INTRODUCTION: The clinical outcomes after revascularization for arterial occlusive disease are associated with sex differences. Although these sex differences are associated with neointimal hyperplasia, sex differences in arterial identity during remodeling are not known. We characterized sex differences in vessel identity, including the arterial determinant Ephrin-B2, that occur during arterial remodeling after injury.

METHODS: Carotid balloon injury was performed in male and female Sprague-Dawley rats, with or without preceding gonadectomy. Balloon injury was performed on the left common carotid artery, and the contralateral artery was used for control. Ultrasoundography was used to assess hemodynamics, and wall composition was assessed using histology, immunofluorescence, and Western blot (day 14).

RESULTS: There were no significant differences among the groups at baseline. Neointimal thickness was decreased in female rats after carotid injury, and associated with less smooth muscle cell proliferation, less type I and III collagen deposition, decreased TNFα- or iNOS-positive CD68+ cells and increased CD206- or TGM2-positive CD68+ cells. There were no sex differences in these parameters among rats pretreated with gonadectomy. The immunoreactivity of VEGF-A, NRP1, and phosphorylated Ephrin-B2 was reduced, and Notch1 was increased, in female rats. Immunoreactivity of phosphorylated Akt1, p38, and ERK1/2 was also decreased in female rats.

CONCLUSIONS: The sex hormone-dependent decrease in neointimal hyperplasia in female rats after carotid balloon injury is associated with altered arterial identity. Changes in vessel identity after balloon injury could contribute to sex differences in arterial remodeling and might represent a new approach to sex-specific treatment of arterial restenosis.

---

Significant Reduction in Aortic Strain after Endovascular Aneurysm Repair Using Novel Ultrasound Elastography Technique

Maxwell L Wang, BA, Adnan Hirad, MD, PhD, Michael Richards, PhD, Michael C Stoner, MD, FACS, Doran Mix, MD
University of Rochester, Rochester, NY
Rochester Institute of Technology, Rochester, NY

INTRODUCTION: To date, physicians use aortic morphometry to verify successful endovascular aneurysm repair (EVAR). However, aortic morphometry is an incomplete metric in predicting aneurysm stability with growing evidence that biomechanical properties can provide additional information. Here we test Lagrangian Speckle Method ultrasound elastography to detect changes in the biomechanical properties of the aorta after EVAR.

METHODS: Seven patients with abdominal aortic aneurysms (AAA) were imaged with ultrasound elastography after EVAR with longitudinal follow-up of AAA morbidity 5 years after operation via chart review. Mean principal strain of the aorta was determined across the entire cardiac cycle using finite element modeling. Two-tailed paired t-test analysis was conducted on mean principal strain of pre-EVAR vs post-EVAR patients.

RESULTS: Parametric imaging shows significant reduction in aortic mean principal strain of post-EVAR compared with pre-EVAR, t(4) = 2.97; p < 0.05 (Fig. 1). The greatest post-EVAR mean principal strain was found in a patient with endoleak. Chart review of these patients 5 years after EVAR demonstrated either no size change or favorable remodeling of their aneurysms.

CONCLUSIONS: Our results demonstrate that ultrasound elastography detects significant differences in aortic strain after EVAR. In addition, these same patients who had noticeable strain decrements did not require further interventions 5 years later. Further investigation is warranted on the ability of ultrasound elastography detect endoleaks.

---

Surgical Treatment of the Neurogenic Etiology of Median Arcuate Ligament Syndrome

Brittany Davis, MD, Brandon Madris, MD, Richard Hsu, MD, PhD
Stamford Hospital, Stamford, CT

INTRODUCTION: Median arcuate ligament syndrome (MALS) is described as a congenital variation with a low-lying median arcuate ligament causing compression on the celiac artery. This compression is believed to cause gastric pain secondary to ischemia. However, given the robust collateralization among the 3 mesenteric arteries, ischemia from a single artery compression is unlikely to be the source of pain. In addition, the high failure rates of nearly 80% with current surgical treatment of MALS, in which the ligament is resected and the celiac artery decompressed, suggest another etiology. We propose a neurogenic etiology of MALS stemming from celiac ganglion inflammation. We hypothesized that a surgical approach that includes a celiac ganglionectomy in addition to MAL resection will result in overall improved outcomes and lower recurrence rates compared with traditional approaches.