Pathognomonic ECG Pattern of Impending Atrial Rupture

Bernard Belhassen MD

Department of Cardiology, Tel Aviv Sourasky Medical Center, affiliated with Sackler Faculty of Medicine, Tel Aviv University, Ramat Aviv, Israel

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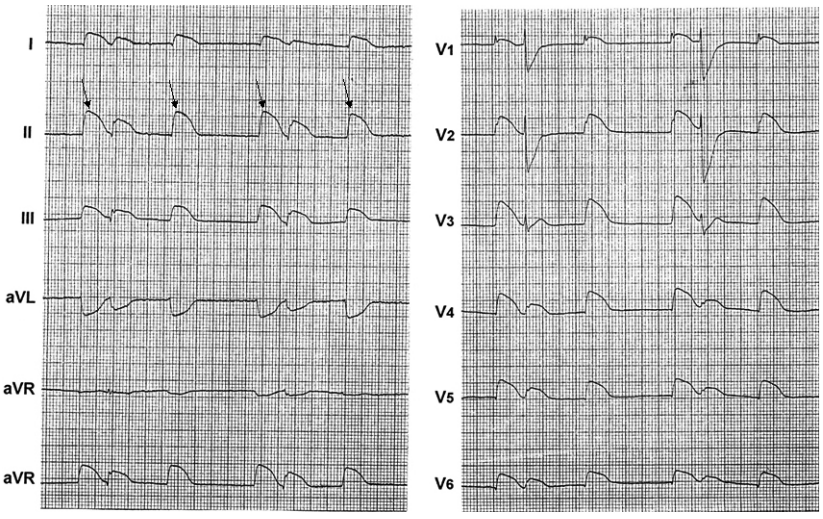
Myocardial rupture is responsible for almost 15% of all in-hospital deaths among patients with acute myocardial infarction. After pump failure, it is the second most common cause of in-hospital mortality among patients with acute MI. Myocardial rupture is frequently diagnosed at postmortem examination of patients with out-of-hospital sudden death. It may involve left ventricular and right ventricular free walls, ventricular septum, and left ventricular papillary muscle, in decreasing order of frequency. It rarely involves the atrial free wall (usually the right) in combination with a ventricular infarction, as was reported in an old pathological study where the incidence reached 6% [1]. Mortality of myocardial rupture is extremely high unless early diagnosis is made and urgent surgical intervention provided.

In patients with ischemic ventricular rupture, persistent ST elevation is frequently observed before the event. Free wall rupture is often associated with the sudden onset of bradycardia and electromechanical dissociation. In contrast, electrocardiographic signs suggesting atrial infarction are modest and less specific than those of the associated ventricular infarction, which explains why they are frequently overlooked. Classical signs of atrial infarction include: a) changes in width and configuration of the P wave, b) changes (depression or elevation) of the PR segment, and c) a previously unrecorded convexity of the first portion of the PR segment [2,3]. To the best of my knowledge, ECG signs of atrial rupture in the setting of an acute MI have not been previously reported.

In this *IMAJ* issue, Marafioti and coworkers from the University of Verona [4] report the case of a 79 year old man with a history of coronary artery disease and complaints of dyspnea and typical chest pain who died shortly after being admitted to hospital. The patient's ECG was extraordinarily abnormal, consisting of two components: the first presumably representing the atrial activity and the second the ventricular activity. The second activity is actually compatible with an evolving acute infero-postero-lateral MI. However, one is struck by the giant waves, especially in the antero-lateral precordial leads assumed to represent the atrial activity. The first impression from that tracing is that these giant P waves represent an artifact. However, a more careful analysis suggests that they represent a major injury pattern affecting the atrium in the same way ST elevation reflects a ventricular injury pattern. Such atrial disorders could suggest the imminence of atrial rupture, just as persistent major ST elevation could indicate the imminence of ventricular rupture. Unfortunately, the lack of postmortem examination did not enable a definite diagnostic confirmation. Nonetheless, I do agree with the authors that these impressive and uncommon prominent ECG changes at the atrial level, combined with the patient's history and the clinical presentation, strongly suggest that atrial wall rupture had occurred in the course of an infero-lateral MI and resulted in the patient's death.

The ECG tracing presented by Marafioti et al. [4] exhibits striking similari-

Twelve-lead ECG showing sinus rhythm (58/min) with 2:1 atrioventricular block. The P waves (arrows) are giant in almost all leads, mimicking Pardee waves. The QRS complex is also markedly abnormal. Note that the atrial beats in this tracing exhibit a morphology similar to those described by Marafioti et al. Tracing reprinted with permission from Elsevier [5]



MI = myocardial infarction

ties with that reported by van Veldhuisen and van den Berg [5] in a previously healthy 20 year old man brought to hospital after he was run over by a car. The patient died shortly after admission to hospital and a right atrial rupture with massive intrathoracic bleeding was found during autopsy. The patient's ECG [Figure] showed abnormalities that were even more pronounced than those described by Marafioti et al. [4]. A 2:1 atrioventricular block was present, which made it possible to individualize the giant P waves (arrows) that mimic the classical Pardee waves observed during the first hours of an acute MI. The similarity in the P wave morphology in both reports is remarkable, supporting the hypothesis that the atrial rupture in the Marafioti case also occurred in the right atrium.

These two unusual reports should encourage physicians encountering such cases to make a quick diagnosis of atrial rupture and initiate surgical intervention as expeditiously as possible. The editors of *IMAJ* should be complimented for publishing both such exceptional ECG tracings.

Address for correspondence:
Dr. B. Belhassen
Dept. of Cardiology, Tel Aviv Sourasky Medical Center, 6 Weizmann St., Tel Aviv 64239, Israel

Phone/Fax: (972-3) 697-4418
email: bblhass@tasmc.health.gov.il

References

1. Maurice P, Arnold E, Lenegre J. Les ruptures du coeur par infarctus du myocarde. *Revue Atherosclerose* 1959; 1: 193-200. Also: L'infarctus du myocarde. Himbert J, Lenegre J. Sandoz, eds. Paris: Laboratoires Sandoz, 1967: 139.
2. Cristal N, Peterburg I, Inbar-Yanai I. Atrial infarction leading to rupture. *Br Heart J* 1979; 41: 350-3.
3. Alonso-Orcajo N, Izquierdo-García F, Simarro E. Atrial rupture and sudden death following atrial infarction. *Int J Cardiol* 1994; 46: 82-4.
4. Marafioti V, Carbone V, Manara F, Oreto G. Suspected atrial wall rupture associated with myocardial infarction. A pathognomonic pattern? *IMAJ Isr Med Assoc J* 2013; 15: 121-2.
5. Van DJ, van den Berg. The electrocardiogram in traumatic right atrial rupture. *Int J Cardiol* 1999; 71: 179-80.

Capsule

Broad and potent neutralization of HIV-1 by a gp41-specific human antibody

Characterization of human monoclonal antibodies is providing considerable insight into mechanisms of broad human immunodeficiency virus-1 (HIV-1) neutralization. Huang and co-authors report an HIV-1 gp41 membrane-proximal external region (MPER)-specific antibody, named 10E8, which neutralizes ~98% of tested viruses. An analysis of sera from 78 healthy HIV-1-infected donors demonstrated that 27% contained MPER-specific antibodies and 8% contained 10E8-like specificities. In contrast to other neutralizing MPER antibodies, 10E8 did not bind phospholipids, was not autoreactive, and bound cell-surface envelope. The structure of 10E8 in complex with the complete MPER revealed a site of vulnerability comprising a narrow stretch of highly conserved gp41-hydrophobic residues and a critical arginine or lysine just before the transmembrane region. Analysis of resistant HIV-1 variants confirmed the importance of these residues for neutralization. The highly conserved MPER is a target of potent, non-self-reactive neutralizing antibodies, suggesting that HIV-1 vaccines should aim to induce antibodies to this region of HIV-1 envelope glycoprotein.

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Eitan Israeli

Capsule

Apoptotic cell clearance by bronchial epithelial cells critically influences airway inflammation

Lung epithelial cells can influence immune responses to airway allergens. Airway epithelial cells also undergo apoptosis after encountering environmental allergens; yet, relatively little is known about how these are cleared, or their effect on airway inflammation. Juncadella and co-workers show that airway epithelial cells efficiently engulf apoptotic epithelial cells and secrete anti-inflammatory cytokines, dependent upon intracellular signaling by the small GTPase Rac1. Inducible deletion of Rac1 expression specifically in airway epithelial cells in a mouse model resulted in defective engulfment by epithelial cells and aberrant anti-inflammatory cytokine production. Intranasal priming and challenge of these mice with house dust mite extract or ovalbumin as allergens led to exacerbated inflammation, augmented Th2 cytokines and airway hyper-responsiveness, with decreased interleukin (IL)-10 in bronchial lavages. Rac1-deficient epithelial cells produced much higher IL-33 upon allergen or apoptotic cell encounter, with increased numbers of nuocyte-like cells. Administration of exogenous IL-10 'rescued' the airway inflammation phenotype in Rac1-deficient mice, with decreased IL-33. Collectively, these genetic and functional studies suggest a new role for Rac1-dependent engulfment by airway epithelial cells and in establishing the anti-inflammatory environment, and that defects in cell clearance in the airways could contribute to inflammatory responses towards common allergens.

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Eitan Israeli