Recurrent Myocardial Infarction or Epistenocardiac Pericarditis: How Can the Surface ECG Be Useful in Clinical Decision Making?

LOVELY CHHABRA1, VINOD K. CHAUBEY2,3, DAVID H. SPODICK2,3

1Dept. of Cardiovascular Medicine, 2Dept. of Medicine, Hartford Hospital, University of Connecticut School of Medicine, Hartford, CT, 3Saint Vincent Hospital, University of Massachusetts Medical School, Worcester, MA, USA

We read with great pleasure the work by Doulaptsis et al. published in a recent issue of the Hellenic Journal of Cardiology.1 We commend the authors for their excellent clinical judgment and appropriate use of cardiac magnetic resonance imaging for confirming the diagnosis of epistenocardiac pericarditis. We would like to draw the attention of your readership towards yet another interesting electrocardiographic finding that can help differentiate between acute pericarditis and acute myocardial infarction (MI). PR-depression, in combination with a slightly downward sloping TP segment, is usually suggestive of acute pericarditis in a majority of patients; this finding is also known as “Spodick’s sign”, after the senior author of this letter. A downsloping TP segment is seen in 80% of cases of acute pericarditis.2-5 This downsloping of the isoelectric TP segment is ostensibly due to concurrent epicardial inflammation and PR-segment depression from atrial injury current.3,6 However, it is prudent to remember that epistenocardiac pericarditis is a localized form of pericarditis and is the result of an extension of inflammation following transmural myocardial infarction. Thus, Spodick’s sign is pragmatically not validated in the epistenocardiac form of pericarditis.

Recent investigations have suggested that QRS prolongation and QT shortening are common in acute MI as opposed to acute pericarditis and can serve as yet another helpful electrocardiographic tool (in addition to classical ECG signs) in differentiating acute MI from acute pericarditis.7-8 In the ECG of acute MI shown in Figure 1A of the report by Doulaptsis et al, the QRS duration appears to be longer than the QRS duration in acute pericarditis (Figure 1B) in the inferolateral leads (II, III, aVF, V5 and V6). This corresponds to the wall of inflammation seen on the cardiac MRI (Figure 2). However, QT shortening is not discernible. We can speculate that these ECG parameters may be useful in distinguishing between acute recurrent MI and epistenocardiac pericarditis, as this is independent of atrial injury current and looks at the particular window of myocardial wall inflammation. Further controlled investigations may be essential to validate this assumption.

Cardiac MR for visualizing a localized form of pericardial inflammation and edema is often underutilized, despite being well validated, probably because of cost concerns, reimbursement issues and even lack of immediate availability at many ter-
We commend the authors for highlighting that cardiac MR is the diagnostic modality of choice if there remains a clinical dilemma about the diagnosis.

References


The authors reply

We appreciate the comments of Chhabra et al on our article. We agree that both the previous as well as the most recently described diagnostic ECG findings for infarct-related pericarditis should be always evaluated in the setting of the patient with acute myocardial infarction and recurrent chest pain. We also agree with the authors that the above criteria will need further validation with imaging modalities such as CMR to be widely adopted in clinical practice. Finally, we would like to stress that other parameters such as C-reactive protein could play a significant role in the diagnosis of patients with post-infarct pericarditis. In a recent study we found that 78% of patients with acute myocardial infarction and high CRP (>52 mg/dL) showed some degree of pericardial inflammation on CMR.

Costas Doulaptsis, MD
24 Liberty Drive, Northampton, UK
doulacost@hotmail.com

References