled successfully with stenting, which achieved a good clinical outcome. Discharge ECG (fig. 4) showed signs of an evolved inferior wall myocardial infarction with extension to the lateral wall.

Atrial infarction still remains underdiagnosed, primarily because its ECG manifestations are subtle and non-specific [3]. Accord-

ing to Liu et al. [5], whose ECG criteria for atrial infarction are the most widely accepted, depression of the PR segment of small magnitude and without accompanying PR segment elevation is not enough by itself to suggest atrial infarction. However, Burch [6] reported that PR segment depression as small as a fraction of a millimetre or more was always associated with atrial infarction detected at autopsy. In another series of histologically confirmed atrial infarction, none of the patients showed PR segment elevation and all showed PR segment depression [7]. Conclusively, the presence of PR segment deviation of any magnitude in a clinical context of acute myocardial ischaemia and its resolution upon amelioration of ischaemia or establishment of a healed infarct favours the diagnosis of atrial infarction [8]. Furthermore, any form of supraventricular arrhythmia occurring in the context of ventricular myocardial infarction should be considered highly suggestive of an accompanying atrial infarction [5, 9].

Here, the ECG pattern of atrial infarction comprised approximately 0.05 mV PR segment depression in leads II and aVF, and slightly less than 0.1 mV PR segment depression in leads V3 and V4 without accompanying PR segment elevation and non-conducted premature atrial depolarisations. Both, PR segment depression and premature atrial depolarisations disappeared after recanalisation of the occluded left circumflex artery and such a temporal association and evolution with acute and resolving myocardial ischaemia favoured their ischaemic origin, specifically atrial infarction. Furthermore, the PR segment deviation vector observed in our case would appear to partly fulfil criteria for right but also for left atrial infarction [3, 5]. Nevertheless, the

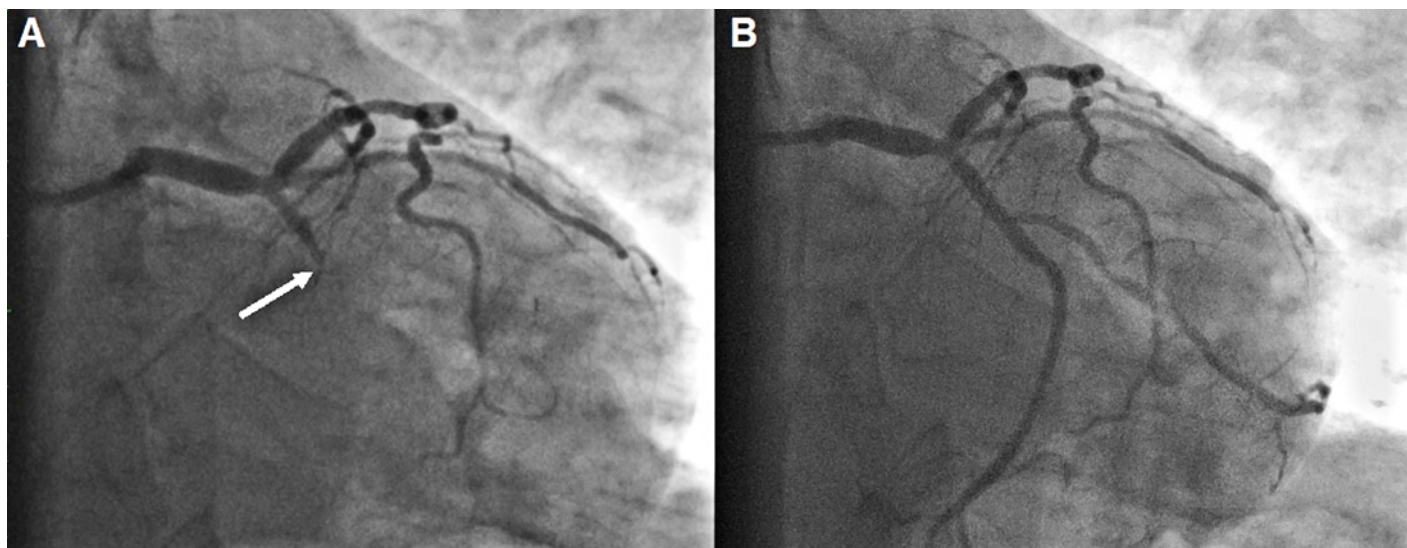


Figure 3: Conventional coronary angiography images. Note the total occlusion in the proximal segment of the left circumflex coronary artery (arrow in A) and the result after stent implantation (B).

Case Report

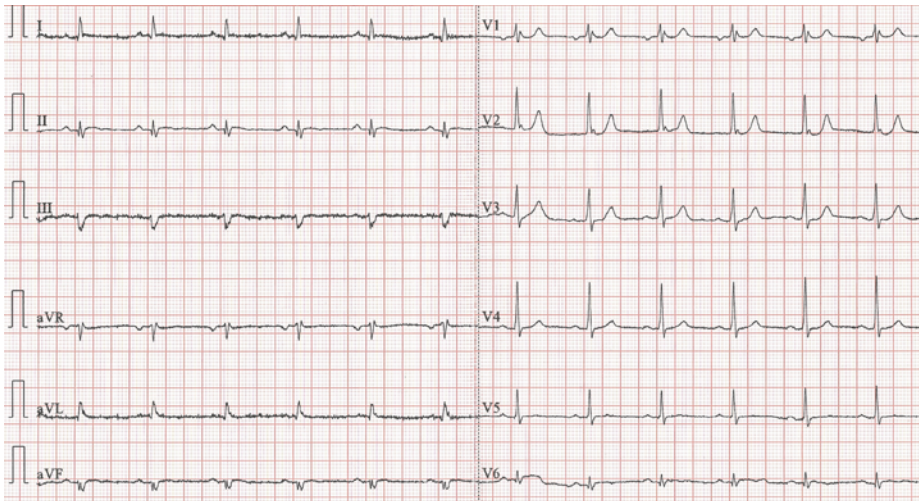


Figure 4: Twelve-lead ECG on discharge. ECG depicts pathological Q waves in lead II and fragmented QRS complexes in leads III and aVF (evolved inferior wall myocardial infarction). Furthermore, the reduced height of the R waves in lead V6 as compared with their height on admission and the tall and broad R waves in lead V2 indicated extension of the infarction to the lateral wall. Note also, that the absence of a positive P wave in lead V2 together with a biphasic P wave in lead V1 with a predominantly negative component and an rSr' pattern in the same lead suggested high placement of the V1 and V2 electrodes.

inverted P' waves in leads II, aVF, III and V6 and the upright P' waves in lead V1 indicated a spatial P' wave vector pointing superiorly, to the right and anteriorly, thus suggesting an ectopic focus in the posteroinferior aspect of the left atrium [1, 2]. If the ectopic focus had been located in the posteroinferior aspect of the right atrium, the vector of atrial premature depolarisation would have been directed superiorly but also posteriorly, thereby yielding inverted P' waves in lead V1 [1]. Knowledge of the site of atrial infarction is still clinically useful even in the absence of complications as in the present case. For example, given the reported association of left atrial infarction with left inferobasal (formerly posterior) myocardial infarction, the diagnosis of the former may raise suspicion of the latter [10]. Indeed, this association is corroborated in the present case. Furthermore, atrial ischaemia/infarction promotes reentry by causing significant local conduction delay, thereby facilitating induction and maintenance of atrial fibrillation [11]. Here, the exceptionally short coupling interval of the premature atrial depolarisations ascribed to reentry indicated that the refractory period of the atrial myocardium is very short. Accordingly, our patient was at high risk of developing atrial fibrillation precipitated by the atrial premature depolarisations; yet such an arrhythmia did not occur, apparently, because the atrial myocardium was completely repolarised at the crucial time.

Conclusion

The case of atrial infarction presented herein reminds us of the particular scrutiny required

to identify ECG signs favouring its diagnosis; it manifested as PR segment depression and premature P' waves. This case highlights spatial vector analysis of premature atrial complexes, as a means to localise atrial infarction. The diagnosis of atrial infarction has important short- and long-term clinical implications and should be sought in cases of left ventricular myocardial infarction.

Correspondence

Andreas Y. Andreou
Department of Cardiology
Limassol General Hospital
Nikeas street, Pano Polemidia
Postal code 3304
PO Box 56060, Limassol
Cyprus
[y.andreas\[at\]yahoo.com](mailto:y.andreas[at]yahoo.com)

Disclosure Statement

No financial support and no other potential conflict of interest relevant to this article was reported.

References

- 1 Harris BC, Shaver JA, Gray III S, Kroetz FW, Leonard JJ. Left atrial rhythm: Experimental production in man. *Circulation*. 1968;37(6):1000–1014.
- 2 Mirowski M. Left atrial rhythm: Diagnostic criteria and differentiation from nodal arrhythmias. *Am J Cardiol*. 1966 Feb;17(2):203–210.
- 3 Lu ML, De Venecia T, Patnaik S, Figueredo VM. Atrial myocardial infarction: A tale of the forgotten chamber. *Int J Cardiol*. 2016 Jan 1;202:904–909.
- 4 Fiol-Sala M, Birnbaum Y, Nikus K, Bayés de Luna A. Chapter 7: Acute Coronary Syndrome with ST Elevation (STE-ACS). In: *Electrocardiography in Ischemic Heart Disease: Clinical and Imaging Correlations and Prognostic Implications*. 2nd Edition. John Wiley & Sons Ltd; 2020. p. 111–179.
- 5 Liu CK, Greenspan G, Piccirillo RT. Atrial Infarction of the Heart. *Circulation*. 1961 Mar;23:331–338.
- 6 Burch GE. Of the p-r segment depression and atrial infarction. *Am Heart J*. 1976 Jan;91(1):129–130.

- 7 Sivertsen E, Hoel B, Bay G, Jörgensen L. Electrocardiographic atrial complex and acute atrial myocardial infarction. *Am J Cardiol*. 1973 Apr;31(4):450–456.
- 8 Lazar EJ, Goldberger J, Peled H, Sherman M, Frishman WH. Atrial infarction: diagnosis and management. *Am Heart J*. 1988 Oct;116(4):1058–1063.
- 9 Gardin JM, Singer DH. Atrial Infarction: Importance, Diagnosis, and Localization. *Arch Intern Med*. 1981;141(10):1345–1348.
- 10 Rose KL, Collins KA. Left atrial infarction: a case report and review of the literature. *Am J Forensic Med Pathol*. 2010 Mar;31(1):1–3.
- 11 Álvarez-García J, Vives-Borrás M, Gomis P, Ordoñez-Llanos J, Ferrero-Gregori A, Serra-Peñaranda A, Cinca J. Electrophysiological Effects of Selective Atrial Coronary Artery Occlusion in Humans. *Circulation*. 2016 Jun 7;133(23):2235–2242.