SATURDAY POSTERS

ORAL PRESENTATIONS IN POSTER AREA

MECHANISMS OF HYPERTENSION

SUBCLINICAL RENAL DAMAGE IS ASSOCIATED WITH A REDUCED CHOROIDAL THICKNESS IN PATIENTS WITH PRIMARY HYPERTENSION

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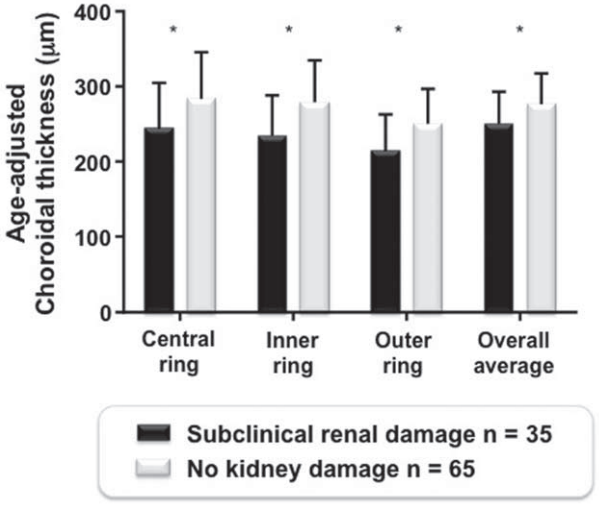
Objective: The retina is considered the easiest accessible window to study the state of the systemic microcirculation, even if the choroid is the most important vascular layer of the eye. Our understanding of the choroid has been greatly increased in last years since the introduction of advanced techniques of optical coherence tomography (OCT). Our study was aimed to assess choroidal thickness by using Swept-Source OCT (SS-OCT) in essential hypertensive patients (EHs) with and without subclinical renal damage (SRD).

Design and method: We enrolled 100 EHs of which 65 without kidney damage and 35 with SRD. In all the participants SS-OCT and a routine biochemical work-up were performed. Glomerular filtration rate (GFR) was estimated by the CKD-EPI equation (eGFR). SRD was defined, by the presence of microalbuminuria or eGFR between 30 and 60 mL/min/1.73 m2. OCT measurements were performed according to the Early Treatment Diabetic Retinopathy Study (ETDRS) protocol, that divides the macula into 9 subfields. The circular grid consists of 3 concentric rings. The inner and outer rings are further divided into quadrants: temporal, nasal, superior, and inferior.
Furthermore, we calculated the average of the individuals values of the four quadrants separately for the inner and the outer ring. The average of all the 9 regions of the ETRDS grid (including the inner, the outer and the central rings) was also calculated.

Results: EHs with SRD showed thinner choroidal thicknesses than those without kidney damage (all $p < 0.05$), even after adjustment for age (figure).Overall choroidal thickness correlated significantly and directly with eGFR ($r = 0.36$) and negatively with urinary albumin excretion ($r = – 0.39$). The association of choroidal thickness with SRD was confirmed in multiple logistic regression analyses once the effect of age, anti-hypertensive therapy and triglycerides was accounted for. The odds ratio of having SRD associated with a standard deviation increase of overall choroidal thickness was 0.43 (0.24–0.75, 95% confidence interval; $p = 0.007$).



Values are given as means + SD
* $0.05 < p < 0.01$

Conclusions: Our study confirms the close relationships between changes in ocular microcirculation and renal dysfunction.

TRANSGLUTAMINASE-2 CONTRIBUTES TO REACTIVE OXYGEN SPECIES PRODUCTION IN MICE INFUSED WITH ANGIOTENSIN-II

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Objective: Transglutaminase type II (TG2) is a pleiotropic enzyme that exhibits various activities and it is involved in diverse biological functions, including cell signaling, cytoskeleton rearrangements, displaying enzymatic activities. We previously demonstrated that TG2 may contribute to angiotensin II-induced reduction of NO bioavailability as well as to the impaired vascular functional and structural alterations induced by angiotensin II. Here we hypothesized that TG2 may contribute to increased production of reactive oxygen species (ROS) in the vasculature of angiotensin-II-treated mice.
Design and method: TG2-knockout mice (TG2-K/O, [8]weeks old, $n = 6$) and age-matched wild type (WT) control mice were treated or not with angiotensin-II. (400ng/kg/min) for 14 days. TG2 activity in aorta was measured by ELISA. ROS. production in aorta was evaluated by dihydroethidium staining. The expression of angiotensin type1 receptor (AT1R), TG2, NOX-1, and ERp72 (the positive modulator of NOX-1) was evaluated in aorta by immunoblotting, coimmunoprecipitation analysis was also performed.

Results: As expected, TG2-K/O lacked TG2 expression and activity. Angiotensin-II significantly increased (2-fold) TG2 expression and activity only in WT. AT1R expression in aorta was not influenced by Angiotensin II treatment in both WT and TG2K/O mice. ROS production was similar in WT and TG2-K/O and increased only in angiotensin-II-treated WT (+9%, $p < 0.01$). NOX-1 and ERp72 expression was similar in WT and TG2-K/O. Angiotensin-II significantly increased NOX-1 (+23%, $p < 0.01$) and ERp72 (+29%, $p < 0.01$) only in WT. Only in aorta from WT and not from TG2-K/O, TG2 was successfully immunoprecipitated by AT1 and ERP72, indicating that TG2 is able to interact with both proteins, and suggesting that it may be involved in angiotensin II- induced NOX modulation and ROS production.

Conclusions: Angiotensin-II increased ROS production and NOX-1 expression and activation only in presence of TG2 in WT. TG2 interacts with both AT1R and ERp72. Thus, TG2 may contribute to NOX-induced ROS production in mice treated with angiotensin-II.

RETINAL ARTERIOLAR MICRO-CONSTRICTIONS EVALUATED WITH ADAPTIVE OPTICS: A NOVEL MARKER IN HYPERTENSION

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Objective: Retinal arteriolar remodeling is an early marker of subclinical target organ damage in arterial hypertension. Through adaptive optics which is totally noninvasive and highly accurate, it is possible to measure changes in arteriolar diameter within 1 mm accuracy. The aim of this study was to evaluate a new marker describing internal diameter variability of the supero-temporal arteriole in hypertensive patients before and after blood pressure control.

Design and method: Adaptive Optics RTX1® Camera (ImagineEye, Orsay, France) was used to capture three consecutive images along the supero-temporal arteriole. Wall Thickness (WT) and internal diameter (ID) were measured to calculate Wall-to-Lumen Ratio (WLR) and Wall Cross-Sectional Area (WCSA). A coefficient of variation (CV) for ID was calculated for each group by the following formula: (standard deviation ID/mean ID)*100 over three consecutive measurements. Subjects with a CV ID > 75% were classified as irregular. Uncontrolled hypertensive subjects in the irregular group were given an antihypertensive pharmacological treatment and were reevaluated 1 month after.

Results: 44 patients were analyzed (mean age 47.7 ± 11). Median CV ID in the irregular group was 11% [IQR 9.0–15.0] as compared to 2.0% (regular group) [IQR 1.0–4.0], $p < 0.001$. Patients in the arteriolar irregular group had an increase in home blood pressure (148.3/96.3 vs 130.7/ 82.6 mmHg, $p < 0.01$). They had