Introduction: Systemic lupus erythematosus (SLE) is associated with an increased prevalence of subclinical atherosclerosis and cardiovascular events. Carotid plaque is prevalent in 20-30% of SLE patients under age 36 and in up to 100% of those over age 65. It is important for clinicians to detect carotid atherosclerosis in their earliest stages of development, which can give them guidance to implement appropriate preventive and therapeutic procedures.

Methods: Patients: We evaluated bilateral carotid arteries of 43 SLE subjects (39 female, 4 male, mean±SD age 38.05±9.07yrs) and 18 controls (17 female,1 male, mean±SD age=38.11±5.97yrs). Inclusion criteria were: 1) age 20-50yrs, disease duration>5years, met the ACR Classification Criteria for SLE; 2) control group was age and sex matched, without heart disease and inflammatory diseases.

MRI protocol: 3T clinical scanner (Achieva 3T, Philips Medical Systems, Best, Netherlands) with black-blood vessel wall imaging, including non-contrast T1-, T2- and proton-density-weighted sequences as well as a T1-weighted dynamic contrast-enhanced sequence (only 28 SLE subjects and 12 controls had contrast injection) was used(Figure 1). Low dose Gd-DTPA (0.05mmol/kg) was injected, followed by a repeat post-contrast T1-weighted sequence.

Image analysis: CASCADE software package was used to detect: 1) any focal or diffuse wall thickening in the segment (3.2 cm) around carotid bifurcation; and 2) vessel wall enhancement in the common carotid artery. Per-slice measurements from control subjects were used to establish the 95% upper limits of maximum wall thickness and maximum-to-minimum wall thickness ratio for each of the three sub-segments (common carotid, carotid bulb, internal carotid), which were subsequently used as reference to define wall thickening in all subjects. Percent wall enhancement at a given time point (180 seconds after contrast injection) was calculated using signal intensity measurements on post-and pre-contrast images.

Results: We collected bilateral carotid arterial in each subject and statistical analysis were slice based. Criteria for the thickening were based on the 95% upper limits of the normal controls(Table 1). Each artery with 2 continuous slices met any of the criteria was defined thickening of the artery. Any wall thickening (in common carotid, carotid bulb or internal carotid; in left or right carotid) defined using segment-specific thresholds of absolute wall thickness or wall thickness ratio was found in 18 (41.9%) subjects with SLE compared to 2 (11.1%) in the control group (p=0.02). Compared with controls, SLE subjects were found to have a significantly higher BMI(P=0.008), Cholesterol (P=0.004), Triglycerides (P=0.028),and Low-density lipoprotein (P=0.035). Disease duration, BMI and LDL (Low-density lipoprotein, mmol/L) 3.53±0.91 and mean wall thickness 0.75mm, both were within normal range. While the max/min wall ratio was 1.80, with a ratio in adjacent slice 1.54, two continuous slices met our criterion for wall thickening in CCA. We defined this artery as a slight thickening in left CCA.

Discussion: In SLE group, patients presented a higher prevalence of wall thickening and wall enhancement in carotid artery than volunteer group. Lower HDL, higher ApoA and ApoB100, higher score of SLAM and SLEDAI were associated with premature atherosclerosis in SLE group. Both traditional factors and disease related factors contribute to the atherosclerosis In SLE patients. Dyslipidemia and disease activity were proved to be the most important factors in our study. The higher wall enhancement in SLE patients demonstrated an abnormal wall permeability.

Conclusion: This was one of the first study using MR to evaluate the subclinical atherosclerosis in SLE patients. It represents one of the first attempts that use novel cardiovascular imaging approaches to understand the pathological basis of increased cardiovascular risk in patients with SLE.

References: