272 Mechanisms of vision loss in early diabetes: Connecting new findings from retinal imaging and functional techniques in patients to basic models - Minisymposium

Program Number: 2089
Presentation Time: 3:45 PM–4:05 PM
Imaging damage to retinal vessels in diabetic patients
Stephen A. Burns. Indiana University, Bloomington, IN.

Presentation Description: Diabetic retinopathy has traditionally been staged according to features visible from retinal examination and retinal photography. Adaptive Optics retinal imaging is now allowing us to visualize and characterize changes that are not readily detected with conventional imaging. This presentation will present change to capillary structure and capillary density, as well as changes to the vascular walls.

Commercial Relationships: Stephen A. Burns, AEON Imaging (I), Nidek (C)
Support: NEI EY04395; Fight for Sight

Program Number: 2090
Presentation Time: 4:05 PM–4:25 PM
Retinal pathology in diabetes and systemic comorbidities

Presentation Description: High resolution imaging techniques such as adaptive optics scanning laser ophthalmoscopy (AOSLO) and spectral domain optical coherence tomography (SDOCT) allow the assessment and quantification of cellular level changes in the diabetic retina in vivo in the human eye. Thinning and disorganization of the inner neural retinal layers as evaluated by SDOCT may have functional implications for patients both prior to the development of clinically visible vascular lesions and once these lesions have become manifest. AOSLO imaging allows identification of subclinical vascular lesions such as microaneurysms that are too small to be visualized on standard fundus photographs or clinical examination. The combination of these techniques potentially provides the ability to better understand interactions within the neurovascular unit in diabetes. The contribution of other systemic diabetic comorbidities to retinal pathology is also being explored and will be discussed, including available evidence for retinal vascular changes with cardiovascular disease and diabetic nephropathy.

Commercial Relationships: Jennifer K. Sun, Boston Micromachines (F), Optovue (F)
Support: NIH/NEI R01 EY024702-01; JDRF 3-SRA-2014-264-M-R, 17-2011-359; NIDDK 5 P30 DK036836-24 P&F Grant; Eleanor Chesterman Beatson Childcare Ambassador Program Foundation Grant; Massachusetts Lion Eye Research Fund; Boston Micromachines

Program Number: 2091
Presentation Time: 4:25 PM–4:45 PM
Changes over time in retinal vessels in patients with early diabetes
Richard B. Rosen. 1New York Eye and Ear Infirmary, New York, NY; 2Ophthalmology, Ican School of Medicine at Mount Sinai, New York, NY.

Presentation Description: Adaptive optics scanning light ophthalmoscopy using an offset pinhole (OP AOSLO) configuration enables non-invasive imaging of the dynamics of retinal microvascular walls, lumen, and blood flow, without the need for any exogenous contrast agent. We used OP AOSLO to survey and monitor subclinical microvascular changes over time in patients with diabetic retinopathy, including capillary perfusion remodeling, loop formation and resolution, microaneurysm expansion and regression. This technique provides a dynamic longitudinal view of the histopathology of aberrant diabetic microvascular development.

Commercial Relationships: Richard B. Rosen, None
Support: Marrus Family Foundation, Bendheim-Lowenstein Foundation, Wise Foundation, Edith C Blum Foundation, GRF, RPB, Burroughgs Wellcome Fund, and NIH grant P30EY001931

Program Number: 2092
Presentation Time: 4:45 PM–5:05 PM
Dysregulation of blood flow in the diabetic retina
Eric A. Newman. University of Minnesota, Minneapolis, MN.

Presentation Description: Activation of the retina by flicker stimulation evokes vasodilation and increased blood flow in the retinal vasculature. This hemodynamic response, termed functional hyperemia, brings added oxygen and glucose to active neurons. Flicker-evoked vasodilation is mediated by neuronal stimulation of retinal glial cells and the release of vasodilating agents, including PGE2 and epoxyeicosatrienoic acids, from the glial cells. Signaling from glial cells to retinal vessels is suppressed by nitric oxide (NO). In patients with diabetic retinopathy flicker-evoked vasodilation and functional hyperemia are substantially reduced, possibly rendering the retina hypoxic. Inducible nitric oxide synthase (iNOS) is upregulated and NO levels are increased in the diabetic retina. Inhibition of iNOS by aminoguanidine reverses the loss of flicker-evoked vasodilation in an animal model of diabetic retinopathy, restoring functional hyperemia to normal. Previous work has demonstrated the efficacy of inhibiting iNOS in slowing the progression of diabetic retinopathy. This effect could be due, in part, to the restoration of functional hyperemia.

Commercial Relationships: Eric A. Newman, None
Support: Fondation Ledaq and NIH grant EY004077

Program Number: 2093
Presentation Time: 5:05 PM–5:25 PM
Early functional changes in diabetic patients measured with mfERG, contrast sensitivity, and SKILL Card
Marcus A. Bearse. UC Berkeley, Berkeley, CA.

Presentation Description: Vision and retinal function changes occur early in diabetes, even in the absence of clinical signs and history of diabetic retinopathy. This presentation will discuss our observations of these changes, focusing primarily on local retinal function measured with the multifocal electroretinogram (mfERG), and on vision changes measured by contrast sensitivity and the Smith-Kettlewell Institute Low Luminance (SKILL) Card. These measures indicate that significant changes are produced by diabetes, and tend to worsen with the onset of clinical diabetic retinopathy. Models based on the mfERG are highly predictive of the appearance of retinopathic lesions.

Commercial Relationships: Marcus A. Bearse, None
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