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Vascular changes in the retina of hypoxic neonatal rats

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The immature retina is extremely susceptible to hypoxic-ischemic conditions resulting in the development of retinopathy. Hypoxia is an underlying factor in many conditions such as compromised pulmonary function and cyanotic heart disease which are important aetiological factors in the development of retinopathy. This study examined vascular changes in the retina of neonatal rats subjected to hypoxia (5% oxygen+95% nitrogen) for 2 h. The expression of endothelial nitric oxide synthase (eNOS), vascular endothelial growth factor (VEGF) and endothelial tight junction proteins such as claudin-5, occludin & ZO-1 was examined in the retina along with ultrastructure of the blood vessels. Increased mRNA and protein expression of VEGF and eNOS along with a reduced expression of claudin-5, occludin & ZO-1 was observed in hypoxic retinas. Specific localization of VEGF was observed in the astrocytes closely associated with the blood vessels. The blood vessels expressed eNOS immunoreactivity and appeared to be dilated as compared to the vessels in the controls. Although the tight junctions between the endothelial cells appeared to remain intact at the ultrastructural level, expression of claudin-5, occludin & ZO-1 was altered in the hypoxic animals. Endothelial cells often showed vacuoles and multivesicular aggregations in the cytoplasm following the hypoxic injury. Increased leakage of intraperitoneally administered tracers rhodamine isothiocyanate and horseradish peroxidase was detected in the retina of hypoxic rats suggesting increased permeability of the blood vessels which may be mediated by VEGF, eNOS and changes in the tight junction protein expression. The structural and molecular changes in the endothelial cells may reflect impairment of transendothelial transport in the developing retina. It is suggested that vascular changes may be a major factor contributing to degenerative changes such as retinal ganglion cell death following a hypoxic injury. Administration of melatonin, a neurohormone, was beneficial in suppressing the vascular permeability and cell death as well reversing the structural changes in the blood vessels.

Biography

Charanjit Kaur completed her Ph.D from the Department of Anatomy, Faculty of Medicine, National University of Singapore. She is a Professor and Research Director at the Department of Anatomy. She has published more than 120 papers in reputed journals, has written many book chapters and is a reviewer for more than 75 journals (e.g Brain, FASEB Journal, Pediatrics, Investigative Ophthalmology and Visual Science and many more). She has reviewed grants for Austrian Science Fund, Wellcome Trust, Swiss National Science Foundation, Research Grants Council Hong Kong among many others. She is serving as a section editor/editorial board member on many journals.