Quantification of post-reperfusion intramyocardial haemorrhage with cardiac magnetic resonance imaging in an ischemia/reperfusion pig model: T2* vs R2* vs R2’

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Topic(s):
T1 and T2 Mapping, T2*

Citation:
European Heart Journal - Cardiovascular Imaging (2019) 20 (Supplement 2), ii3

Funding Acknowledgements:
Competitive grant from the Carlos III Institute of Health-Fondo de Investigacion Sanitaria- and the European Regional Development Fund (ERDF/FEDER)

Background: R2*, the reciprocal parameter of T2* (1/T2*) has a linear correlation with iron concentration and is well established for the quantification of liver iron deposition. However, its accuracy for post-myocardial infarction (MI) intramyocardial haemorrhage (IMH) quantification is unknown. Furthermore, post-MI IMH co-occurs with oedema, which significantly increases the T2 signal by prolonging T2 mapping values. Given that T2* values are T2-dependent, the intensity of post-MI oedema is predicted to impair the ability of T2* to detect IMH. T2 prime (T2’) is obtained by eliminating T2 effects from T2* values, hence it should be less affected than T2* by the presence of post-MI oedema.

Purpose: This study sought to determine whether R2* (1/T2*) is more accurate than T2* for the detection of IMH and to evaluate whether R2’ (1/T2’) is less affected by the presence of oedema than R2*, and thus more suitable for the accurate identification and quantification of post-MI IMH.

Methods: Closed-chest 40-min left anterior descending coronary artery occlusion followed by reperfusion was performed in 20 male pigs, which were sacrificed at 120 min (n=5), 24 h (n=5), 4 days (n=5), and 7 days (n=5). At each time point, cardiac magnetic resonance (CMR) T2- and T2*-mapping scans were recorded, and myocardial tissue samples were collected and processed for histological quantification of IMH and myocardial water content. T2’ was obtained by eliminating T2 from T2* (1/T2’=1/T2*−1/T2) and R2’ from the inverse of T2’.

Results: Relative to remote tissue, histological IMH increased 5.2-fold, 10.7-fold, and 4.1-fold at days 1, 4 and 7, respectively. IMH was absent at 3 h. To avoid artefacts in linear correlations, pigs without IMH (n=8) were excluded from the evaluation of the association between CMR values and histological IMH. The presence of IMH was correlated more strongly with R2* (r=0.71; p=0.009) than with T2* (r=-0.52; p=0.086). The correlation with IMH was even stronger for R2’ (r=0.76; p=0.004). For myocardial oedema, the correlation was stronger for R2* (r=−0.66; p=0.019) than for R2’ (r=-0.52; p=0.080). Multivariate linear regressions confirmed that R2* values were significantly explained by both IMH and oedema (p=0.021 and p=0.041), whereas R2’ values were mostly explained by histological IMH (p=0.013) and were little influenced by myocardial oedema(p=0.203).

Conclusion: Using CMR mapping analyses and histological validation in a pig model of post-reperfused MI, R2’ more accurately detected IMH and was less influenced by myocardial oedema than R2*, making R2’ better...
Abstract: Quantification of post-reperfusion intramyocardial haemorrhage with cardiac magnetic resonance imaging in an ischemia/reperfusion pig model: $T2^*$ vs $R2^*$ vs $R2'$


Background: $R2^*$, the reciprocal parameter of $T2^*$ ($1/T2^*$) has a linear correlation with iron concentration and is well established for the quantification of liver iron deposition. However, its accuracy for post-myocardial infarction (MI) intramyocardial haemorrhage (IMH) quantification is unknown. Furthermore, post-MI IMH co-occurs with oedema, which significantly increases the $T2$ signal by prolonging $T2$ mapping values. Given that $T2^*$ values are $T2$-dependent, the intensity of post-MI oedema is predicted to impair the ability of $T2^*$ to detect IMH. $T2'$ is obtained by eliminating $T2$ effects from $T2^*$ ($1/T2' = 1/T2^* - 1/T2$) and $R2'$ from the inverse of $T2'$.

Purpose: This study sought to determine whether $R2^*$ ($1/T2^*$) is more accurate than $T2^*$ for the detection of IMH and to evaluate whether $R2'$ is less affected by the presence of oedema than $R2^*$, and thus more suitable for the accurate identification and quantification of post-MI IMH.

Methods: Closed-chest 40-min left anterior descending coronary artery occlusion followed by reperfusion was performed in 20 male pigs, which were sacrificed at 120 min (n=5), 24 h (n=5), 4 days (n=5), and 7 days (n=5). At each time point, cardiac magnetic resonance (CMR) $T2$- and $T2^*$-mapping scans were recorded, and myocardial tissue samples were collected and processed for histological quantification of IMH and myocardial water content. $T2'$ was obtained by eliminating $T2$ from $T2^*$ ($1/T2' = 1/T2^* - 1/T2$) and $R2'$ from the inverse of $T2'$.

Results: Relative to remote tissue, histological IMH increased 5.2-fold, 10.7-fold, and 4.1-fold at days 1, 4 and 7, respectively. IMH was absent at 3 h. To avoid artefacts in linear correlations, pigs without IMH (n=8) were excluded from the evaluation of the association between CMR values and histological IMH. The presence of IMH was correlated more strongly with $R2^*$ ($r=0.71; p=0.009$) than with $T2^*$ ($r=-0.52; p=0.086$). The correlation with IMH was even stronger for $R2'$ ($r=0.76; p=0.004$). For myocardial oedema, the correlation was stronger for $R2^*$ ($r=-0.66; p=0.019$) than for $R2'$ ($r=-0.52; p=0.080$). Multivariate linear regressions confirmed that $R2^*$ values were significantly explained by both IMH and oedema ($p=0.021$ and $p=0.041$), whereas $R2'$ values were mostly explained by histological IMH ($p=0.013$) and were little influenced by myocardial oedema ($p=0.203$).

Conclusion: Using CMR mapping analyses and histological validation in a pig model of post-reperfused MI, $R2'$ more accurately detected IMH and was less influenced by myocardial oedema than $R2^*$, making $R2'$ better suited for the characterization of post-MI IMH.