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Journal of Electrocardiology 38 (2005) 195–203

JOURNAL OF Electrocardiology

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Electrocardiographic prediction of the severity of posterior wall perfusion defects on rest technetium-99m Sestamibi myocardial perfusion imaging

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Received 12 July 2004; revised 14 February 2005; accepted 1 March 2005

Abstract To identify electrocardiogram (ECG) variables predicting the severity of previous posterior wall myocardial infarction as measured by technetium-99m-Sestamibi rest single-photon emission computed tomography, we assessed agreement between ECG criteria and posterior wall perfusion defects (PWPDs) in 236 patients. Established ECG criteria for posterior and posterolateral infarctions were present in 22% and 19% of patients, respectively, and did not predict severity of PWPD (P = NS). Univariate predictors of severity were the Selvester QRS score (SQS) (P = .001) and an upright T wave in V_1 (UTV_1) greater than 0.2 mV (P = .001). Regression analysis demonstrated that SQS (P = .0001) and UTV_1 greater than 0.2 mV (P = .006) were highly predictive of severity (c statistic = 0.793). All severe PWPDs had an SQS of 2 or higher. Established ECG patterns for diagnosis of posterior infarction are insensitive and poor predictors of severity. The SQS and UTV_1 are effective for the diagnosis of posterior infarction and useful for the estimation of infarct severity.

Keywords: Posterior infarction; Electrocardiography; Selvester QRS score; Myocardial perfusion imaging; Posterior wall perfusion defect

1. Introduction

In a review of 9 large cohort studies, unrecognized myocardial infarctions have been found to represent up to 44% of all infarctions, suggesting that many myocardial infarctions are clinically unrecognized [1]. Because mortality rates after unrecognized and recognized infarctions are similar [2,3], the need for better diagnostic strategies is evident.

The electrocardiographic diagnosis of a previous myocardial infarction is based primarily on the identification of Q waves; thus, infarctions without identifiable Q waves will often not be detected. The diagnosis of posterior wall myocardial infarction by electrocardiogram (ECG) is particularly difficult because of the distance between the posterior wall and the anterior chest, the effect of summation forces, the electric position of the heart, and the absence of dorsal leads in the standard ECG. Existing ECG criteria for the diagnosis of posterior wall myocardial infarction are specific but insensitive [4-6]. Furthermore, it has not been determined whether electrocardiographic factors provide an indication of the severity and extent of posterior wall infarction.

Various electrocardiographic scoring methods have been used to estimate the size of a myocardial infarction. In a direct comparison using an identical reference population of anatomically measured infarcts, the Selvester QRS score (SQS) demonstrated the best correlation between anatomical and estimated infarct size in the posterolateral and inferior locations compared with the Minnesota Q-QS code, Novacode Q-wave scoring system, and the Cardiac Infarction Injury Score [7]. The SQS correlated very well with single infarct size in these territories (R = 0.70 for inferior and 0.74 for posterolateral). However, when multiple areas of infarction were included, the correlation was less robust (R = 0.36). Nevertheless, the scoring system developed and

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0022-0736/$ – see front matter © 2005 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2005.03.007

validated by Selvester et al [8], simplified and modified by Wagner et al [9], and further modified by Hindman et al [10] has proven to be a powerful tool in the evaluation of infarct size. This scoring system has been validated for posterolateral and inferior infarctions after correlation with quantitative anatomical findings [11,12].

Single-photon emission computed tomography (SPECT) myocardial perfusion at rest is routinely used in the diagnosis of myocardial infarctions. Gibbons et al [13] published a review of the validity of technetium-99m Sestamibi SPECT for infarct sizing, concluding that this technique is the best available measurement tool. In a multicenter trial assessing the feasibility of defect sizing on cardiac phantoms with ^{99m}Tc Sestamibi, the average correlation coefficient between the true and the measured defect size was 0.99 ± 0.01 [14,15]. In a larger animal study, Sinusas et al [16] found in 17 dogs that the defect area defined by ^{99m}Tc Sestamibi correlated with the postmortem infarct area ($\rho = 0.98$). In a scintigraphic/pathological study, Medrano et al [17] have also reported a close correlation ($r = 0.89$) between amount of fibrosis and ex vivo ^{99m}Tc Sestamibi defect in 15 human hearts with ischemic cardiomyopathy, explanted at the time of cardiac transplantation. It is possible that ^{99m}Tc Sestamibi may overestimate infarct size in patients who have suffered a recent infarction [18]; this overestimation is more likely to occur in the presence of hibernating myocardium [19-21]. A potential source of underestimation is the presence of significant subendocardial infarction [22]. The addition of electrocardiographic gated Sestamibi acquisitions with quantitative assessment of perfusion has demonstrated improved accuracy [23-25]. Myocardial perfusion with ^{99m}Tc Sestamibi SPECT has also been used for the validation of advanced ECG diagnostic software for the detection of prior myocardial infarction [26].

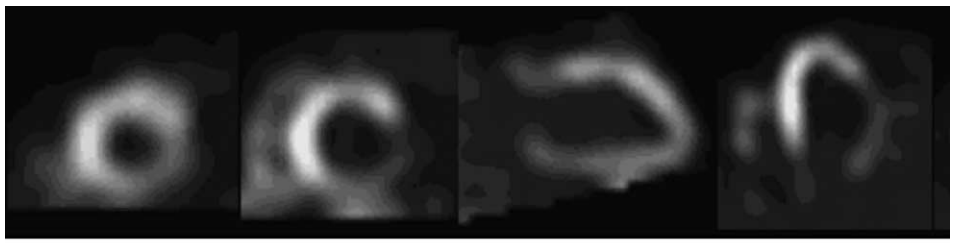
Few studies have compared electrocardiographic criteria for posterior myocardial infarct severity with those determined using the commonly used standard of SPECT myocardial perfusion imaging (MPI). The first aim of our study was to correlate accepted ECG criteria for posterior infarction, posterolateral infarction, and other ECG variables to the severity of posterior wall perfusion defects (PWPDs) as identified on ^{99m}Tc Sestamibi rest SPECT MPI.

Our second aim was to assess the predictive value of the SQS in determining the severity of PWPD as defined using MPI as the noninvasive surrogate of myocardial infarction.

2. Materials and methods

2.1. Patient selection

In this retrospective study, previous posterior infarctions were identified based on the myocardial perfusion studies alone. We reviewed 2066 consecutive ^{99m}Tc Sestamibi (Cardiolite) rest-stress SPECT MPI studies acquired between January 1998 and July 1999. Patients with a PWPD on the rest component of their ^{99m}Tc Sestamibi SPECT MPI were eligible for inclusion. Patients included in this study had either known or suspected coronary artery disease. Electrocardiographic exclusion criteria were ventricular pacing,



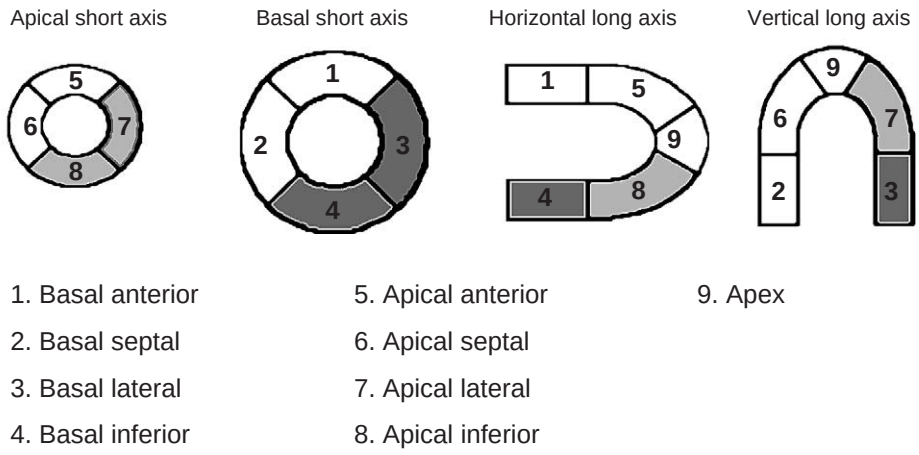


Fig. 1. Segmental perfusion regions making up the posterior myocardial wall on ^{99m}Tc SPECT Sestamibi MPI (segments 3 and 4, dark gray), and the possible extension of a posterior infarction (segments 7 and 8, light gray).