ABSTRACTS - Noninvasive Imaging

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1021-54 Effects of Various Microbubble Composition on the Interactions With Activated Leukocytes

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Background: Interactions between activated neutrophils and microbubbles including phosphatidylserine and phosphatidylcholine have been utilized to deliver microbubbles to the site of inflammation. The aim of this study was to examine the effect of various characteristics of microbubbles on this application. Methods: Sonicated albumin (SA: albumin shell, air), Optison® (OPT: albumin shell, octafluoropropane gas), BRA (phosphatidylcholine shell, perfluorobutane gas) and BR14 (phosphatidylserine shell, perfluorobutane gas) were tested. The 4 agents were respectively incubated in RPMI 1640 medium containing serum on the cell culture slides with human neutrophils (2x10^6/mL) activated by phorbol-12-myristate-13-acetate at 37°C to allow interactions on an intravital microscope. Results: At 3 minutes after the onset of reaction, aggregation of 10-15 microbubbles to some of the leukocytes was observed for all agents. At 15 minutes, the number of leukocytes that phagocytosed microbubbles per 50 leukocytes was 30±1 cells for SA, 12±2 cells for OPT, 14±2 cells for BRA, and 27±3 cells for BR14. Conclusions: This study demonstrated that the phosphatidylserine-coated microbubbles are stable as compared with those with albumin shell in the cytoplasm of the leukocyte after phagocytosis. A stable acoustical property may be better provided by those microbubbles at the site of inflammation.


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Background: Peri-infarction ischemia, perifocal deficit adjacent to or around a fixed resting defect, is well detected by nuclear imaging. While real-time perfusion imaging (RTPI) using ultrasound contrast agents has been shown to detect infarcts with good sensitivity and specificity, it's accuracy in the assessment of peri-infarction ischemia (where a resting perfusion defect already exists) is not known. Methods: We employed RTPI modality (Axiolab and ATL Philips) in a canine model (15 dogs) of distal coronary occlusion (CO) and proximal coronary stenosis. Using coronary flow probe recordings, physiologic significance of proximal coronary stenosis was established by confirming abolition of coronary reserve. Contrast agent, Optison was given as slow bolus injections at baseline, during prolonged distal CO, during adenosine bolus stress and during dobutamine stress. Triphenyltetrazolium chloride (TTC) staining was used to verify distal infarction. RTPI recordings at baseline, distal CO and stress protocols were randomly mixed and reviewed blindly. Results: In all but one dog, RTPI detected distal infarct as small as 6% of the left ventricle. The sensitivity, specificity and overall diagnostic accuracy of RTPI in the detection of distal infaracts were: 99%, 87%, and 90%. Sensitivity, specificity, and overall diagnostic accuracy of RTPI in the assessment of peri-infarction ischemia (where a resting perfusion defect already exists) was 67%, 77%, and 71% for adenosine stress and 92%, 92%, and 92% for dobutamine stress. The spatial extent of perfusion defect related to peri-infarction ischemia was similar during both adenosine and dobutamine stress (w=0.92, p<0.001). Conclusion: 1) Even small distal infarcts can be detected by RTPI; 2) Peri-infarction ischemia can be accurately recognized by RTPI during stress; 3) Adenosine and dobutamine stress appear equally reliable in RTPI evaluation of peri-infarction ischemia.