Abdominal aortic aneurysms 1

Cardiovascular
Time: 15:10 - 16:40
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Posters for this session are on display on Wednesday 11th July in Liffey A.

Chairs: Elena DiMartino and Thomas Christian Gasser

O1371 - Predicting growth and rupture of abdominal aortic aneurysms; What have we learnt from retrospective clinical studies based on finite element modeling of wall stress and strength?

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Abstract

Surveillance and treatment of abdominal aortic aneurysms (AAAs) is presently guided by repeated maximal external diameter measurements (Dmax). However, it is estimated that 5-30% of all ruptures in different patient cohorts rupture at diameters less than 60 mm. Furthermore, many patients with large aneurysms never rupture, suggesting that many patients undergo unnecessary surgery. We therefore need to look for better predictors of AAA rupture besides Dmax.

In a longitudinal retrospective follow up of 100 patients with at least a minimum of a 4 year follow up period at our center, finite element modeling (FEM) derived Peak wall rupture index and semi automatic Dmax measurements were superior to the clinical DMax in predicting growth and the need for surgery. No ruptures occurred in this cohort. Furthermore, we show that FEM can be performed in a clinical setting and integrated into the work care flow of AAA patients

In the Stockholm Aneurysm Rupture cohort we identified 283 patients that had been admitted to a hospital with ruptured AAA (rAAA) and of those 85 patients (30%) had a previously identified AAA. 14 % of the patients had a Dmax less than 60 mm and 7.2% less than 55 mm. In a subset of the patients, Computer tomography examinations had been performed 1 year or more before the rupture and these examinations were compared to an intact AAA cohort with similar DMax. . We identified the peak wall rupture index, the suprarenal aortic size index and the lumen area as potential novel predictors of rupture. However, only in 50% of the patients could the site of rupture be predicted and some patients with low rupture indices did rupture.

In summary, retrospective clinical studies based on FEM of AAAs have added valuable

O1373 - Micro-structural damage during the early phase of Angiotensin II-induced dissecting aortic aneurysm: the role of aortic biomechanics

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Abstract

Angiotensin II-infused ApoE$^{-/-}$ mice are a popular model for aortic aneurysm and dissection. We have recently demonstrated that the thoraco-abdominal lesions in these mice start with a medial tear near the ostia of celiac and mesenteric arteries. Given the location-specific nature of the disease, we hypothesized that the local mechanical equilibrium may drive disease initiation [1]. In order to investigate this hypothesis we subsequently developed a novel computational approach to evaluate the in-vivo strain field in the abdominal aorta. Combining ex vivo synchrotron images with in vivo micro-CT, we incorporated model features such as non-uniform aortic wall thickness, non-uniform stretch field and the inclusion of small aortic side branches into our computational models and showed how these often overlooked features impact the location of hotspots in the computed strain field [2]. In our current work we validate these simulations with image-guided histology in order to investigate whether regions of high strain collocate with sites of micro-structural. N=10 ApoE$^{-/-}$ mice were infused with Angiotensin-II for 3 days and subsequently underwent a contrast-enhanced micro-CT scan prior to euthanasia. The aorta was imaged ex-vivo using high-resolution Phase-contrast X-Ray Microscopy (PCXTM) at 6.5 um isotropic resolution. The same protocol was followed for n=6 saline-infused controls. An in-house automated framework was implemented to morph the non-pressurized non-stretched ex-vivo PCXTM geometry onto the pressurized stretched in-vivo micro-CT geometry [2]. For each animal the output was a mouse-specific structural finite element simulation. Contrast agent infiltration in the aortic wall was used to detect the location of micro-ruptures in the tunica media [1] and image-guided histology was performed to validate and quantify the vascular damage. Preliminary results show good agreement between hotspots of early vascular damage and hotspots of computed maximal strain. The highest strain values occurred invariably in the vicinity of the celiac and mesenteric arteries and collocated with intramural micro-ruptures and leukocyte infiltration. Moreover, the inter-subject variability of the maximal strain locations (cranial/caudal or right/left of the ostium) corresponded qualitatively to the inter-subject variability of PCXTM-detected contrast agent leakage. We conclude that strain concentrations near side branches may play an important role in disease initiation and could partially explain the focal nature of the disease.

References

[1] *Trachet, B., Aslanidou L., Piersigilli A., Fraga-Silva R., Sordet-Dessimoz J., Villanueva-Perez P., Stampanoni M., Stergiopulos, N and Segers, P.,"Angiotensin II infusion into ApoE-/- mice: a model for aortic dissection rather than for abdominal aortic aneurysm?", Cardiovasc Res,* ***113****, 1230–1242 (2017).*