(S4)

Patofizjologia zmian jaskrowych

Pathophysiology of glaucomatous damage

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Glaucoma is phenomenologically defined as a disease with characteristic loss of retinal ganglion cells and their axons combined with a tissue remodeling of the optic nerve head and the innermost layer of the retina. This leads clinically to a visible cupping of the disc and to a measurable thinning of the nerve fiber layer of the retina. The patients experience a progressive visual field damage together with a decrease in contrast and color sensitivity.

A number of major risk factors are meanwhile well known. Some can be influenced such as IOP increase, blood pressure drops or a vascular disorders. Others can not be influenced, such as genetic predisposition, refraction, race, gender and age.

The exact mechanisms by which these risk factors lead to the damage is not yet known. We know that some ganglion cells die by apoptosis, but we do not know exactly the individual steps that initiate the apoptosis. We know that the glial cells are involved in the tissue remodeling and probably also in the neural cell damage. Astrocytes are activated both by mechanical as well as by ischemic stress.

Based on the known facts, we can assume today that ischemia /reperfusion plays a major role in these patho-mechanisms. Glaucoma patients have on the average a reduced ocular perfusion. Whereas, the baseline blood flow in some glaucoma patients is still normal, an insufficient response to a challenge such an increase of IOP or a decrease of blood pressure can already be observed. Most of the patients with progressive glaucomatous damage despite a only moderately elevated or even normal IOP have altered autoregulation of ocular perfusion. Interestingly, signs of reperfusion damage could even be found in the circulating lymphocytes. Gene expression analysis of lymphocytes gives further insight into the patho-mechanisms of individual patients and indicates, whether a damage is present or not.

The future basic research will establish the patho-mechanism on a molecular level and the clinical research will establish further the connection of the risk factors with the damage.

(S5)

Protezy drenujące w leczeniu jaskry

Glaucoma drainage devices in therapy of glaucoma

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Glaucoma drainage devices also termed aqueous shunts (AS) consist of a silicone tube that is inserted into the anterior chamber, and a plate made of silicone or polypropylene, the explant. The later is positioned between the rectus' muscles and within some weeks the surrounding tissue forms a fibrous bleb around the plate. This serves as permanent filtration reservoir. AS are widely used in the USA. Recurrent failure of filtrating surgeries is the main indication for the use of AS. Other indications include situations, in which the formation of a filtering bleb seems to be unpromising because of extent conjunctival scarring. Qualified success is achieved for many years in 50 to 100% of the treated eyes, according to the patients selection. The most serious complication is postoperative hypotonia, that can lead to serious chorioidal detachment, suprachorioidal haemorrhage, flat anterior chamber and corneal decompensation. To avoid this complication some devices i. e. the Ahmed Glaucoma – and the Krupin valve have integrated mechanisms to sustain a residual i. o. pressure. With other devices i. e. the Molteno and the Baerveldt devices the tube has to be temporarily ligated until the bleb formation is on the way. The lecture will be given about the different types of AS, including our own experience with the Ahmed Glaucoma Valve. There are no obvious differences between the different AS regarding the success of pressure regulation. With appreciation of indications and management of complications AS are an useful option in the management of complicated glaucoma, that carry a high risk of failure from conventional filtering surgery.

ISSN 0023-2157 Index 362646