JVS Supplement Instructions/Checklist

Required Components

- Basic Components:
  - Abstract title
  - Byline
    - Author’s full name and any co-author(s) full name(s) - first name and last name (surname)
    - Degree credentials for all authors (i.e. MD, MBBS, PhD, etc.)
    - Institutions - when multiple institutions are present, use superscript numbers to indicate which authors belong to which institutions
    - IF THE WORK HAS BEEN PREVIOUSLY PUBLISHED, PROVIDE THE FULL CITATION AFTER THE BYLINE
  - Use common headings: Objective, Methods, Results, Conclusions
  - Cite figures and tables in the abstract (see below) *
    - FIGURES and TABLES SHOULD BE ORIGINAL, UNPUBLISHED
    - Please limit to 1-2 Figures + tables (2 Tables, <<or>> 1 table and 1 Figure) per abstract for best layout
  - Author disclosure for each author and co-author. Include even if there is nothing to disclose.

*FIGURES AND TABLES:

*Figures must be sharp and easy to read at 3 inches wide. If an image is illegible due to small font size, color use, etc., and a new image cannot be provided, the image will be omitted from the final published version. Figure cites in the abstract and figure titles are required. Please limit to 1-2 figures + tables per abstract for best layout

*Tables must be provided in an editable format in the word document as shown below. Table cites in the abstract and table titles are required:

OK (Editable word version, with title):

Table 1: Title of the Table here.

<table>
<thead>
<tr>
<th>Title 1</th>
<th>Title 2</th>
<th>Title 3</th>
<th>Title 4</th>
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<tbody>
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</table>

NOT ACCEPTABLE (Image, not editable, no title):

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place a shunt during CEA. Recent stroke, contralateral ICA occlusion, or contralateral high-grade ICA stenosis are not an indication for intraoperative shunting. CEA for stroke is best done early, with no additional increase in perioperative stroke, thus shielding the patient from the early risk of recurrent stroke.

Author Disclosures: N. Angle: None.

Long-Term Outcomes After Repair of Symptomatic Nonruptured Abdominal Aortic Aneurysms

Karen Trang, BS, Venita Chandra, MD, Whitt Virgin-Downey, BA, Jason T. Lee, MD, E. John Harris, MD, Ronald L. Dalman, MD, Matthew W. Mell MD, MS, Stanford School of Medicine, Stanford, Calif

Objectives: Long-term outcomes comparing symptomatic nonruptured abdominal aortic aneurysms (sxAAA) to asymptomatic AAA (aAAA) have never been reported. We describe long-term outcomes of sxAAA and aAAA after repair at a single academic institution.

Methods: Patients receiving infrarenal AAA repair for sxAAA and aAAA from 1995 through 2015 were included. Ruptured AAA were excluded. Long-term mortality was the primary outcome, determined by medical record review or link to Social Security Death Index. Additionally, long-term mortality and reinterventions were compared after propensity matching.

Results: AAA repair was performed for 1054 asymptomatic aneurysms (383 open repair, 671 [64%] EVAR), and 139 symptomatic aneurysms (60 open repair, 79 [57%] EVAR). Age (73 vs 74, P = 13) and aneurysm diameter were similar between sxAAA and aAAA (6.0 cm vs 5.8 cm, P = 5). The proportion of women was higher for sxAAA (26% vs 16%, P = .003, Table). After propensity matching, there were no significant differences between groups for patient characteristics, AAA diameter, treatment modality, or comorbidities. Perioperative mortality was 5.0% for sxAAA and 2.3% for aAAA (P = .55). By life-table analysis, sxAAA had lower 3-year (62% vs 71%) and 10-year (39% vs 51%) survival (P = .01; Fig) compared with aAAA for the entire cohort. Similar trends were observed for 5-year and 10-year mortality after propensity matching (63% and 40% vs 71% and 52%; P = .08). When stratified by repair type, 5-year and 10-year survival trended lower after open surgery (68% and 42% sxAAA vs 84% and 59% aAAA; P = .5). By life-table analysis, sxAAA had lower 3-year (62% vs 71%) and 10-year (39% vs 51%) survival (P = .01; Fig) compared with aAAA. Reinterventions were more common after EVAR compared with open repair (22% vs 7% for sxAAA, P = .015; 20% vs 4% for aAAA, P = .007). Stratified by repair type, reintervention was similar after EVAR (22% sxAAA vs 20% aAAA, P = .8) and open repair (7% sxAAA vs 5% aAAA, P = .5).

Conclusions: Patients with sxAAA had worse long-term survival and similar aneurysm-related reinterventions compared with patients with a AAA undergoing repair, suggesting a different pathophysiology for sxAAA.

Author Disclosures: V. Chandra: None; R. Dalman: None. E. Harris: None. J. Lee: None. M. Mell: None. K. Trang: None; W. Virgin-Downey: None.

Role of Intraplaque Lipid in Plaque Vulnerability

Gayatri Raghuhaman, PhD; Mary Zuniga, BS; LiXin Wang, MD; Wei Zhou MD; IVA Palo Alto Health Care System, Palo Alto, Calif; University of Arizona, Tucson, Ariz

Objectives: Plaque rupture is a major complication in atherosclerotic cardiovascular disease, and diabetes mellitus exacerbates cardiovascular complications. Cell death, either of macrophages or vascular smooth muscle cells (VSMCs), is believed to be a primary feature contributing to the necrotic core formation and plaque vulnerability. The presence of intraplaque lipid and its role in plaque vulnerability is still underinvestigated. In this study, we examined the extent of lipid oxidation in plaques from diabetic vs nondiabetic subjects who underwent carotid endarterectomy. Oxidative stress within carotid plaques was measured using protein carbonylation assay. Extracted lipids were quantified using oleic acid standard and analyzed for lipid peroxidation. Human coronary artery SMCs were treated with lipid extracts for 18 hours. Cells were analyzed for gene expression with RT-PCR and apoptosis using Annexin V and Caspase-3.

Results: Carotid plaques showed a large variation in oxidative stress in various plaque regions. There was higher lipid peroxidation and protein oxidative modification in the central, more diseased region of carotid plaques than in less diseased peripheral regions. Lipid extracts from diabetic plaques have ~1.8-fold more oxidatively modified lipids compared with the nondiabetic plaques. Human coronary artery SMCs treated with lipids isolated from diabetic plaques show a significantly augmented rate of apoptosis by threelfold. Cytokine analysis of the conditioned medium from these samples showed fourfold higher levels of tumor necrosis factor-α in the diabetic samples. Gene expression data revealed increased resistin and CD40 expression in diabetic vs nondiabetic plaques.

Conclusions: Collectively, these data suggest that lipids within atherosclerotic plaque are not inert. Increased oxidative stress in the vascular bed of diabetic patients results in amplified lipid/protein oxidation. These oxidized lipids in turn facilitate VSMC apoptosis, triggering a possible secondary event leading to plaque vulnerability.

Author Disclosures: G. Raghuhaman: None; L. Wang: None; W. Zhou: None; M. Zuniga: None.

Table. Comparison of symptomatic (sxAAA) and asymptomatic abdominal aortic aneurysm (aAAA) repairs

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<thead>
<tr>
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<th>Propensity-matched cohort</th>
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<tbody>
<tr>
<td>sxAAA (n = 139)</td>
<td>aAAA (n = 1054)</td>
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<td>sxAAA (n = 136)</td>
<td>aAAA (n = 136)</td>
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<td>Age, years</td>
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<td>Non-Caucasian, %</td>
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<td>AAA diameter, cm</td>
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<td>32</td>
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</tr>
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</table>

*Continuous data are shown as mean ± standard deviation and categoric data as indicated.