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PS194.
VEGF-A Neutralization Suppresses Experimental Abdominal Aortic Aneurysm (AAA) Formation
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Objectives: Aortic mural angiogenesis is a key pathologic feature of AAA disease. We previously reported that VEGF-receptor 2 expression correlated with experimental AAA progression. This study evaluated the influence of anti-VEGF-A monoclonal antibody (mAb) therapy on AAA formation and progression in the porcine pancreatic elastase (PPE) infusion model in C57Bl/6J mice.

Methods: PPE-infused mice were given anti-VEGF-A neutralizing mAb or control mAb. Aortic aneurysm progression was evaluated via serial transabdominal in vivo ultrasonography and histology at sacrifice.

Results: In control mAb-treated mice, persistent and significant aortic enlargement was noted beginning the third day following PPE infusion. Anti-VEGF-A mAb treatment initiated 3 days prior to PPE infusion significantly attenuated PPE-induced aortic expansion. Histologically, the VEGA-A neutralization resulted in preserved medial smooth muscles and elastin, and reduced mural leukocyte infiltration and angiogenesis. Although anti-VEGF-A mAb treatment initiated 5 days following PPE infusion reduced mural inflammation, aortic diameters continued to enlarge.

Conclusions: Anti-VEGF-A mAb prevents the initiation and subsequent progression of experimental AAAs. The mAb treatment has limited effects on existing aneurysms.

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PS196.
Endotension: Net Flow through an Endoleak Determines its Visibility
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Objectives: Unexplained aneurysm growth despite multimodality imaging following EVAR is often attributed to endotension. We tested a hypothesis that endotension may be from a type 1a endoleak (EL) pressurizing the aneurysm sac without net forward flow, not visualized on standard angiographic imaging.

Methods: A patient-specific aortic aneurysm phantom was constructed of polyvinyl alcohol using 3D molding techniques. A bifurcated stent graft was implanted and the phantom connected to a hemodynamic simulator for testing. Type 1a ELs were created using seven Fr catheters. Three scenarios were studied: complete exclusion (no EL); inflow with no sac outflow; and inflow with sac outflow. DSA imaging was performed at 48kVp at 5fps followed by delayed imaging (1 frame/min) over 30 minutes.

Results: With no EL, the systemic MAP (sMAP) averaged 113 mmHg and Aneurysm Sac MAP (asMAP) averaged 101 mmHg. (Table) With EL without outflow, the sMAP averaged 116 mmHg and asMAP averaged 120 mmHg. EL flow was bidirectional with no net forward flow. With EL with aneurysm sac outflow, the sMAP averaged 119 mmHg and asMAP averaged 105.5 mmHg. EL flow was 39 cc/min across the EL channel. With DSA imaging, the EL with no outflow was noted after >9 min of delayed imaging.

Conclusions: Our model demonstrated a Type 1a EL in the absence of aneurysm sac outflow resulting in full pressurization of the aneurysm sac with biphasic (zero net) flow. This EL was not visible on standard contrast DSA until >9 min. This model may serve as a first step in explaining both the mystery of endotension and in vivo aneurysm sac growth with no detectable ELs using current standard imaging modalities.