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Review Article

A REVIEW ON DIABETIC RETINOPATHY AND ITS MANAGEMENT IN AYURVEDA

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ABSTRACT

Diabetes Mellitus is a common metabolic disorder in which there is high blood sugar level over a prolonged period and occurs in one of two forms: Type1 or IDDM and Type2 or NIDDM. This disease results in generalized macro and microvascular complications linked to glycaemic control and affect the kidneys, eyes and peripheral nerves. The most common and serious eye complication of diabetes is diabetic retinopathy, which may result in poor vision or even blindness[1]. Despite of better understanding of its pathogenesis, satisfactory treatment is yet not available. Ayurveda is well recognized for its role in preventing the disease, but as such no description is available in text which clarifies the progression of prameha to loss of vision. So ayurvedic treatment purely lies on the basis to pacify the pathological changes which occurs in eye as a result of diabetes according to modern parameters. This paper reviews the pathophysiology of diabetic retinopathy with a view to understand therapeutic target and discusses the possible role of ayurveda in its management.

Keywords: Diabetic Retinopathy, Diabetic Mellitus, Exudates, Haemorrhages, Neovascularization, Prameha, Madhumeha.

INTRODUCTION

Diabetic Retinopathy refers to the retinal changes seen in patients with diabetes mellitus. It is the leading cause of blindness and develops frequently in the long standing cases of diabetes mellitus, controlled or uncontrolled. The severity of diabetic retinopathy generally parallels the duration of diseases and the adequacy of its control and not the severity of disease. The longer the patient has diabetes, higher are his or her chances of developing diabetic retinopathy[2]. Ayurveda plays a significant role in the integrated management of this condition. Incidence of diabetic retinopathy depends on following factors:

- Duration of diabetes: It is most important determining factor. Roughly 50% of patients develop diabetic retinopathy after 10 years, 70% after 20 years and 90% after 30 years of onset of disease.
- Sex: Incidence is more in females than males (4:3).
- Poor metabolic control: It is less important than duration, but is nevertheless relevant to the development and progression of diabetic retinopathy.
- The course and severity of diabetic retinopathy are also affected by the presence of nephropathy, systemic hypertension, positive family history of diabetic retinopathy, smoking, obesity and hyperlipidaemia[3].

Diabetic Retinopathy is a microangiopathy which affects the retinal precapillary arterioles, capillaries and venules**.** This microangiopathy causes:

(1) Microvascular leakage (2) Microvascular occlusion.

1. Microvascular leakage

Normally capillaries are lined by single layer of endothelial cells and basement membrane. But in retinal capillaries, they are also lined by Pericytes. These pericytes are responsible for structural integrity of vessel wall. These pericytes are specifically lost early in diabetic retinopathy.[4,5] Physical weakening of capillary walls due to loss of pericyte result in localized saccular outpouching of vessel wall, termed microaneurysm. It appear as a small red spot. Some of the thin walled microaneurysms and fragile retinal capillaries may rupture and cause retinal haemorrhages, results in deep haemorrhages (dot and blot haemorrhages) and superficial haemorrhages (flame shaped). In addition there is breakdown

of blood retinal barrier due to many factors, especially as a result of opening of tight junction between adjacent microvascular endothelial cell processes. Breakdown of blood retinal barrier causes leakage of plasma constituents in the retina and form hard exudates and retinal oedema. Hard exudates are deposits of plasma proteins and lipids. All the lesions often occur more near macula and optic disc.[6]

2. Microvascular occlusion

Due to prolonged diabetes mellitus there occurs thickening of capillary basement membrane, capillary endothelial cell damage and proliferation, changes in R.B.C's (i.e elasticity of R.B.C reduced) and increased stickiness and aggregation of platelets. All together leads to microvascular occlusion which in turn lead to retinal hypoxia, results in retinal ischaemia, which initially develops in the mid retinal periphery. Appearance of ischaemic areas due to occlusion of capillaries may manifest as "cotton wool spots" or soft exudates. These are microinfarct of nerve fibre layer of retina. Venous dilation, beading and looping of the veins occurs secondary to ischaemia.

The two main effects of retinal hypoxia are 1) Arteriovenous shunts 2) Neovascularisation. All these occur in an attempt to revasclularise the hypoxic areas of retina. Formation of arteriovenous shunts from arterioles to venules associated with significant capillary occlusion are reffered as intraretinal microvascular abnormalities (IRMA). Retinal hypoxia leads to release of vasoproliferative substance such as vascular endothelial growth factor (VEGF). It results in development of neovascularisation i.e. proliferation of new vessels from the capillaries in the form of neovascularisation at the optic disc (NVD*)* or elsewhere (NVE*)* in the fundus along the course of major temporal retinal vessel and occasionally on the iris (rubeosis iridis) and angle of anterior chamber (neovascular glaucoma). This neovascular tissue is more fragile, bleed easily and incites a fibroblastic response. These new vessels may proliferate in the plane of retina or spread into the vitreous as vascular fronds. Later on condensation of connective tissue around the new vessels result in formation of fibro vascular epiretinal membrane. Vitreous detachment and vitreous haemorrhage may occur in this stage. Later fibrovascular and gliotic tissue contracts to cause retinal detachment and blindness[7].

Diabetic Retinopathy has been variously classified. Presently followed classification is as follows:

I. Non Proliferative Diabetic Retinopathy(NPDR)
   - Mild NPDR
   - Moderate NPDR
   - Severe NPDR
   - Very severe NPDR

II. Proliferative Diabetic Retinopathy(PDR)

III. Diabetic Maculopathy

IV. Advanced Diabetic eye diseases

PRESENTATION OF DISEASE/CLINICAL FEATURES

There may be no symptoms even though advanced changes are present in the retina. We can say that there is painless progressive diminution of vision. Therefore diabetics should have their fundus examined regularly. Non proliferative or Background diabetic retinopathy is the most common type of diabetic retinopathy. It is characterized by multiple microaneurysms (earliest detectable lesion), venous dilatation, hard exudates, dot and blot and flame shaped haemorrhages and retinal oedema. The microaneurysms appear as multiple, minute, round, red dots. They are usually assosciated with yellow-white waxy –looking exudates with crenated margins (hard exudates). Macular oedema occurs in a large number of eyes and, with central hard exudates is the commonest cause of diminution of vision in diabetic retinopathy. Ischaemic changes superimposed on background diabetic retinopathy produce a preproliferative diabetic retinopathy. The fundus shows intraretinal microvascular abnormalities (IRMA) or shunt vessels, and evidence of ischaemia such as more than 8-10 areas of cotton-wool spots. These changes indicate progression towards the more devastating form of proliferative diabetic retinopathy. Neovascularization of the optic disc (NVD) as well as neovascularization elsewhere (NVE), posterior detachment and collapse of vitreous, vitroretinal fibrovascular bands and vitreous haemorrhages characterize proliferative diabetic retinopathy. Neovascularization is the hallmark of proliferative diabetic retinopathy. The proliferation of fibro-vascular tissue on the surface of the retina and in the vitreous may cause formation of epiretinal membrane and irregular fibrovascular bands, respectively. The contraction of these bands may lead to tractional retinal detachment and blindness.[8]

DIAGNOSIS

- Fundus examination
- Blood sugar

TREATMENT

Medical treatment of diabetic retinopathy is aimed at prevention of retinopathy. Tight glycaemic control is assosciated with reduction in the development of retinopathy. Good metabolic control and proper management of hypertension or other assosciated conditions prevent the progression of diabetic retinopathy. No treatment is required for background diabetic retinopathy with normal visual acuity except periodic annual examination. The only ocular treatment available for macular diabetic retinopathy and proliferative diabetic retinopathy is photocoagulation.[9] Satisfactory treatment is yet not available.

Diabetic retinopathy can be well controlled by ayurvedic treatment as ayurvedic herbs not only reverse the blood clots formed in the retina and vitreous but also strengthen the metabolic function so that further chances of blood leakage can be minimized. Ayurveda controls the disease and increases blood circulation and nourishes retina. Use of ayurvedic therapies disables the disease and possibility of being cured increases. As such Pramehajanya Netraroga (diabetic retinopathy) is not mentioned in ayurvedic text. Symptom wise it is a complication of prameha. The word prameha is derived from Pra-means excess, Meha- ksharana-passing of urine. So prameha is passing excess urine and turbid in colour (Prabhoota avil mootrata)[10]. Twenty types of prameha if ignored and not treated properly in time can convert into madhumeha and become incurable. Diabetes mellitus in ayurveda is known as madhumeha. In madhumeha dosha is kapha pradhan tridosha. Dushya is meda (predominance), mansa, rakta, vasa, majja, lasika, kleda,

shukra, oja[12,13]. In the above it is clear that all the body tissues are vitiated in madhumeha. This is a disease in which all the tissues or organs may be damaged. The disease is not localized in any one organ of the body but may vitiate any of the important organs. From this it may be clear that eyes are also affected by Madhumeha. According to Acharya Charaka and Vagbhata, eye is afraid of kapha dosha.[10,13] So Samanya chikitsa sidhanta in ayurveda for diabetic retinopathy can be considered as follows: (1) Pramehahar chikitsa, (2) Kaphahar chikitsa, (3) Srotorodhhar chikitsa, (4) Urdhavraktapittahar chikitsa. In case of haemorrhages: according to Pratimargharan chikitsa sidhanta virechana is the main shodhan chikitsa[12], urdhavraktapittahar shamana chikitsa, bahya chikitsa includes takradhara, shirolepa or shiropichu with sheeta stambhan aushadi.[13] In case of Sanga (occulsion): Srotorodhhar chikitsa can be done. In case of Macular oedema: Shophahar chikitsa is to be done.

As such no description is present on diabetic retinopathy (prameha updrava) in our texts but it can be treated by following measures:

- Lepa over eyes
- Netra seka or Netra dhara
- Takradhara
- Shirodhara
- Netra tarpana(if no active bleeding)
- Shiro lepa or pichu with sheet stambhan aushadhi
- Putpaaka
- Oral medicines e.g. Saptamrita lauh, Triphla ghrita, Mahatriphla ghrita, Patoladi ghrita, Jivantyadighrita, Triphla churan, Shatavari churan, Rasayan chikitsa.[16,17,18]

The disease cannot be cured 100% but can keep stable in that condition. Many patients suffering from diabetic retinopathy become blind by other means of treatment whereas ayurveda controls this disease and increases blood circulation and nourishes retina.

CONCLUSION

Diabetic Retinopathy is a microangiopathy involving the retinal precapillaries arterioles, capillaries and postcapillaries venules. It is the leading cause of blindness in elderly subjects. As no satisfactory treatment is available for diabetic retinopathy, new approaches are needed to slow the progression and limit the damage caused by this disease. Ayurveda can play significant role in the integrated management of this condition. The disease cannot be cured100% but can keep stable in that condition. Ayurvedic drugs and therapy controls the disease and increases blood circulation and nourishes retina.

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