Retinalneurodegeneration in diabetes

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Diabeticretinopathyiscommonlyclassifiedas a chronicretinalmicrovasculardisorder, characterized by vessel hypermeability and progressiongretinal ischemia. Thereisgrowingevidencethat the neural retina isalso, and perhaps more precociously, alteredby hyperglycaemia. Therefore, retinalneurodegenerationmay be a new target forclinicalinvestigations and future treatment approaches. Spectraldomain OCT hasbeenrecentlyused to analyze in detailretinallayers in order to detectsubtleinitiallesions in differentretinaldisorders. The diabetic retina hasbeen just partlyevaluated to detectprecociouschangesinvolvingallneuralcomponents, including pure neuralcells, macroglia and microglia. A group of diabeticeyeshasbeeninvestigated, using a spectraldomain OCT with an automaticretinallayeringtool, toevaluate retina neurodegeneration.

No statisticallysignificantdifferenceswerefoundfor ageamongallgroups, and for diabetesdurationamongdiabetics. In the no diabeticretinopathy (no DR)and diabeticretinopathy (non proliferative retinopathy, DR) groups, usingautomaticlayering, the meanthickness of inner retina wassignificantlyreducedcompared to controls ($p<.001$), no changewasdetected in the outer retina. both in the fovea and pericentral area. A significantincrease of innerlimiting membrane, innerplexiform and nuclearlayerswasfound in DR eyes versus controls,whereas a significantdecrease of retinalganglioncell and retinalnervefiberlayerswasdocumented. The inter-grader agreementwasatleastsubstantial for allmeasurement, confirming the reliability of spectral domain OCT quantification.Increasedthickness of retinallayersmainlycorresponding to retinal(macro)glialcells , evenbefore the appearance of clinicallydetectableretinopathy, confirms in vivo early(macro)glialcellsactivation in diabetic retina. These data stronglysuggestsa veryearlyreactive and degenerative processsboth in neural and glialcells of diabetic retina .