A Review of Microvascular Complications

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ABSTRACT

Complications of Diabetes mellitus include chronic damage, dysfunction and disappointment of various organs mainly affecting the eyes, kidneys, heart and blood vessels. Various minerals and vitamins acts as cofactors in regulation of insulin secretion and action, among which magnesium plays a key role. Magnesium is an essential mineral and plays a major role in carbohydrate metabolism. It acts as a co-factor in glucose transport mechanism and for various intracellular enzymes involved in carbohydrate oxidation. Deficiency of magnesium results in increase in insulin resistance and decrease in glucose uptake of the cells in the body. Serum magnesium levels in healthy individuals are constant, but 25 to 39% people with Type 2DM have low levels of magnesium. Hypomagnesemia has been found to have deleterious effect on glucose homeostasis and insulin sensitivity in Type 2DM. Low levels of magnesium have also been attributed to the progression and development of micro and macrovascular difficulties in Type 2DM.

INTRODUCTION

In this segment represents introduction of this research work. Chronic hyperglycemia along with existence of other factors like hypertension and dyslipidemia predisposes to the origin of various complications causing metabolic derangements, producing advanced glycation end crops (AGE) and abnormal activation of signaling cascades such as Protein kinase C, increased hexosamine pathway flux, increased polyol pathway (all these integrated by increased production of ROS in mitochondria) leading to damage of blood vessels [1, 2]. Microvascular complications in Type 2 DM include diabetic retinopathy, diabetic neuropathy and diabetic nephropathy. [3]

Diabetic retinopathy is the primary cause of visual impairment worldwide. Prevalence of diabetic retinopathy increases with poor glycemic control and longer duration of Type 2 DM. The degree of severity of retinopathy varies from non-proliferative and pre-proliferative to more severely proliferative diabetic retinopathy. [4]

Diabetic retinopathy is characterized histologically by the presence of basement membrane thickening, loss of endothelial tight-junctions, loss of pericytes along with increased vascular permeability, capillary occlusions and formation of microaneurysms. [5] Neovascularization is the hallmark of Proliferative diabetic retinopathy. It usually occurs near the optic disc termed as neovascularization of the disc (NVD) or within 3 disc diameters of the major retinal vessels which is known as neovascularization elsewhere (NVE). Progression of PDR may lead to tractional retinal detachment (with or without involving macula). [6]

In these articles represents sector 2 of these articles explains the feature on the related works. In section 3 presents the materials and methods adopted and
RELATED WORKS

In this segment represents focuses the related works of this research work. Diabetes mellitus refers to metabolic disorders associated with chronic hyperglycemia with changes in carbohydrate, protein and fat metabolism which results from defects occurring in insulin secretion or insulin action or both. It is the commonest metabolic disorder in the world and the leading cause of death and disability. Patients should be subjected to examination of power of the muscles, sensory system examination and vibration sense. Sensory system is examined on all the four limbs individually. Vibration is examined using 128Hz tuning fork. Touch is examined using monofilament.

Autonomic functions in diabetes mellitus patients are monitored by recording the changes noted in blood pressure and heart rate in response to special maneuvers like thermoregulatory sweat test, head up tilt test and valsalva maneuvers.

Diabetic neuropathy is diagnosed by ruling out other causes in patients who present with neuropathic symptoms and high blood sugars. Confocal microscopy is the new modality which has gained more importance in recent times for the diagnosis of diabetic neuropathy. In confocal microscopy – Bowman's layer of the cornea is screened for its length, nerve fibre density, and branch density. These factors markedly alters according to the degree of severity of the diabetic neuropathy.

It is one of the common causes of chronic kidney disease worldwide. It is characterized by presence of diabetic nephropathy.

MATERIALS AND METHODS

In this segment represents the materials and methods of this research work. Patients with Type 2DM either on oral hypoglycemic mediators or insulin treatment presenting to outpatient department and self-confessed in the general medicine department in Chennai. (Figure 1, Table 1)

1. 105 patients with Type 2DM were assessed
2. An informed consent was obtained from the patient.
3. Estimation of serum magnesium was done by methylthymol blue method

4. The normal range for serum magnesium used in this study was 1.8-2.6 mg/dl

Study Design

1. Prospective Observational Study

Inclusion Criteria

1. All Patient with Type 2 DM presenting to department of general medicine.
2. Age above 18 yrs.

Diabetic retinopathy

Diabetic retinopathy is the primary motive of visual impairment worldwide. Prevalence of diabetic retinopathy increases with negative glycemic manipulates and longer length of Type 2 DM. The degree of severity of retinopathy varies from non-proliferative and pre-proliferative to extra seriously proliferative diabetic retinopathy.

Diabetic retinopathy is characterized histologically via the presence of basement membrane thickening, loss of endothelial tight-junctions, lack of pericytes together with multiplied vascular permeability, capillary occlusions and formation of microaneurysms. Diabetic retinopathy is mainly classified into Non-proliferative diabetic retinopathy(NPDR) Proliferative diabetic retinopathy(PDR).

NPDR

The lesions encompass micro-aneurysms, minimum 'dot and blot' haemorrhages, splinter haemorrhages, intraretinal microvascular abnormalities and cotton wool exudates inside the retina.
Table 1: Diagnosis of Type 2 DM

<table>
<thead>
<tr>
<th></th>
<th>Normal Glucose Tolerance</th>
<th>Pre diabetes Impaired Glucose Tolerance</th>
<th>Diabetes mellitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting Blood Sugar</td>
<td>&lt;100 mg/dl</td>
<td>100-125 mg/dl</td>
<td>≥126 mg/dl</td>
</tr>
<tr>
<td>Post-Prandial Blood Sugar</td>
<td>&lt;140 mg/dl</td>
<td>140-199 mg/dl</td>
<td>≥200 mg/dl</td>
</tr>
<tr>
<td>HbA1C</td>
<td>&lt;5.6%</td>
<td>5.7-6.4%</td>
<td>≥6.5%</td>
</tr>
</tbody>
</table>

Presence of 1 or greater microaneurysm along side dot, blot or flame-shaped haemorrhages in each of the 4 quadrants inside the fundus.

**Moderate NPDR**

Presence of retinal aneurysms and severe degree of dot and blot haemorrhages, in a single to 3 quadrants. Mild degree of cotton wool spots, changes in venous caliber in shape of venous beading, and microvascular abnormalities in the retina are present.

**Severe NPDR**

Any one of the following need to be present,

1. Microaneurysms and marked hemorrhage in all 4 quadrants of the fundus
2. Marked venous beading in quadrants
3. Excessive intraretinal microvascular leisons in atleast one quadrant.
4. Very Severe NPDR
5. Two or extra of the findings covered in excessive NPDR however no longer proliferative retinopathy

**Proliferative diabetic retinopathy (PDR)**
Neovascularization is the hallmark of Proliferative diabetic retinopathy. It usually occurs close to the optic disc termed as neovascularization of the disc (NVD) or within 3 disc diameters of the important retinal vessels which is called neovascularization elsewhere –NVE). Progression of PDR may cause tractional retinal detachment (without or with concerning macula).

**CONCLUSION**

Finally this work concludes, Various factors like metabolic and hemodynamic alterations mainly activation of Renin-angiotensin system stimulates cell signaling cascades leading to infiltration of mononuclear cells in to the kidney and activation of macrophages. Activated macrophages produces inflammatory cytokines, cellular injury, fibrosis and reduction of GFR. Podocyte loss, loss of function of endothelial cells, thickening of glomerular basement membrane and tubular injury leads to loss of proteins leading to development and progression of diabetic nephropathy 24 hours urinary protein level and kidney biopsy are the hallmark investigations used for diagnosis of diabetic nephropathy. Recent studies have suggested that urine advertisement albumin cretonne ratio and urine spot protein keratinize ratio can be used as alternative markers for diagnosis of diabetic nephropathy. In our study, Urine spot PCR is being used as a tool to measure the prevalence of diabetic nephropathy.

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**Conflict of Interest**

Authors declared no conflict of interest.

**REFERENCES**


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