Assessment of Size and Shape of Myocardial Infarction by Late Enhancement MRI in the Time Course of Infarct Healing

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Objective:

The purpose of this clinical study was to analyze the remodeling process of the infarct territory in the time course of infarct healing.

Background:

Cardiac remodeling following myocardial infarction (MI) includes alterations in left ventricular (LV) chamber size, chamber shape and function.[1] Expansion of the infarct zone[2] and an increase in endocardial perimeter length of noninfarcted myocardium[3,4] are hallmarks of LV remodeling in the first days post-MI. Later on progressive or limited dilatation of the LV may follow.[5] We hypothesized that Late Enhancement (LE) can be used to measure changes in MI size and MI shape in the time course of infarct healing.

Methods:

70 LE studies were performed in 28 patients following first reperfused MI. All patients were studied at least twice, some were studied three times. Measurements were performed on days 5±2 and 13±3 post-MI (mean ± standard deviation, groups A and B, resp.) and after three months (group C). LE MR imaging was carried out using a segmented inversion-recovery turbo FLASH sequence[6] on a 1.5 T clinical MR scanner. On each study day the entire LV was imaged from base to apex using consecutive double oblique imaging planes (no slice spacing, slice thickness 8mm). Images were analyzed using the ImageJ software package (NIH, Bethesda). Image analysis was done in blinded fashion without knowledge of patient name or infarct age. We measured infarct size (IS, im mm^3), infarct extent (IE, in mm^2) and the mean infarct thickness (MIT, in mm): To obtain IE we added the circumferential extents of infarct in each slice and multiplied by 8 mm slice thickness. The physiologic meaning of an increase in IE would be infarct expansion in either circumferential and/or longitudinal direction. To obtain MIT we divided IS by IE. We compared IS, IE and MIT of groups B and C with the corresponding data of the preceding measurements by calculating the percentual size. Data are presented as mean ± standard error.

Results: Infarct shrinkage was found in the first 3 months of infarct healing. Infarct shrinkage was due to infarct thinning and - to lesser extent - to shrinkage of IE. The table shows the results for comparisons of IS, IE and MIT between groups. Note that not all 28 patiens were studied at all three time points, therefore the number of comparisons between groups (shown in the fourth column, n) is less than 28:

	Infarct Size	Infarct Extent	Mean Infarct Thickness	n
Groups B vs. A	85,5 ± 9,6 %	92,2 ± 5,7 %	91,2 ± 7,0 %	13
Groups C vs. B	95,9 ± 8,5 %	102,5 ± 5,3 %	92,5 ± 5,4 %	18
Groups C vs. A	68,0 ± 4,5 %	87,6 ± 4,3 %	77,8 ± 3,5 %	23

Further analysis of IE from day 5 until 3 months post-MI revealed that 18 of 23 infarcts shrank, 5 infarcts expanded. Infarct thinning was found in almost all patients (21 of 23) between day 5 and 3 months post-MI. Infarct thinning did occur in 4 of 5 patients *with* infarct expansion, however, infarcts thinned in 17 of 18 patients *without* infarct expansion as well.

Discussion:

The incidence of infarct expansion in our study is low. It should be noted that patients were enrolled in the study as soon as the clinical status did allow to perform LE MR imaging. This was never before day 3 following MI. Thus, the early infarct expansion process, as described by Hutcins and Bulkley,[2] was missed in this clinical study.

Infarct expansion, as seen in some of our patients, may explain previous findings of LV dilatation post-MI.[5] On the other hand, LV enlargement may result from increase in contractile (viable) segment length as well.[7] Future analyses will have to include remote myocardium and will have to correlate the combined findings in viable and nonviable tissue with LV volume measurements.

Infarct thinning did also occur in patients who did not reveal infarct expansion. Assuming constant infarct mass, infarct thinning would only be possible if infarcts expand in either circumferential and/or longitudinal direction. The finding that infarcts may thin independent of infarct expansion (no increase in IE) suggests, that thinning is - at least in part - due to the underlying infarct healing process. Experimental studies which relate infarct remodeling with the underlying physiology of infarct healing[8] and evaluate the mechanisms of infarct thinning may further elucidate this point.

Conclusion:

LE allows to analyze the infarct remodeling process in the time course of infarct healing. Infarcts shrink during infarct healing. This shrinkage is due to infarct thinning and - to lesser extent - due to shrinkage of IE. Infarct thinning may occur without infarct expansion.

References:

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