Lupus systemic erythematosus is characterised by an increasing risk of premature cardiovascular disease (CVD). CVD is one of the most common causes of death in SLE. Subclinical atherosclerosis in comparison to general population is also more prevalent.

**Methods** A cross-sectional study was carried out from March to November 2015. Patients (119; 94.1% women) were recruited from consultation at the Systemic Autoimmune Diseases Unit for a routine medical check.

The population was divided into three groups: patients with lupus free cardiovascular disease (free CVD), lupus with subclinical cardiovascular disease (sub CVD) (with endotheal dysfunction in ultrasonography but no clinic events) and people with clinical cardiovascular disease.

Patients with subclinical disease have higher cholesterol level when compared to patients with established cardiovascular disease (p value=0.008) and to patients with SLE without vascular disease (p value=0.043). This is due to the lack of knowledge of subclinical damage by the clinician and, in addition to the absence of visible vascular damage, strict control of cholesterol levels is not performed in this group. Likewise, LDL cholesterol levels are elevated in the same context as triglycerides (differences between SLE free and SLE with subclinical SLE).

There were no differences in the activity index, what is likely to be in relation to the low activity they had at the time of inclusion in the study. We do have indeed detected differences in the time of evolution of those who present vascular damage (longer evolution time, 22.88 years) compared to patients without vascular involvement (12.32 years) suggesting that at the same time as more years of disease are accumulated vascular damage accumulates. Likewise patients with vascular damage and subclinical vascular damage were older (55.12 and 54.29, respectively), compared to those with no involvement (42.94 years).

No differences were detected between the use of antimalarials or the deleterious effect of corticosteroids and immunosuppressants (traditionally associated with increasing vascular risk) in the presence of clinical, subclinical or absence of vascular damage.

It is important to know the lupic population with a higher tendencies to have vascular damage for the purpose of greater control.

**Results** A total of thirty-two patients with anti-RNP positive patients were identified during 2008 to 2017 (female n=28, male n=4). The most common initial clinical symptoms were musculoskeletal symptoms including arthritis, arthralgia, myositis, myalgia, and puffy hands (n=10, 31.1%), followed by mucocutaneous symptoms including photosensitivity, malar rash (n=9, 28.1%), cytopenia including hemolytic anaemia, leucopenia, and thrombocytopenia (n=8, 25%), Raynaud’s phenomenon (RP n=7, 21.9%), renal involvement (n=3, 9.4%), serositis (n=3, 9.4%), and enteritis (n=2, 6.3%). Average 9 years of follow-up duration, 87.5% of patients were diagnosed SLE (n=28) while 18.8% of patients were diagnosed SSc (n=6). None of patients diagnosed SSc showed initial clinical symptoms such as enteritis, serositis, cytopenia, or renal involvement. Patients who had RP at the beginning showed significantly high risk for developing SSc (Risk ratio 7.1 95% confidential interval 1.6—31.3). Patients with positive anti-dsDNA (n=17, 53.1% of all patients, p=0.004) and anti-Sm (n=17, 56.3%, p=0.03) anti-Ro/SSA (n=16, 50%, p=0.07) antibody were significantly frequently diagnosed as SLE during follow-up periods. Higher in SLE than those with SSc, while patients with SSc. Of six patients with SSc, three patients (50%) showed anti-Scl 70 antibody, one patient (16.7%) showed anti-centromere antibody, and, of note, two patients (33.3%) showed no SSc specific antibody.

**Conclusion** In patients with anti-RNP positive, patients with Raynaud’s phenomenon have increased risk of developing systemic sclerosis even without SSc specific autoantibodies, while patients with serositis, cytopenia, enteritis, or renal involvement have higher probability to have SLE.