

**Aortic Remodeling After TEVAR for Intramural Hematoma of the Thoracic Aorta**

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**Objectives:** Our goal was to investigate the extent of aortic remodeling after thoracic endovascular aortic repair (TEVAR) for intramural hematoma (IMH) of the thoracic aorta.

**Methods:** A retrospective review from 2006 to 2012 was conducted of consecutive patients who underwent TEVAR for IMH. Computed tomography scans were analyzed using the TeraRecon digital workstation, and primary data points included IMH thickness, diameter, and volume measurements for aortic true lumen (TLD, TLV) and for total aorta (TAD, TAV) at the site of maximal pathology. Aortic remodeling was evidenced by a TAD/TLD ratio closest to 1.0.

**Results:** Forty-four patients underwent TEVAR for IMH. Twenty-five patients had an IMH with concomitant penetrating atherosclerotic ulcer. Mean age was  $71 \pm 11$  years, with 57% female patients. Operative indications included intractable pain in 31 (70%), rapidly progressing IMH or conversion to dissection in 13 (30%), rupture in 10 (23%), and uncontrolled hypertension in 6 (14%). Technically successful TEVAR was performed in all patients, with 42 (95%) reporting complete relief of symptoms. The 30-day mortality rate was 5%, with a 5% rate of paraplegia or paraparesis. At a mean follow-up of 26 months, there were no additional aortic-related deaths, and the reintervention rate was 11%. At a mean computed tomography scan follow-up of 13 months, all measured data points were statistically improved from pre-TEVAR to post-TEVAR: thickness of IMH (12 mm vs 4 mm;  $P = .01$ ), mean TLD (35 mm vs 37 mm;  $P = .04$ ), mean TAD (47 mm vs 42 mm;  $P = .02$ ), TAD/TLD ratio (1.35 vs 1.14;  $P < .01$ ), and IMH volume ( $103 \text{ cm}^3$  vs  $14 \text{ cm}^3$ ;  $P < .01$ ). The mean  $\Delta$  in TAD/TLD ratio preoperatively to postoperatively for the reintervention group was  $\Delta 0.14$ , whereas the mean  $\Delta$  in the TAD/TLD ratio for the group without reintervention was  $\Delta 0.29$  ( $P = .05$ ).

**Conclusions:** TEVAR is safe and effective in treating IMH, and based on longitudinal computed tomography scan analysis, aortic remodeling is evidenced by normalization of all measured indices.

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**Penetrating Aortic Ulcers: The Fate of the Untreated Aorta**

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**Objectives:** Diffuse atherosclerotic disease increases the risk of penetrating atherosclerotic ulcer (PAU) development; however, much of the literature has classified and studied PAUs as a localized pathology. This study examined the changes in the anatomic characteristics throughout the aorta along with outcomes in patients who underwent thoracic endovascular aortic repair (TEVAR) for a PAU.

**Methods:** This was a retrospective review of patients who underwent TEVAR for a PAU from 2000 to 2012. Standardized anatomic assessment protocol of the untreated aorta (diameter, thrombus, calcification, ulcer) in preoperative and postoperative scans was recorded as well as clinical outcomes.

**Results:** Of 196 TEVARs, seven of eight patients who underwent TEVAR for a PAU had follow-up imaging available. The mean age was  $74 \pm 5.5$  years, and 42.9% were male. Five of seven patients had a single PAU, and two patients had three PAUs. The locations were aortic arch ( $n = 2$ ), descending thoracic aorta ( $n = 8$ ), and infrarenal aorta ( $n = 1$ ). The mean PAU neck diameter and depth were  $16.09 \pm 3.0$  mm and  $12.9 \pm 1.5$  mm, respectively. Six patients were symptomatic, and three had ruptured. Four patients had a concomitant intramural hematoma, and there were two pseudoaneurysms. The mean length of aortic coverage was 122 mm. One postoperative stroke occurred. The median follow-up duration was 27 months. Two patients underwent a second TEVAR: one for pseudoaneurysm expansion and one for stent migration; one postoperative death occurred. The mean maximal calcification index of the aorta ( $0.51 \pm 0.12$ ) did increase slightly by  $0.04 \pm 0.02$  ( $P = .06$ ). Overall, there was no significant change in mean maximal diameter throughout the aorta. Lastly, no new PAUs developed.

**Conclusions:** This study suggests that PAUs are a localized pathological process with no further development of PAUs or significant change in aortic anatomy during follow-up in the untreated segments of aorta. Aortic reinterventions were relatively common in this small series, although they reflected progression of disease at the treatment site, not elsewhere in the aorta.

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**Synchrotron Mapping of Carotid Artery Plaque—A Pilot Study**

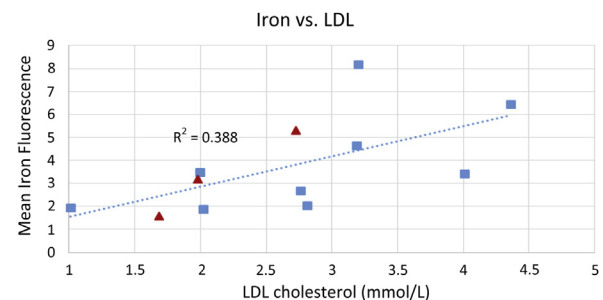
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**Objectives:** This study compared the characteristics of symptomatic and asymptomatic carotid plaques using x-ray fluorescence maps of key elements, including calcium, zinc, iron, sulfur, and potassium.

**Methods:** Patients undergoing carotid endarterectomy in the Regina Qu'Appelle Health Region between April 2012 and May 2013 were recruited. Clinical data were collected on study participants, including age, gender, diabetes, ethnicity, smoking status, blood pressure, cholesterol (total, high-density lipoprotein, low-density lipoprotein [LDL]), symptomatic status of the carotid plaque, date of last ipsilateral neurologic symptoms, and date of surgery. Carotid plaques from participants were frozen, sectioned in 10- $\mu\text{m}$  slices, and desiccated onto Thermanox cover slips. Sections from the most active portion of each carotid plaque were selected for synchrotron imaging. X-ray fluorescence maps were obtained at the Stanford Synchrotron Radiation Lightsource, using an energy of 13.45 KeV, with 40- $\mu\text{m}$  resolution and 200-msec dwell time. Bright light microscopy was also performed to correlate synchrotron images with plaque location.

**Results:** Zinc, calcium, potassium, and sulfur colocalized in areas of plaque mineralization, both in symptomatic and asymptomatic carotid plaques. The intensities of zinc, calcium, potassium, and sulfur fluorescence were unrelated to the symptomatic status of the plaque. Iron localized in areas of plaque away from zinc and calcium. Iron-rich areas were subintimal and were present in both symptomatic and asymptomatic plaques. Microscopy suggests that iron deposits could be related to areas of previous plaque hemorrhage. The intensity of iron fluorescence did not correlate with the symptomatic status of the carotid plaque. A correlation between iron fluorescence in the plaque and LDL cholesterol in the source patient's blood was noted (Fig).

**Conclusions:** Synchrotron imaging of zinc, calcium, sulfur, potassium, and iron did not show differences between symptomatic and asymptomatic carotid plaques. Plaque iron content appears to be related to the source patient's LDL cholesterol.



**Fig.** Mean iron x-ray fluorescence within carotid plaque plotted against the source patient's low-density lipoprotein (LDL) cholesterol in symptomatic (blue squares) and asymptomatic (red triangles) carotid plaques ( $R^2 = 0.388$ ; two-tailed  $P = .03$ ).

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**Carotid Endarterectomy in Patients Undergoing Coronary Artery Bypass Grafting in the Regina Qu'Appelle Health Region**

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**Objectives:** The timing of carotid endarterectomy (CEA) in patients who have severe asymptomatic carotid stenosis and who are undergoing