Title: Fast functional CT technique in assessing myocardial edema in acute myocardial infarction: A validation study

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Structured Abstract:
Introduction: Every year more than 60,000 Canadians suffer from heart attack (acute myocardial infarction, AMI), which is caused by partial or complete occlusion of one or more coronary arteries. Lack of blood flow and oxygen delivery are detrimental to the myocardium, causing acute edema (accumulation of fluid) in the injured myocardial tissue. Myocardial edema (ME) is a hallmark of the ischemic myocardium that is at risk of irreversible injury (infarction), and can be assessed noninvasively with MRI. Imaging the at-risk myocardium would be useful to inform decision on interventional reperfusion treatment, particularly for the patients who cannot receive the treatment promptly after AMI. However, MRI is not available around the clock which may limit its use for assessing patients with AMI. CT is a fast imaging technique and is widely available at hospitals. We have developed a functional CT method for measuring the distribution volume of contrast in the myocardium. We hypothesize that contrast distribution volume (CDV) assessed by CT can be a surrogate marker of ME in infarcted myocardium. The objective of this project was to validate the CDV measurement with functional CT as a useful marker of ME against the gold standard T2W MRI in a pig model of reperfused AMI.

Methods: MI was induced in 7 pigs by occluding the distal left anterior descending artery using a specialized balloon catheter for 1 hr followed by reperfusion. Each pig had CT and MRI studies at 3 days and 12±3 days after the surgical procedure. In each CT study, the heart was scanned with a multi-phase dynamic acquisition protocol after intravenous contrast injection. The dynamic images were analyzed with a new tracer kinetic model to estimate CDV in the normal and infarcted myocardium. The results were compared to the edema assessment with T2W MRI performed on the same days.

Results: Mean CDV in the injured myocardium was 0.39±0.11 ml/g at day 3 post and 0.46±0.20 ml/g at day 12±3 post, which were higher than the corresponding measurements in the normal myocardium (0.14±0.03 ml/g at day 3 and 0.24±0.21 ml/g at day 12±3, p<0.05 for both). T2W MR images confirmed the injured myocardium with a higher CDV on both time points was edematous. The T2 signal intensities in injured and normal myocardium on day 3 and 12±3 post were 77.8±55.4 A.U. vs. 41.9±38.1 A.U. and 65.6±55.7 A.U. vs. 34.0±39.0 A.U. (p<0.05 from infarcted on both days).

Discussion: Accumulation of fluid in the injured myocardium expanded the space for which CT contrast could reside, hence CDV in the injured tissue was higher than normal. Our results showed that increase in CT-measured CDV aligned with a higher T2W signal, and hence the proposed functional CT technique may be useful in assessing ME in the AMI setting. Further testing is warranted to determine if this new imaging method is useful in delineating the extent of salvageable myocardium via edema imaging to inform decision on optimal reperfusion therapy.