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Poster presentation

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Can 3D-CMR solve the apparent disassociation between carotid artery plaque and outcomes?

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Aggressive pharmacologic strategies dramatically lower incidence of MI and CVA. Yet, when examined at the arterial lumen, only *nominal* changes are seen. CMR, able to detect by 2D (single slice) underlying plaque characteristics, currently has not identified such features by 3D. A 3D volumetric approach may better define plaque 'vulnerability' while disentangling this conundrum.

Hypothesis

We hypothesize that in statin-naive pts with high grade carotid artery disease, 3D CMR integrates plaque components with lipid fractions superior to 2D while solving the 'disassociation' between outcome and % stenosis.

Methods

Via CMR (1.5 T GE, WI), 860-two mm contiguous *in vivo* slices of advanced carotid disease (>50%; mean 63 ± 22) representing 38 complete bilateral human plaques (age 65 ± 13 yrs) were analyzed for 2D and 3D extent of vascular wall: lipid pool, fibrous cap, matrix and minima/maxima of each. All were related to fasting lipids relative to %stenosis via QPlaque (Medis, The Netherlands). Plaque morphology was determined by T1 and T2/PD.

Results

35/38 *in vivo* plaques were successfully imaged. Mean resolution: 1 × 1 × 2 mm. The mg/dL range of LDL was 63-186, HDL: 25-70 and TG: 81-213. Lipid pool represented 19 ± 8% and fibrous plaque 9 ± 24% of total vessel wall. LDL, not total cholesterol ($Chol_T$), was related to mean fibrous cap (mm) ($r = 0.6$ $p = < 0.05$) while triglycerides were related to max fibrous cap but inversely to the lipid pool ($r = 0.6, -0.5$, $p < 0.05$ for both). The LDL:HDL and $Chol_T$:HDL ratios were related to fibrous cap ($r = 0.6$, $p < 0.05$ for both). The $Chol_T$: LDL was related to min fibrous cap (mm) ($r = 0.7$ $p < 0.05$). Via 3D volumetrics only HDL was related to lipid volume while $Chol_T$, LDL, LDL: HDL and $Chol_T$:HDL ratios were all related to fibrous cap volume ($r > 0.5$-0.7, $p < 0.005$ for all). Importantly, relating % stenosis to any vessel wall component or its ratio revealed no relationship.

Conclusion

CMR can depict plaque composition demonstrating important relationships with common lipid fractions and even stronger relations via 3D volumetrics not visible by 2D approaches. Critically, in statin-naive pts, 3D CMR defined plaque morphology is highly related to 'clinical risk' *not* % luminal stenosis, potentially serving as an easily identifiable marker and thus solving the quandary.