DIABETIC RETINOPATHY

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DIABETIC RETINOPATHY IN TYPE I DIABETES. INCIDENCE AND RISK FACTORS IN 10-YEARS STUDY.

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Purpose. The aim of this study is: 1) to report the incidence of diabetic retinopathy in a group of 85 IDDM patients with normal fundi at the first control, observed for a period of 10 years; 2) to verify if the long-term follow-up of ERG oscillatory potentials (OPs) can be useful to detect the onset of the early diabetic retinopathy (DR).
Methods. The patients were submitted to one control per year for 10 years including: ERG registration, refraction and corrected visual acuity, fundus oculi examination and fluorescein angiography at the first control and after, if necessary.
Results. At the end of the follow-up normal fundus was still present in 55 patients (Group A); the other patients showed a diabetic retinopathy (Group B) which was moderate (non proliferative) in 27 and severe (proliferative) in 3 cases. The mean duration of disease was significantly different between group A and group B. Moreover, the patients who developed a DR showed a slow and progressive decrease of the OP amplitude during the follow-up.
Conclusions. In conclusion the "duration of disease" parameter is a true risk factors for DR; a decrease of OP amplitude during the follow-up, is suggestive for the risk of DR and it worsen the prognosis.

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VEGF DISTRIBUTION IN DIABETIC RETINOPATHY

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Purpose. To determine the staining profiles of VEGF in diabetic retinopathy.
Method. Immunochemical localisation of VEGF, using an antibody raised against the 165 amino acid form of human VEGF, was carried out using the ABC method on a) specimens of normal human retina (n=15), b) diabetic retinas with no overt retinopathy (n=25), c) diabetic retinas with clinically defined retinopathy of varying stages (n=17), and d) surgically resected preretinal membranes (n=19). The degree and pattern of positive immunostaining was recorded.
Results. VEGF staining was absent in normal retinas. In the majority of diabetic retinas with no overt retinopathy staining was localised to the thickened basement membrane of the retinal vessels. In retinas with retinopathy moderate immunostaining was observed throughout the inner retina. Intense staining was observed associated with intraretinal vessels adjacent to areas of active preretinal neovascularisation. VEGF staining was intense in all excised PDR membranes, being localised to blood vessels in the actively growing regions of the membranes and to many regions of acellular matrix.
Conclusions. This study supports a role for VEGF in the aetiology of diabetic retinopathy.

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GLIAL CELL INVOLVEMENT IN VASCULAR OCCLUSION OF DIABETIC RETINOPATHY

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Purpose: Occlusion of the retinal vascular bed is a key factor in the development of proliferative diabetic retinopathy. Thus, it is assumed that neovascularizations develop because of growth stimulation from vasogenic factors released from retinal tissue that is ischaemic and hypoxic because the normal retinal vessels have been occluded. However, the pathophysiological basis for retinal vascular occlusion in diabetic retinopathy is largely unknown. In the present study the role of basement membrane thickening, endothelial cells and glial cells for vascular occlusion in diabetic retinopathy were studied by immunohistochemical technique.
Methods: Immunohistochemistry to type IV collagen (basement membrane marker), von Willebrand factor (endothelial cell marker) and to glial fibrillary acid protein (glial cell marker) was performed on serial sections through localized areas of vascular occlusion in twelve eyes obtained post mortem from eight diabetic patients. The localized areas of vascular occlusion were identified on the basis of casts of the retinal vascular system.
Results: In all areas of vascular occlusion the ghosts of occluded vessels showed immunoreactivity to glial fibrillary acid protein centrally corresponding to the former lumen. The former walls of ghost vessels showed normal or decreased immunoreactivity to von Willebrand factor and increased immunoreactivity to type IV collagen. However, the material accumulated centrally to occlude the former lumen did not display any immunoreactivity to these compounds.
Conclusion: Glial cells, but not endothelial cells or basement membrane thickening, are involved in occluding the central retinal vascular lumen in diabetic retinopathy.

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TITLE: "VITREOUS HEMORRHAGE IN THE DIABETIC PATIENT: EVALUATION OF THE ECOGRAPHIC SEMIOLOGY IN IT'S EVOLUTIVE CLINICAL PROGNOSTIC".

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PURPOSE: The ecography constitutes a diagnostic exploration fundamental for the demonstration of the nature and extension of alterations in diabetic patients with vitreous hemorrhage. In the present work we have tried to demonstrate it's role in the establishment of the evolutive clinical prognostic.

METHODS: Ultrasounds in mode B were used to evaluate the ecographic findings which were discovered in 297 eyes with diabetic retinopathy and vitreous hemorrhage. At the same time, we compared the data of the initial ecographic exploration with the clinical evolution in 204 of these cases using a model of multiple logistic regression.

RESULTS: The ecographic findings of the worst prognostic over the clinical evolution are: the discovery of neovascular or not membranes of retinal proliferation as in the evidence of tractioned vitreoretinal adherences.

CONCLUSIONS: The factors of higher influence over the evolutive probabilities in one or the other sense in a diabetic vitreous hemorrhage are the ecographic parameters of prehyaloid hemorrhage and retinal detachment together with the time of evolution of the hemorrhage and the verification of having undergone treatments of previous panretinophotocoagulation.