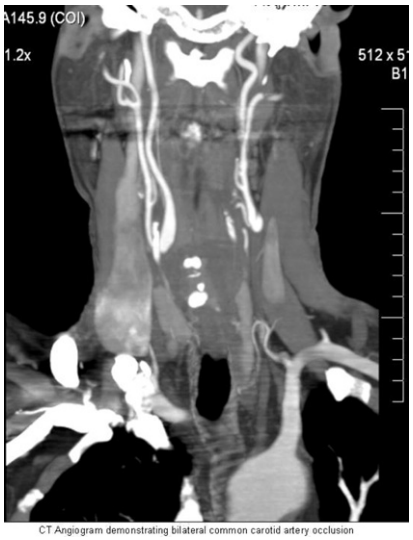
aneurysm formation or arterial dissection. Due to vessel fragility, operative therapy for such disorders has been reserved for compelling indications in which benefit clearly warrants risk, yet assessment of risk is largely clinical with operative decisions guided by factors such as response to previous operations and age at onset of index vascular complications. We report the case of a patient with vascular EDS who required extra-anatomic carotid revascularization.

Methods: A 52-year-old male presented with multiple daily episodes of dizziness and blurring of vision in the left eye. These symptoms had escalated in frequency and severity over a period of 3 months. The patient had documented vascular EDS. He had experienced a stroke in childhood and had required enucleation of his right eye due to trauma. Carotid duplex demonstrated bilateral common carotid artery occlusion with retrograde blood flow in the left internal carotid artery and antegrade blood flow in the right internal carotid artery. CTA confirmed findings of bilateral common carotid artery occlusion (Fig) and internal carotid patency. After reviewing the patient's operative history including an uneventful nephrectomy performed 2 years earlier, operative revascularization was recommended.

Results: We proceeded with cerebral revascularization with a left subclavian to left internal carotid bypass using a 6 mm heparin-bonded PTFE graft. Operation was accomplished and patient recovered uneventfully. He has enjoyed complete resolution of symptoms with unilateral bypass, thus, the contralateral carotid occlusion has not required intervention.

Conclusions: Vascular EDS is caused by a spontaneous point mutation in the COL3A1 gene that encodes the chains of type III procollagen. Increased tissue and particularly vessel fragility results in a higher risk of intraoperative and late surgical complications; thus, elective surgery for individuals with vascular EDS is generally discouraged. To our knowledge, this is the first reported case of symptomatic bilateral common carotid occlusion in a patient with vascular EDS. Our patient had compelling symptoms that raised concern for imminent stroke, thus, prompting a decision to operatively intervene. This case demonstrates that successful outcomes may be achieved with operative management of vascular complications of EDS. However, long-term surveillance for anastomotic aneurysm formation and development of lesions in other vascular beds is mandatory.



CT Angiogram demonstrating bilateral common carotid artery occlusion

Proteomic Analysis of Microparticles from Patients with Abdominal Aortic Aneurysms

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Background: The purpose of this study is to identify proteins that are differentially expressed in subjects with abdominal aortic aneurysm (AAA) and matched controls. This information has the potential to increase understanding of the pathophysiology of AAA and to identify biomarkers suitable to serve as a screening tool for AAA.

Methods: Blood was collected from patients with AAA (maximum diameter greater than 4.5 cm) and matched controls (who had recent imaging studies that excluded AAA). It was processed into plasma, and the microparticles (MPs) were isolated by gel filtration chromatography followed by ultrafiltration. The microparticle proteins were reduced, alkylated, and digested with trypsin into peptides. The peptides were separated by liquid chromatography and analyzed by tandem mass spectrometry. The spectra were searched using SEQUEST™ using a human international protein index database and identified. Manual validation of at least one MS/MS spectrum-peptide sequence match per protein was performed for all proteins determined to be differentially expressed. *T* test and resampling-based significance analysis for spectral counts methods were used to determined statistical significance.

Results: Peptides from 761 proteins were identified from the samples. Spectral count analysis demonstrated that 149 proteins are differentially expressed ($P < .05$). Proteins associated with hemolysis (13; 8.7%) were excluded from further analysis because their presence was thought to be an artifact of the blood collection process. Most of the remaining proteins (106; 71.6%) are over-expressed in AAA, but some (29; 19.6%) are under-expressed. Categories of proteins that are differentially expressed include cell location (nuclear, mitochondrial, extracellular matrix, cytoskeleton, and surface receptors), cell function (signaling proteins, ion pumps and channels, angiogenesis, calcium handling, oxidative stress, small GTPases, inflammation, metabolic processes, transcription factors), cell types (mast cell and platelet), and heat shock proteins.

Conclusions: Proteomic analysis of plasma MPs identified a large number of proteins that are differentially expressed in patients with AAA compared to matched controls. Plasma MPs from individuals with AAA have a dramatically different MP proteome than matched controls. These differences may serve as a marker for AAA and may provide insight into the pathophysiology of AAA.

Does Estrogen Replacement Improve Exaggerated Hyperplasia Following Endarterectomy?

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Background: We have observed that rats fed homocysteine-supplemented diet exhibited increased vascular stenosis as a result of neointimal hyperplasia following a carotid endarterectomy. Since neointimal hyperplasia remains an important clinical limitation of both percutaneous and open vascular procedures, we sought to determine if estrogen replacement may prevent or attenuate neointimal hyperplasia in an environment with hyperhomocysteinemia in rat carotid endarterectomy model.

Methods: Forty young adult female Sprague-Dawley rats were divided into six groups: (1) ovariectomized receiving 17-β estradiol 20 μg/kg/day on homocysteine diet (n = 7), (2) ovariectomized receiving17-β estradiol 20 μg/kg/day on rat chow diet (n = 7), (3) ovariectomized receiving placebo on homocysteine diet (n = 5), (4) ovariectomized receiving placebo on rat chow diet (n = 5), (5) intact receiving placebo on homocysteine diet (n = 8), and intact receiving placebo on rat chow diet (n = 8). Animals on rat chow diet served as control group while those on homocysteine-supplemented diet (4.5 g/kg of DL-homocysteine) served as exaggerated intimal hyperplasia response group. Prior to an open endarterectomy, rats were implanted with an osmotic mini-pump to receive estradiol or placebo and continued with their respective diets for 2 weeks before and after carotid endarterectomy with vein patch closure. Carotid arteries were harvested 2 weeks after the endarterectomy for histological determinations. Endpoints included plasma homocysteine, serum estrogen, and intimal hyperplasia (% lumenal stenosis).

Results: Hyperhomocysteinemia did not produce any significant changes in estradiol level in the rats on homocysteine diet vs those on control diet. Plasma homocysteine level was significantly ($P < .05$) elevated in all homocysteine supplemented groups vs controls except estradiol treated group. Mean serum level of estradiol in intact animals was 20.31 ± 2.12 pg/mL. Placebo-treated ovariectomized rats had mean serum estradiol level of 13.04 ± 1.31 pg/mL whereas ovariectomized animals receiving estradiol had a sevenfold higher level: 94.93 ± 7.40 pg/mL. Intimal hyperplasia or percent lumen stenosis was significantly increased in the placebo-treated ovariectomized homocysteine group vs intact on homocysteine or control diets (47.8% ± 1.74% vs 35.5% ± 1.57%, $P = .0007$ and vs 23.8 ± 0.96%, $P = .0001$, respectively). The exaggerated intimal hyperplasia in the placebo-treated ovariectomized homocysteine group was significantly reduced by estrogen treatment and so was the plasma homocysteine level (47.8 ± 1.74% vs 29.6 ± 0.69%, $P < .0001$ and 19.76 ± 1.67 μmol/mL vs 4.65 ± 0.66 μmol/mL, $P < .0001$, respectively). Estrogen replacement in ovariectomized group on homocysteine diet reduced intimal hyperplasia to that of the intact or ovariectomized rats on control diets (23.8% ± 0.96%, $P = .06$ and 29.0% ± 5.51%, $P = .87$, respectively).

Conclusions: This preliminary study shows that estrogen has a beneficial effect on the hyperplastic response. Estradiol was effective at lowering plasma homocysteine levels. Treatment of ovariectomized rats with estradiol resulted in reduction of intimal hyperplasia response following carotid endarterectomy in rats on homocysteine or control diets as well as reducing the homocysteine level.