Suspected Atrial Wall Rupture Associated with Acute Myocardial Infarction: A Pathognomonic ECG Pattern?

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In autopsy studies a trial infarction is not an uncommon finding, occurring in 17% of patients who died from myocardial infarction [1]. Its clinical recognition, conversely, is far more rare since it is essentially based on the presence of abnormalities of the P-Ta segment, which may be either elevated or depressed [2]. In most cases, atrial infarction occurs in association with ventricular infarction; under these circumstances, the subtle electrocardiographic signs of atrial injury are often overlooked, being obscured by the more prominent and obvious changes induced by the concomitant ventricular infarction.

We report a curious ECG whose features strongly suggest massive atrial involvement in the course of a ventricular infarction.

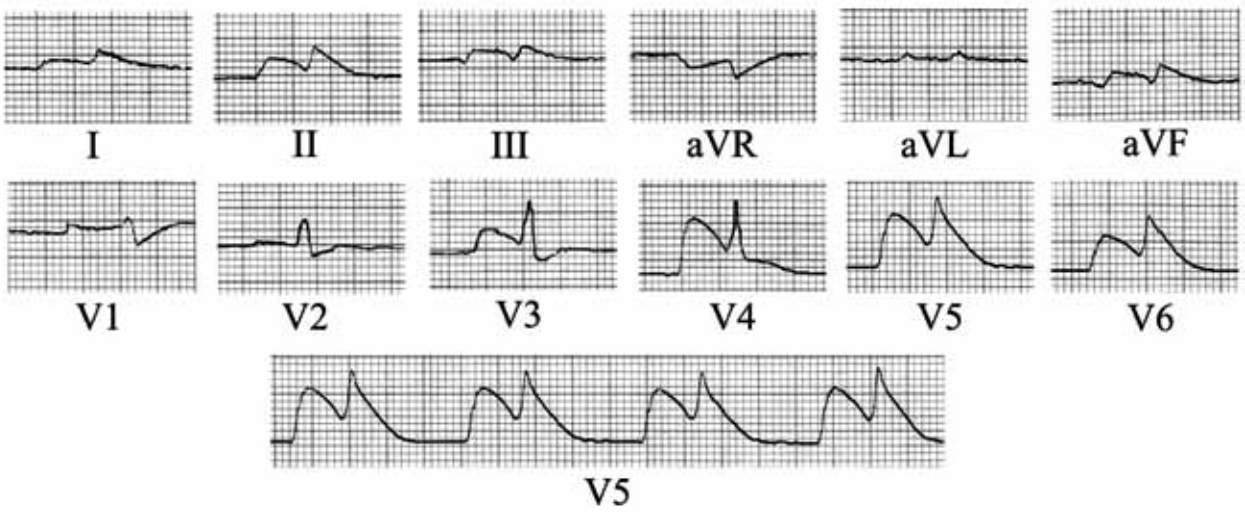
PATIENT DESCRIPTION

A 79 year old man presented at the emergency department because of dyspnea and chest pain. He had a history of coronary artery disease. On admission a 12-lead ECG was recorded and laboratory studies and Doppler echocardiogram were scheduled. Before these additional tests could be performed, however, the patient quickly deteriorated and eventually expired, despite intensive treatment including cardiopulmonary resuscitation. No permission for autopsy was obtained from his relatives.

The ECG taken on admission [Figure] shows ventricular complexes of low voltage in the limb leads, preceded by very large P waves (duration 0.26 sec, voltage about 0.8 mV in V4, V5 and V6). The P-R segment is markedly elevated in leads I, II, III, aVF and from V4 to V6. In the same leads an S-T segment elevation clearly appears as an oblique straight line slowly descending from the peak of R wave to the isoelectric line. No T waves are detectable in the tracing. The unusually wide and tall P waves, associated with the P-R interval elevation, suggest the diagnosis of atrial infarction likely leading to atrial wall rupture. A ventricular infarction must also be assumed since there is S-T segment elevation in several leads.

The clinical presentation (typical chest discomfort shortly followed by hemodynamic impairment and death) and the patient's history are consistent with the above hypothesis.

ECG showing extremely wide and tall P waves with elevation of both the PR and ST segments



COMMENT

Atrial infarction is more frequent on the right side and is complicated by rupture in 5% of cases. It is poorly evident on clinical evaluation but may be suspected from a careful analysis of the ECG.

Electrocardiographic diagnosis of atrial infarction, however, represents a difficult challenge because the criteria reported in the literature can be discovered only in the P wave and, especially, in P-R segment (expression of P-Ta segment) abnormalities. The atrial repolarization wave (Ta), as well as the P-Ta segment (extending from the onset of the P wave to the end of the Ta wave), are usually undetectable on the ECG. This is because the Ta wave is small and, also, is obscured by both the QRS complex and the early part of the S-T segment, the P-Ta coinciding with the P-R interval. In atrial infarction the lesion wave affecting atrial repolarization produces displacement of the P-Ta segment, resulting in elevation or depression of the P-R segment [2]. The P wave morphology may also be modified by atrial infarction and therefore represents a useful diagnostic marker. Unfortunately, the above P and P-R changes are usually modest, non-specific and transient.

The reported ECG, unlike others, shows striking features whose detection is extremely easy and unequivocal. The unusual configuration of these "P waves" likely expresses a profound and extensive atrial myocardial involvement that possibly resulted in atrial wall rupture, a catastrophic event that could have caused the rapid deterioration and death. The abnormalities observed in the figure are indeed very similar to those seen in a previously reported case of traumatic right atrial rupture [3]. Such an analogy could support, in our patient, the mechanism of atrial infarction complicated by atrial wall rupture.

A major limitation of this report is the lack of postmortem confirmation for the suspected diagnosis. The patient's death, however, occurred so soon after the emergence of symptoms (less than 1 hour) that there was not enough time for the structural changes typical of ischemia-induced necrosis to appear. Autopsy, therefore, would have been of poor diagnostic usefulness, since a period of at least 2–3 hours after the coronary artery occlusion is needed to allow the early detection of myocardial infarction [4]. On the other hand, the impressive ECG changes observed in this patient reliably support the diagnosis of infero-lateral ventricular infarction with coexisting atrial myocardial infarction.

The ECG signs of atrial infarction, particularly those of atrial wall rupture, have not received proper attention and have been largely neglected [5]. However, the cardiologist's sharp eyes, if they have seen such a pattern even once, can recognize it.

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Capsule

β-arrestin 2 regulates Aβ generation and γ-secretase activity in Alzheimer's disease

β-arrestins are associated with numerous aspects of G protein-coupled receptor (GPCR) signaling and regulation and accordingly influence diverse physiological and pathophysiological processes. Thathiah and co-authors report that β-arrestin 2 expression is elevated in two independent cohorts of individuals with Alzheimer's disease. Overexpression of β-arrestin 2 leads to an increase in amyloid-β (Aβ) peptide generation, whereas genetic silencing of Arrb2 (encoding β-arrestin 2) reduces generation of Aβ in cell cultures and in Arrb2–/– mice. Moreover, in a transgenic mouse model of Alzheimer's disease, genetic deletion of Arrb2 leads to a reduction in the production of Aβ40 and Aβ42. Two GPCRs implicated previously in Alzheimer's disease (GPR3 and the β2-adrenergic receptor) mediate their effects on Aβ generation through interaction with β-arrestin 2. β-arrestin 2 physically associates with the Aph-1a subunit of the γ-secretase complex and redistributes the complex toward detergent-resistant membranes, increasing the catalytic activity of the complex. Collectively, these studies identify β-arrestin 2 as a new therapeutic target for reducing amyloid pathology and GPCR dysfunction in Alzheimer's disease

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Eitan Israeli

"A pessimist sees the difficulty in every opportunity; an optimist sees the opportunity in every difficulty"

Winston Churchill (1874-1965), British politician, best known for his leadership of the United Kingdom during the Second World War