Objective: Aneurysm sac shrinkage following endovascular repair (EVAR) of an abdominal aortic aneurysm (AAA) is an established indicator of surgical success. However, even with a completely excluded aneurysm, the degree of aortic sac shrinkage may vary. This study evaluates AAA sac regression after EVAR in relationship to morphological features of the thoracic aortic wall that are associated with dissection.

Methods: Patients who underwent EVAR at Mount Sinai Hospital between 1996-2017 were reviewed. Patients with type I and type III endoleaks were not included in this sample. The non-aneurysmal segment of the thoracic aorta was evaluated by CT for aortic wall pathology: A) Presence of intimal tears and projections, B) Intimal ulceration, C) Aortic wall thickness > 6mm (Figure 1). An independent investigator, who was blinded to the outcome of sac regression, performed CT analysis. Infrarenal aneurysm sac shrinkage 3 years after EVAR was compared between patients who had all three thoracic aortic wall findings (group 1, 120 patients) and patients without any thoracic aortic wall abnormalities (group 2, 69 patients).

Results: The AAA diameter after EVAR in group 1 patients decreased by 10.54mm±12.97mm compared to a decrease of 0.57mm±15.69 in group 2 patients (mean±std, p<0.001). Additionally, patients in group 1 had fewer type II endoleaks in post-operative follow up scans (22.5% vs 52.5%, p<0.001). AAA related mortality after three years was not significantly different between the two groups (2 deaths vs 0 deaths, p=0.734).

Conclusions: Thickening of the thoracic aortic wall, irregular intimal defects, and ulcers correlated with greater AAA sac shrinkage and fewer type II endoleaks after EVAR. These 3 thoracic aortic morphologies are associated with chronic aortic dissections. Assessing these imaging features in patients undergoing EVAR may be useful in determining the likelihood of aneurysm regression and occurrence of type II endoleaks after EVAR. Identifying differential rates of aneurysm sac regression may have implications regarding the role of subclinical aortic dissection in the etiology of AAA development.