INTRODUCTION: Ruptured abdominal aortic aneurysm (rAAA) requires prompt diagnosis and intervention. Recent guidelines recommend a door-to-intervention time 90 minutes or less. However, there is a paucity of literature assessing the scope of racial disparity among patients with rAAA. We sought to evaluate the impact of race on timing of rAAA repair.

METHODS: Data were obtained from the Vascular Quality Initiative database on all patients who underwent rAAA repair between 2010 and 2020. End points included door-to-intervention time and perioperative mortality.

RESULTS: Of 5,198 rAAA repairs, 6.9% were performed on Black patients. Black patients were younger and more likely to be female. Other baseline differences included a higher rate of smoking history, coronary artery disease, and chronic kidney disease among White patients, and Black patients were more likely to have diabetes and hypertension and to be offered endovascular aneurysm repair rather than open repair. Door-to-intervention time was significantly longer for Black patients compared with their White counterparts (median 2.8; interquartile range 1 to 6 hours vs 1.5; interquartile range 0.8 to 3 hours; p < 0.001). On univariable analysis, Black patients exhibited significantly lower perioperative mortality (21.8% vs 28.4%; p = 0.007). However, after adjustment, an effect of race on mortality was not observed (adjusted odds ratio 0.9; 95% CI, 0.6 to 1.2; p = 0.53).

CONCLUSIONS: Treatment of Black patients with rAAA was more likely to be delayed beyond the recommended 90-minute benchmark. However, this delay did not seem to have an impact on perioperative mortality. Further research is warranted to determine the underlying factors for this disparity and reassess the impact of the 90-minute time threshold on perioperative mortality.

Figure 1. Predictors of delayed rAAA repair.

Jmd3 Regulates Stimulator of Interferon Genes-Mediated Chronic Inflammation in Diabetic Tissue Repair

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INTRODUCTION: Nonhealing diabetic wounds are a leading cause of lower extremity amputations in vascular surgery. Recent work has identified the Stimulator of Interferon Genes (STING) protein as a regulator of nuclear factor κB-mediated inflammation. Given that chronic inflammation prevents the transition of macrophages (Mφs) from a proinflammatory to a reparative, anti-inflammatory phenotype, the purpose of this study was to investigate cGAS-STING in diabetic wound Mφs.

METHODS: Using a murine model, wound healing curves of STING<sup>−/−</sup> mice were generated. Wound curves were generated for diet-induced obese (DIO) mice after treatment with STING inhibitor. Wound Mφs sorted from control and diet-induced obese (DIO) mice (n = 5/group) on day 5 were analyzed for STING expression by Western blot. Chromatin immunoprecipitation of trimethylation of lysine 27 on histone 3 (H3K27me3) was performed.

RESULTS: STING<sup>−/−</sup> mice display worse wound healing in the later days of wound healing compared with wild-type control. Inhibition of STING confers improved wound healing (p < 0.05) in DIO mice during the latter wound healing phase (days 4–6). DIO wound Mφs exhibited increased cGAS-STING production (p < 0.05) and drove an increase in type I interferons and nuclear factor κB-mediated inflammatory cytokines on day 5 post injury. Chromatin immunoprecipitation assay demonstrates decreased levels of H3K27me3 on the STING promoter in wound macrophages.

CONCLUSIONS: The cGAS-STING pathway is a regulator of Mφ-mediated chronic inflammation in diabetic wounds. It is regulated by JMJD3 histone demethylase, which methylates H3K27. Targeting the STING pathway in a Mφ-specific manner has therapeutic implications for improved wound healing in diabetes.

Low-Volume Hospitals Are Not Associated with Inferior Outcomes after Thoracic Endovascular Aortic Repair

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INTRODUCTION: This study aimed to assess the impact of hospital volume on clinical outcomes after thoracic endovascular aortic repair (TEVAR).

METHODS: Patients undergoing TEVAR for aneurysm, dissection, and trauma between January 2015 and December 2019 were identified in the Vascular Quality Initiative database, a nationally representative vascular surgery database. The participating centers were grouped into either low-volume hospitals (LVHs) or high-volume hospitals (HVHs), based on whether their average annual numbers of TEVAR cases were below or above the overall median. Multivariable logistic regression was used to evaluate the impact of hospital volume on 30-day mortality and major postoperative complications.

RESULTS: A total of 3,584 TEVAR cases (asymptomatic n = 1,720; symptomatic/ruptured n = 1,864) were identified at 147 centers. Among both asymptomatic and symptomatic patients, the median average annual number of TEVAR cases at LVHs and HVHs was 6 and 17 cases, respectively. Asymptomatic patients were less likely to undergo repair of aortic dissection at LVHs vs HVHs (10.2% vs 14.2%; p = 0.04). There was no significant difference in 30-day mortality between LVHs and HVHs (asymptomatic: 3.7% vs 3.7%, p = 0.98; symptomatic/rupture: 9.3% vs 7.3%, p = 0.13). After adjusting for multiple clinical and anatomical factors, being treated in LVHs was not associated with increased 30-day mortality nor an increased risk of major complications.

CONCLUSIONS: Using a large national database, we demonstrate that LVH are not associated with inferior outcomes after TEVAR. The simplicity of procedure might play a role in the similarity of outcomes across the different institutional experiences.

Minimal Change in Abdominal Aortic Aneurysm Sac Regression for Diabetics after Endovascular Repair, Unchanged by Metformin Exposure

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INTRODUCTION: Diabetes is protective for abdominal aortic aneurysms (AAAs), possibly due to metformin, the commonest diabetes treatment with substantial anti-inflammatory properties. Endovascular aneurysm repair (EVAR) is the standard of care for AAA. Annual monitoring after EVAR shows that AAA sacs regress up to 15 mm in the first year, and others continue to expand and require intervention. We hypothesize that metformin will promote AAA sac regression after EVAR in diabetics.

METHODS: Our cohort study included diabetics undergoing elective EVAR (2010-2019) at a multihospital health care system, comparing patients with or without metformin prescriptions. Our primary end point was the yearly change in AAA sac diameter. Secondary outcomes included 1-year reinterventions.

RESULTS: Of 108 patients (age 72.4 ± 7.6 years; 86% male; 94% White), 63 (58.3%) were prescribed metformin with similar comorbid conditions and HgbA1c (6.9 ± 0.8 vs 6.0 ± 1.0; p = 0.67), but smaller pre-EVAR sac sizes (48.0 mm; interquartile range [IQR] 45.0 to 53.0 mm vs 51.0 mm; IQR 48.0 to 54.0 mm; p = 0.037) compared with those without metformin. In a median of 4.0 years (IQ 1.9 to 5.0 years) of follow-up, there was no difference in AAA sac regression (metformin, −3.6 mm; IQR −6.6 to −1.0 mm vs no metformin, −2.4 mm; IQR −5.3 to −0.7; p = 0.22) (Fig.) or reinterventions (metformin n = 13 [20.6%] vs no metformin n = 5 [11.1%]; p = 0.19) across treatment groups. However, sac regression in our overall diabetic cohort (−2.6 mm/y; IQR −5.1 to −0.9 mm/y) was minimal compared with historic observations in all-comers.

CONCLUSIONS: Among diabetics with AAA undergoing EVAR, there were no difference in sac regression associated with metformin prescriptions. However, diabetics experienced minimal sac regression suggesting that diabetics have reduced AAA remodeling capacity and warrants further investigation.

Targeted Delivery of a Nanotherapeutic to Prevent Arterial Restenosis in a Diabetic Environment

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INTRODUCTION: Cardiovascular disorders are the leading cause of death in diabetic individuals with treatment complicated by development of neointimal hyperplasia. We developed a biocompatible peptide amphiphile (PA) nanofiber that incorporates a nitric oxide donor and a collagen-binding peptide targeted to type IV collagen (CBP-PA-SNO). We sought to determine whether our therapy localizes to arterial injury in a diabetic environment.

METHODS: Our cohort study included diabetics undergoing elective EVAR (2010-2019) at a multihospital health care system, comparing patients with or without metformin prescriptions. Our primary end point was the yearly change in AAA sac diameter. Secondary outcomes included 1-year reinterventions.