Fat infiltration in the heart

Liron Pantanowitz

Current teaching is that fatty infiltration into the myocardium (lipomatosis or cor adiposum) rarely affects cardiac function. This may not be entirely true. Rupture during acute myocardial infarction has certainly been shown to be more common in the fatty heart.1 Fat infiltrating in the region of the conduction system is responsible for causing sudden death.2 Furthermore, it is possible for lipomatous hypertrophy in the heart to even undergo malignant transformation.3 Since fat is a normal constituent of the heart, it remains undefined as to exactly how much, and in which locations, one considers fatty infiltration to be pathological. Whether fat extends into the myocardium from subepicardial stores, normally increased with aging and obesity, or arises de novo from pluripotential interstitial cells or cardiac myocytes, is also undetermined. The distinction is important if fat present in the myocardium were to signify prior episodes of hypoxia, during which intracellular lipids no longer able to be metabolised by mitochondria get extruded and phagocyted.4 5 Transdifferentiation of cardiac muscle into mature adipocytes has indeed been described in association with cardiomyopathy.5 This transdifferentiation has been proposed as a possible pathogenetic mechanism for arrhythmogenic right ventricular cardiomyopathy.6

The mechanism by which fatty infiltration promotes arrhythmogenicity and/or causes sudden death has never been well addressed. Infiltrating fat may provoke automaticity, and produce atrophy and degeneration of adjacent myocardial cells,7 or separate the myocardial cells, thereby reducing the number of sites or interstitial cells or cardiac myocytes, is also undetermined. The distinction is important if fat present in the myocardium were to signify prior episodes of hypoxia, during which intracellular lipids no longer able to be metabolised by mitochondria get extruded and phagocyted.4 5 Transdifferentiation of cardiac muscle into mature adipocytes has indeed been described in association with cardiomyopathy.5 This transdifferentiation has been proposed as a possible pathogenetic mechanism for arrhythmogenic right ventricular cardiomyopathy.6

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1 Roberts WC, Roberts JD. The floating heart or the heart too fat to sink: analysis of 55 necropsy patients. Am J Cardiol 1983;52:1286–9.