

Pathogenic Mechanism of Diabetic Retinopathy

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Abstract Diabetic retinopathy is the leading cause of blindness in industrialized countries. Pathogenetic changes in retinal microvessel structure as diabetic retinopathy is a major cause of structural changes include thickening of the capillary basement membrane, loss of perisit retina, increased vascular permeability, and the formation of capillary microaneurysms. Structural changes were accompanied by a decrease in retinal blood flow, angiogenesis, capillary occlusion, bleeding, formation of tissue fibrosis, and tractional retinal detachment. Some events, or all of the combinations of events, can eventually cause damage to or total loss of sight.

This study gives an overview how the pathogenic mechanisms of diabetic retinopathy.

Keywords: *pathogenetic, diabetic retinopathy, capillary microneurism*

1. Introduction

Patients with diabetes who are treated in a modern have a lower risk of microvascular complications than patients treated traditionally. However, diabetes is the most common cause of blindness. Therefore, there is strong reason to increase our knowledge about the cellular and molecular mechanisms of these complications can be obtained so that a rational strategy for the prevention and treatment Microvascular complications of diabetes including retinopathy, nephropathy, and neuropathy. Due to complications, pathological changes and cellular dysfunction visible on nonvascular tissue at an early stage and can not be explained by changes in the circulatory [1].

Diabetic retinopathy is a microvascular complication of diabetes mellitus that can lead to blindness. Blindness due to diabetic retinopathy is characterized by the emergence of microvascular bleeding in the retina. Early Treatment Diabetic Retinopathy Study Research Group (EDTRS) divides diabetic retinopathy is divided into two forms of non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR). NPDR a clinical reflection of hiperpermeabilitas and incompetence of the blood vessels caused by the blockage and capillary leak. Characteristics of non-prolifatif diabetic retinopathy begins with the bleeding that resulted microaneurysm, hard exudate, cotton wools, microvaskuler intraretinal and venous disorders. NPDR levels are classified into:

1. Mild NPDR is marked by the emergence of a mikroneurisma.
2. Moderate NPDR is marked by the emergence haemoradge and mikroneurisma, soft exudates, venous circuits such as beading and intraretinal microvascular abnormal apparent.
3. Severe NPDR is characterized by the emergence and mikroneurisma haemoradge in 4 quadrants, venous circuit like beads in 2 or more quadrants, intraretinal microvascular abnormal least 1 quadrant.
4. Very Severe NPDR is marked by the appearance of two or more of the signs Severe NPDR.

While the PDR is the most severe stage of NPDR advanced due to leakage of plasma on the wall continues accompanied ischemic retina, PDR levels are classified into:

1. Early PDR (Early PDR) is characterized by the emergence of new blood vessels in the retina, High Risk PDR definition is not found.
2. High-risk PDR (High Risk PDR) is characterized by the emergence of new blood vessels in the retina of optical disc consisting of 1/4 sd 1/3 or more of the area of the disk, some blood vessels and vitreous or preretinal or vitreous hemorrhage [2].

Diabetic retinopathy is the leading cause of blindness in industrialized countries [3].

Pathological changes in retinal microvessel structure and function has been considered as the main cause of diabetic retinopathy [4],[5]. Structural changes include thickening of the capillary basement membrane, increased vascular permeability, loss of perisit retina, and the formation of capillary microaneurysms. Structural changes were accompanied by a decrease in retinal blood flow, capillary occlusion, angiogenesis, bleeding, formation of fibrotic tissue, and tractional retinal detachment. Some events, or all of the combinations of events, can eventually lead to blindness [4].

2. Diabetes Mellitus

The effects of diabetes mellitus include long-term damage, dysfunction, and failure of various organs, especially the eyes, kidneys, heart, and blood vessels. Diabetes can be present with typical symptoms such as thirst, polyuria, blurred vision, weight loss, and polyphagia, and in the most severe form, with ketoacidosis or nonketotic hyperosmolarity, which, in the absence of effective treatment, leading to stupor, coma, and death. Often symptoms are not severe or may not even exist. Hyperglycemia enough to cause functional changes may quite often present pathological until long time before being diagnosed. As a result, diabetes is often found only abnormal results of routine blood or urine glucose tests or due to complications. In some cases of diabetes may not be seen only occasionally, such as, for example, with glucose intolerance in pregnancy or gestational diabetes, which can be affected after parturition. In some individuals the possibility of developing diabetes can be recognized even before any obvious abnormalities of glucose tolerance. During the evolution of type 1 diabetes, for example, immunological disorders such as islet cells or other antibody is present, and this may precede clinically apparent disease by months or even years [6].

3. Microvascular and macrovascular complications of diabetes

Diabetes is a chronic disease characterized by hyperglycemia. Modern medical care is widely used intervention for lifestyle and pharmaceuticals aimed at preventing and controlling hyperglycemia. In addition to ensuring adequate delivery of glucose to tissues, treatment of diabetes attempt to reduce the possibility of body tissue harmed by hyperglycemia.

The importance of protecting the body from excessive hyperglycemia order, the direct and indirect effects on human vascular buildings are a major source of morbidity and mortality in both type 1 and diabetes type 2. In general, the adverse effects of hyperglycemia separated into macrovascular complications (coronary artery disease, illness peripheral arteries, and stroke) and microvascular complications (diabetic nephropathy, neuropathy, and retinopathy) [7].

4. Pathogenesis of Diabetic Retinopathy

The development of diabetic retinopathy begins NPDR, initially triggered by circumstances hyperglycemia thus causing structural changes include thickening of the capillary basement membrane, increased vascular permeability, loss of retinal pericytes, and the formation of capillary microaneurysms. Structural changes were accompanied by a decrease in retinal blood flow, capillary occlusion, angiogenesis, bleeding, formation of fibrotic tissue, and tractional retinal detachment.

Perisit caused by lack of regulation of blood flow that is contractive in the bloodstream. People with diabetes have increased viscosity of the blood flow in capillaries, decreased red blood cells and an increase in platelets [8].

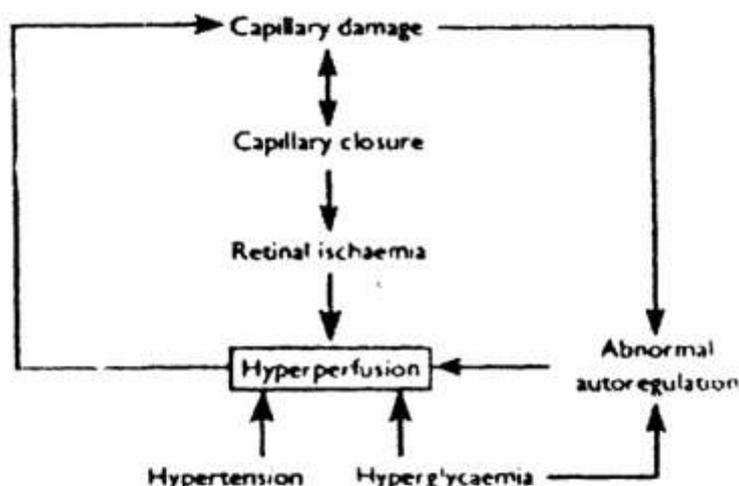


Figure 8. Model pathogenesis of diabetic retinopathy [8].

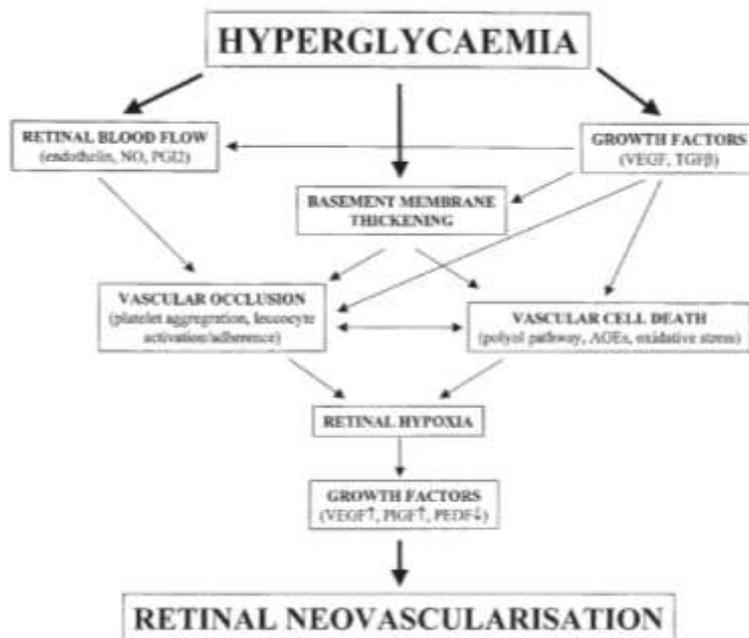


Figure 9. Schematic diagram of the pathogenesis of diabetic retinopathy. Abbreviations: NO = Nitric Oxide, PGI 2 = Prostacylin, VEGF = vascular endothelial growth TGFβ = transforming growth factor beta, advanced glycation endproducts AGEs =, PIGF = placenta growth factor, PEDF = pigment epithelium-derived factor [4].

If the patient is not treated immediately NPDR cause more severe disease. When this phase is already emerging new neovascular, then according EDTRS classified as PDR. Phase could continue to cause total blindness.

5. Conclusion

Based on the study of this paper can be concluded that NPDR occurred beginning with structural changes include thickening of the capillary basement membrane, increased vascular permeability, loss of retinal pericytes, and the formation of capillary microaneurysms. Structural changes were accompanied by a decrease in retinal blood flow, capillary occlusion, angiogenesis, bleeding, formation of fibrotic tissue, and tractional retinal detachment.

PDR is due to the leakage of plasma on the wall continues accompanied ischemic retina, the level of PDR.

6. Suggestions

At the writing of this scientific work needed advice for the future include:

1. The study of the effect on intraretira to diagnose microvascular bleeding heavier.
2. Assessment of the presence of oxygen and nutrients in influencing loss of retinal vessels perisit.

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