54) **Intraretinal neovascularization in experimental renovascular hypertensive rats.**

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Introduction Intraocular neovascularization is a common pathological change in some ischemic conditions; arteriolosclerotic retinopathy, diabetic retinopathy, retinopathy of prematurity, senile macular degeneration and etc. Its developmental mechanism, however, has not been fully understood. In this paper, we studied the histologic characteristics of intraretinal newly formed vessels which were observed in rats with a long duration of severe retinopathy induced by experimental renovascular hypertension.

Material and Method Eleven Wistar-Kyoto strain rats were used. Renovascular hypertension was produced by Goldblatt-Drury's method (1938) in 4 rats. After a duration of sustained hypertension with severe hypertensive retinopathy for 6 months or more, the eye ball was enucleated for the histopathologic study and the changes were compared with those of 7 age-matched control normotensive rats. Removed eyeballs were immediately immersed in 2.5% glutaraldehyde with a phosphate buffer pH 7.2. The eyeballs were bisected at the equator in the same fixative and the posterior half was dissected into small pieces. The specimens were reimmersed for 2 hours, post-fixed in phosphate buffered cold 2% O_3O_4 for 2 hours, dehydrated and embedded in Epon 812 resin. Then the specimens were processed for light and electron microscopy. For light microscopy, the sections were stained with toluidine blue and for electron microscopy, the thin-sections were stained with uranyl acetate and lead citrate.

Result The animals with severe hypertension for more than 6 months marked dystrophy of the outer layer. Few capillaries were seen in both of the retina and choroid. In the atrophied areas of the retina, proliferative tissues composed of retinal pigment epithelial cells and newly formed microvessels were frequently observed. Most of these microvessels were characterized by a large nucleus of the endothelial cell and markedly narrowed lumina. Similar vessels also observed in the pigment epithelial layer, inner plexiform layer and, though it was rarely, in the nerve fiber layer. No evidence was obtained which suggested that these vessels through Bruch's membrane extending from the choroid. Some of these vessels surrounded cytoplasmic processes of retinal pigment epithelial cells, however, showed fenestrations in their vascular wall (endothelial cells).

Discussion and Conclusion Although intraocular proliferative changes have been reported in human hypertensive retinopathy, there has been no report which documented such a pathological change in spontaneously hypertensive rat (SHR). In present study, we demonstrated that retinal neovascularization is able to be produced by tissue ischemia due to long standing hypertensive state even in the experimental renovascular hypertensive rat, though the cause of difference between the retinopathy in SHR and retinopathy in Goldblatt type renovascular hypertensive rats referring the occurrence of retinal vascularization is not clear.

Present findings suggest that these newly formed vessels in the retina originated from the retinal vessels but not from choroidal vessels, and the retinal pigment cells play some roll, as a mediator, in the cellular differentiation for intraretinal reovascularization.