Inferior myocardial infarction scars could be more arrhythmogenic than anterior ones

Pascale et al. reported that the scars of old inferior infarctions without areas of potential ischaemia and accompanied by, on average, even better left ventricular systolic function were significantly more associated with the occurrence of ventricular arrhythmias than anterior scars. The authors proposed that the right ventricle damage and sympathetic anterior scars. The authors proposed that the right ventricle damage and sympathetic hyperactivity due to the destruction of afferent vagal fibres in the inferior infarctions may be underlying mechanisms. However, there is an observation suggesting a temporary 6-week impairment of cardiac parasympathetic activity associated with anterior infarctions and preserved parasympathetic function in inferior infarctions which conflicts with the latter mechanism Pascale et al. have proposed. Several additional mechanisms should also be considered.

It has been generally accepted that vagal fibres are mainly located in the infarctoposterior cardiac wall, whereas the distribution of sympathetic fibres is less well defined. Although there is a report suggesting that sympathetic nerves are distributed equally to the infarctoposterior and anterior left ventricular wall, a body of evidence suggests that sympathetic endings follow coronary arteries and are preferentially distributed across the anterior wall. At least part of these fibres are destroyed by anterior infarctions. This may reduce sympathetic efferent activity and abolish its arrhythmogenic consequences, particularly when cardiac vagal fibres are preserved. In inferior infarctions, preserved sympathetic endings in the anterior wall may be the loci of significantly increased sympathetic activity associated with anterior infarctions because of a shorter tachycardia total activation time, frequent intramural re-entrant or monorregional activation patterns, and involvement of papillary muscles. They suggested that the proximity and relationship among anatomic structures such as the mitral annulus, papillary muscles, posterior septum, and posteroseptal space may be responsible for this complexity.

Both the authors and Hikurri noted in the accompanying editorial that the inclusion of only patients who survived a life-threatening arrhythmia and those with stable sustained ventricular tachycardia did not allow a significant advance in risk assessment in the present study. Further study of the pathophysiological differences and electrophysiological consequences of anterior and inferior infarctions, stratification of post-infarction patients and reliable tools for identification of arrhythmogenic loci are required for a better definition of high-risk subgroups for sudden cardiac death.

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References


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