Comparison of Retinal Thickness in Diabetic Patients without Retinopathy and Classification of Diabetic Retinopathy by Slit Lamp

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***Abstract*—**The retina is the neurosensorial tissue of the eye and is extremely rich in polyunsaturated lipid membranes. This feature makes it especially sensitive to oxygen and/or nitrogen activated species and lipid peroxidation. Several authors have postulated the importance of superoxide and peroxynitrite production in the development of diabetic complications. Analyze the validity of the non-mydriatic camera for diabetic retinopathy and the severity levels which referred to the ophthalmologist. In the present study, we have used two different antioxidants, that present as a common feature their peroxynitrite scavenging capacity, to ameliorate the oxidative stress that exists in the retina in diabetic patients. Analyze indicators of severity of retinopathy. Diabetic they can help increase the sensitivity of screening. Were used as evaluation criterion validity indicators, predictive values, indicators of clinical utility, and consistency. Hyperglycemia was accomplished by the intraperitoneal injection of Alloxan in a mouse model of diabetic retinopathy. Hyperglycemia was accomplished by the intraperitoneal injection of Alloxan in a mouse model of diabetic retinopathy

***Index Terms*—** Diabetes mellitus; retinopathy; neuropathy Blurring, Blurry vision.

I. INTRODUCTION

Diabetic patient 's eye may be affected by various pathological processes in which the most important is diabetic retinopathy. Diabetes is the leading cause of vision loss, being 25 times more common blindness in diabetics than in controls. Diabetic retinopathy usually has a progressive course, although in its early stages certain lesions may subside spontaneously. Non-proliferative retinopathy is the appearance of microangioaneurismas small intraretinal hemorrhages and exudates. In this first phase the reduction in visual acuity may be mild or absent, except when macular edema or exudates and hemorrhages appear appear in this location. In diabetic retinopathy, capillaries exhibit thickening of basement membrane, microaneurysms, degeneration of pericytes, poor perfusion and obstruction. There is also increased vascular permeability, with leakage from microaneurysms local or generalized, giving rise to hard exudates. Most common retinal edema may occur in the posterior pole and macula region is also the site where exudates predominate. If retinopathy progresses, there obliteration of vessels with large patches of acellular capillaries originating from terminal arterioles occlusion, appearance of nests of microaneurysms and tortuous vessels are called intraretinal microvascular abnormalities.

The diabetic compromises the vision essentially with two retinal pictures: diabetic maculopaty and the complications of the proliferative diabetic retinopathy (retina and optic disc neovascularization). The management of the retinopathy is: metabolic control, ophthalmologist control, risk situations control (HTA, pregnancy, etc.). The photocoagulation, panphotocoagulation and the vitrectomy are invasive treatment that allows the improvement of the prognostic diabetic retinopathy and reduce the blindness risk. Diabetic retinopathy, diabetic maculopaty, blindness in diabetics.

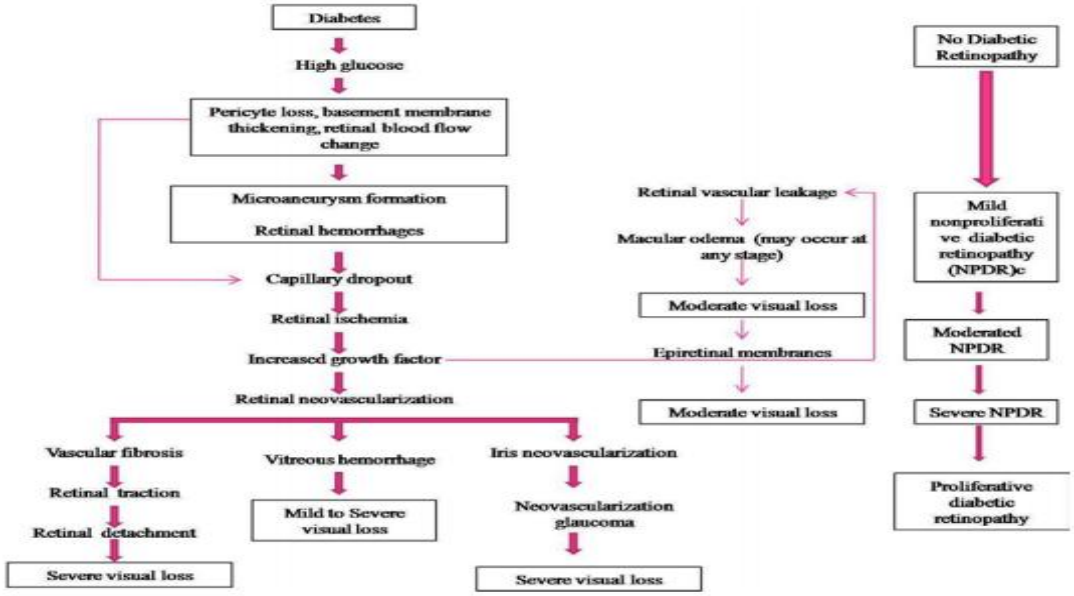

Fig 1 Stages & Symptoms of Diabetic Retinopathy

Diabetic retinopathy is essentially retinal microangiopathy. Therefore, it is necessary to understand the structure of the vascular and neural elements that constitute it. The retina is a neural tissue, whose operation requires an isolated microenvironment preserved as much or more than the brain. Therefore the Blood-Retinal Barrier is narrower than the blood-junctions. Tight junctions between endothelial cells were separately kept airtight and retinal tissue surrounding the vessel, the intravascular space. The endothelial cells surrounding basement membrane and pericytes, which give stability to the structure.

Hyperglycemia and its sequelae produce cellular changes that alter the tight junctions between endothelial cells and spaces which allow the leakage of plasma into the retina. This causes edema, which may be subclinical long, except that involves the macula, in which case a decrease in visual acuity occurs. Filtration is difficult to assess when the ophthalmoscope is not very marked. To confirm a thorough slit lamp examination is required at the slit lamp.

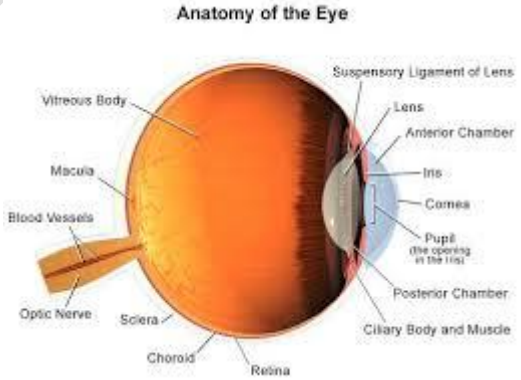

Fig 1.1 Anatomy of the Eye

When the barrier is more deteriorated and the intercellular spaces are wider, can extravasate, which are expanded by the retinal tissue, placing lipids at the edge of the infiltrated zone. The interaction between the joints as exudate from the layer of the retina causes serious functional consequences. It may therefore be necessary to stop the leakage laser, especially if the exudate, expanding, threat involving the fovea.

II. OPTIC DISC AND MACULA DETECTION METHODS

Diabetes mellitus is a disorder of carbohydrate metabolism of proteins and lipids caused by a relative or absolute deficiency of insulin. The Diabetic Retinopathy (DR) is the leading cause of blindness in the age between 20 and 64 years, and is the most frequent and the most important complication of diabetes mellitus. Diabetic retinopathy is common in both types of diabetes, but patients with IDDM go on to have a higher risk of complications. The nonproliferative shows microaneurysms, small hemorrhages, hard exudates, and intraretinal microvascular abnormalities. As the disease progresses macular edema can occur, ie, the presence of fluid within the retinal cells, which represents the major cause of decreased visual acuity in the non- proliferative DR. The evolution of the edema can vary, sometimes is stable, while in other cases it aggravates slowly giving rise to the formation of true eproprie cysts in the retina. Macular edema is connected to the presence of alterations in the parietal and blood With the increase of retinal, the patient may develop an RD proliferating.

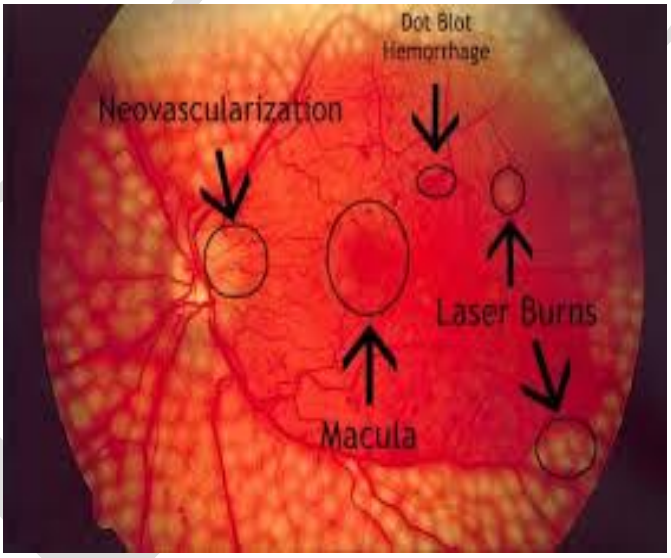

Fig 2.1 Background diabetic retinopathy

The R.D. including all forms of proliferative R.D. in which there is a vascular proliferation. The R.D. proliferating is more frequent in young patients with IDDM where control is more difficult. It may be an evolutionary stage of R.D. nonproliferative or occur early in the formation of newly formed vessels. These are formed by capillaries that have a wall very fragile and can break easily, causing a vitreous hemorrhage. The bleeding can be reabsorbed, but if nothing is done with an appropriate laser therapy, tend to recur, and in such cases, their consumption is increasingly difficult. The following may appear a reaction with formation of fibrovascular membranes that can penetrate into the vitreous, causing a retinal tear and detachment of the retina. In more severe cases, there may be the onset of a neovascular.

III. METHODOLOGY

Vascular endothelial deterioration progresses and the inner walls of the vessels are beset with leukocytes and erythrocytes rigid damaged due to the of the proteins of the membrane. By a combination of these causes capillary occlusions or closures occur, probably derived from, retina areas left without irrigation. These areas are the small first, and then can be extended by preventing blood circulation in ever larger areas. The hypoxia retina suffers no irrigation, the main stimulus for the synthesis of growth factors that induce proliferation of new vessels. The tissue hypoxia can only clinically evident if seen cottony exudates or capillary closure is very close to the fovea and visual acuity reduced sharply.

Cotton wool or soft is an accumulation of axonal flow ganglion cells are altered operation with hypoxia, seen as white patches of fuzzy edges and abiding sense of axons in the fiber layer. Usually transient and disappear without visible consequences. When a tissue suffering from hypoxia, increases the synthesis of mediators communicate form factors as lack of oxygen and the need for increased blood flow through the vascular dilation or the formation of new vessels from impersonating not meeting its function. In this process the diabetic eye is devastating. The new vessels grow, stimulated by hypoxia in the retina, then on the surface and finally, they grow into the vitreous cavity at the posterior hyaloid adhering. Have very poor vessel wall producing increasingly larger filtration, more edema, increased flow and small or massive hemorrhages which enable the arrival of fibroblasts within the eye. Vessels can be seen with the ophthalmoscope located in the papillary or extradiscal disc out of the disc. Alteration of the caliber of the veins as segmental and sequential. It is a reflection of the increased flow and loss of self-regulation of vascular tone. It is one of the most valuable signs to predict progression to proliferative retinopathy. Capillaries are abnormal route, which are located in the retina, tortuous, dilated and irregular caliber, which is probably formed from shunts between pre-existing vessels and as an effort to provide greater blood flow. It is the most frequent complication dramatic proliferative retinopathy. They may be minor and superficial, or be massive and occupy all the vitreous. They may resolve spontaneously. The presence of blood in the vitreous may be the prologue to the development of fibrosis and retinal traction.

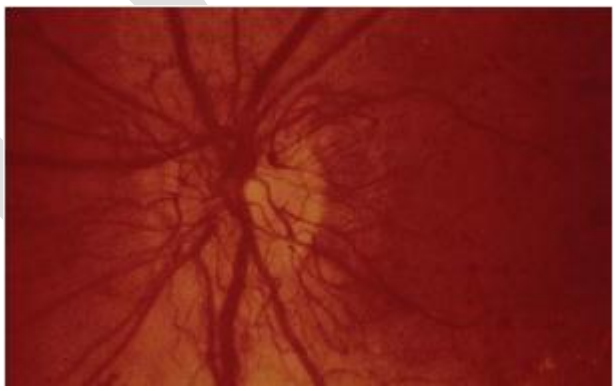

Fig 3.1 Proliferative diabetic retinopathy. Papillary Neovascularization.

The presence of blood in the vitreous with all over the fibrin formed elements and plasma mediators, complete with a common healing process, within the eye that has tragic consequences. The vitreous humor, a gel consisting of different types of collagen, is mixed and serves to support the aforementioned elements of the blood and within days to a fiber unit is formed begins to retract. The vitreous is attached to the retina in loosely and most of its surface, but very strongly in some sectors such as disc, macula , the route of the great vessels and peripheral retina (base body vitreous). Significant reductions vitreous during scar

retraction is apparent, but remains attached to these places. Then, the shrinking process continues and, since both the papilla as the errata are impossible to detach, detachment occurs mostly in the area of the macula, which explains the reason for severe visual loss.

IV. RESULT

The most effective treatment of diabetic retinopathy, especially in the early stages, is the laser. This technique uses a narrow beam of light energy to heal damaged blood vessels. The small scars caused by the laser blocking the outflow of blood from the capillaries of the retina. It's a treatment that does not require surgery and can be performed on an outpatient basis. In the early forms of diabetic retinopathy, laser coagulation can stop the progression of the disease; in advanced retinopathy, it can still limit the damage even if you can not perfectly control the evolution of the disease. In the case where the vitreous is clouded by the presence of blood must be used another type of treatment: the vitrectomy, which is a surgical procedure that allows the removal of the blood and severely limit the evolution of the disease.

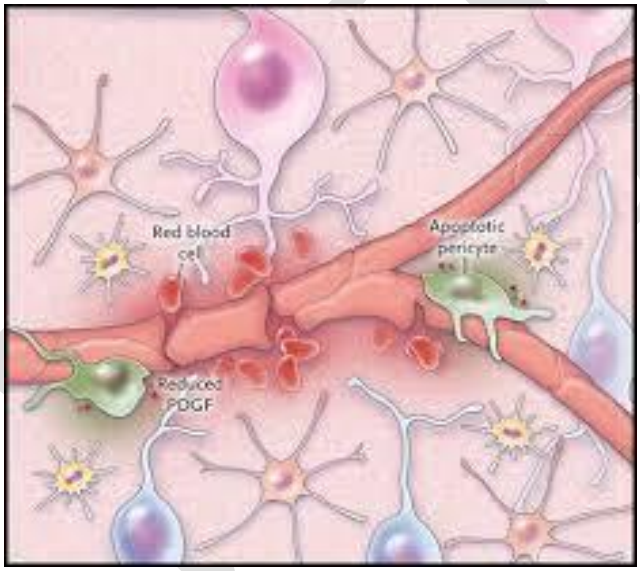

Fig 4.1 The incidence of diabetes is increasing, but that of diabetic retinopathy is falling,

The worst complication of diabetic retinopathy is the transitional retinal detachment, which can cause serious vision loss to blindness; also in this case the vitrectomy is the only effective treatment. Once diabetic retinopathy is diagnosed, the ophthalmologist takes into account factors such as the patient's age and the degree of damage suffered by the retina, before deciding whether to treat immediately with the laser or perform periodic checks only. In many cases, a therapy is not required; in other cases it is recommended to stop the progression of retinopathy and sometimes also to improve the view. The successful treatment of diabetic retinopathy depends not only on the early diagnosis and therapy prescribed by an ophthalmologist, but also by the attitude of the patient and the care he has of himself. In fact, the antihyperglycemic therapy and diet are the remedies necessary to preserve the good health of people with diabetes; physical activity is useful for the initial retinopathy, may instead aggravate bleeding in proliferative diabetic retinopathy.

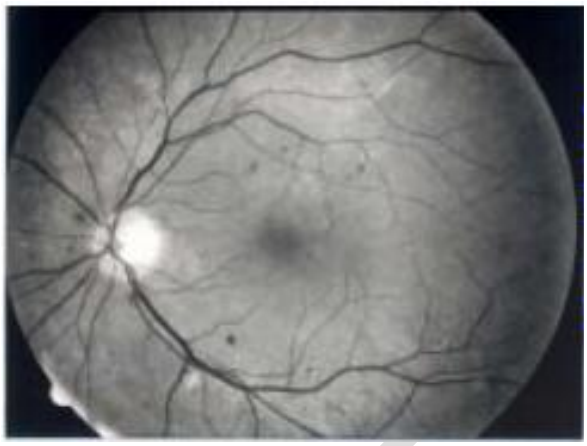

Fig 4.2 Binocular indirect ophthalmoscopy under mydriasis.

An early diagnosis of diabetic retinopathy is the best way to prevent vision loss. Patients with diabetes should schedule an eye exam at least once a year, even when no symptoms. After that diabetic retinopathy has been diagnosed with eye exams should be more frequent in order to start treatment before vision is affected.

V. DISCUSSION AND CONCLUSION

The need to provide a framework for improved communications and transfer of information among the primary care physician, endocrinologist, ophthalmologist, and other eye care providers was a major impetus to develop simplified clinical disease severity scales that could be used internally Perhaps the most important groups in the classification system are those indicating that a patient is at risk for vision loss from diabetic retinopathy. The levels of grading of retinopathy The Diabetic retinopathy are a complication eye of diabetes that is caused by the deterioration of blood vessels that supply the retina. Damage to the blood vessels of the retina may result in them to undergo fluid leakage or blood. If the disease progresses, new blood vessels are formed and the fibrous tissue proliferation in the retina, which has the effect of deteriorating vision since the image sent to the brain becomes blurred. It is possible that in the beginning not evidencing symptoms, pain or loss of vision, but as the disease progresses, severe illness, occur as macular edema and other complications that lead to a major loss of vision.

Increased permeability of the capillaries in the retina. The result is the output of fluid inside the vessels and the formation of deposits in the retina that are called exudates. Proliferation of new vessels and fibrous tissue. The body tries to compensate for the deficiency of oxygen to form new blood vessels, but these new vessels are fragile, easily broken and lead to further complications. Contraction of fibrous tissue, intraocular hemorrhage and retinal detachment due to traction. This is the last phase of the disease that can lead to a significant loss of vision. Furthermore, new vessels grow in other parts of the eye such as the anterior chamber (rubeosis iridis) and block the flow of aqueous humor which leads to a final complication, neovascular glaucoma.

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