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AN OBSERVATIONAL STUDY OF LIPID PROFILE IN MADHUMEHA PATIENTS AND ITS ASSOCIATION WITH RAKTAGATA VATA

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ABSTRACT

In Ayurveda, Madhumeha and Raktagata vata both diseases are caused by vitiated vata dosha. Acharya Charaka included Abadha meda in dushya of madhumeha. In modern medicine these disorders are k/a life style disorders. Diabetes Mellitus is a metabolic disorder in which disturbances in glucose, fat, protein metabolism. The association of dyslipidaemia and HTN with DM Type-2, increase the risk of cardiovascular complication in Diabetic patients. This study is planned to assess the lipaemic changes and HTN in Dibeitc patients. The data of 30 patients were taken from the previous research work ‘‘The Scientific Study of Dyslipidaemia in DM Type-2 Patients by Dr. Ashok kumar, Deptt. Of Roga Nidana Evum Vikriti Vigyana, Gov. Ayurveda P.G. College and Hospital, Varanasi. Total cholesterol, Triglyceride, LDL-C, HDL-C level, plasma glucose and were studied in serum of dibetic patients. Systolic Blood Pressure and Diastolic Blood Pressure were measured. Out of 30, 40% patients suffered from more than 5 yrs, 60% patients had Diabetes with FBS level above normal range and 86% patients having alteration in normal lipid value, 67% cases had hypertension. In this study, strong positive correlation between chronicity of Diabetes and HTN, Total Cholesterol & Triglyceride. The risk of Cardiovascular complication are high in these patients because strong association of HTN and Dyslipidaemia with Type-2 DM.

KEYWORDS:- *Madhumeha, Diabetes mellitus, Hypertension, Raktagatavata, Dyslipidaemia.*

INTRODUCTION

In Ayurveda, twenty types of prameha are discribed by Acharyas. Madhumeha is one of these which is mainly due to Vata dosha. Madhumeha is also caused by all type of Prameha if they are untreated or ill-treated besides of vata dosha according to Acharya Shushurut and Vagvhata. Madhumeha is clinical entity in which the persion voides mutra having similarity like madhu. Acharya Shushuruta coined term "Kshaudrameha" for madhumeha. Acharya Charak and Shushuruta has mentioned specific color of mutra of madhumehi patient as Pandu varna and Ksaudra rasa respectively. In madhumeha body acquires sweetness. Ojomeha is described by acharya Charaka to denote madhumeha. In this, depletion of Oja through urine takes place by vitiated vata dosha along with the canges in color and taste.[1,2,3,4,5,6] In modern medicine Madhumeha is correlate with a endocrine disorder, Diabetes mellitus.

DM is characterized by hyperglycaemia with disturbances in glucose, protein and fat metabolism which is due to alteration in insulin secretion and function. Insulin resistance and β-cell dysfunction in type-2 Diabetes Mellitus are main factors.

The global prevalence of DM among adults is estimated to be 6.4%, affecting 285 million people in 2010 and 1 out of the 11adult had Diabetes mellitus in the year 2017 and its prevalence like to increase to 7.7% affecting 439 million people by 2030[7] and 592 million people by 2035.[8]

In Ayurveda, Acharya Charaka has described the term Abadha Meda as Dushya (where the pathogenesis takes place) of Prameha.[9] Abadha Meda means abnormal form of Meda Dhatu which may correlate with Dyslipidaemia.

Disorders of lipoprotein metabolism is collectively refered to as dyslipidemias. Dyslipidemias are generally characterized clinically by increased plasma levels of cholesterol, triglycerides or both, variably accompanied by reduced level of HDL cholesterol. The majority of patients with dyslipidemia have some combination of genetic predisposition and environmental contribution like- life style, medical condition. There are a limited number of discrete "nodes" that regulate lipoprotein metabolism. These include; (1) assembly and secretion of triglyceride-rich VLDLs by the liver; (2) lipolysis of triglyceride-rich lipoprotein by LPL; (3) receptor-mediated uptake of apoB-containing lipoproyeins by the liver; (4) cellular cholesterol metabolism in the heoatocyte and the enterocyte; and (5) neutral lipid transfer and phospholipid hydrolysis in the plasma.[10]

Insulin has important role in fat metabolism, it stimulates synthesis of glycogen in liver, and the excess glucose is used to synthesize fatty acids which are export from the liver as lipoproteins in the circulation. These lipoproteins are used by other tissue, including adipocyets as free fatty acids to synthesize triglycerides.

Insulin also inhibits breakdown of fat in adipose tissues by inhibiting the intracellular lipase that hydrolysed Tg to release fatty acids.

From whole body perspective, insulin has a fat sparing effect. Not only does it drives most cells to preferentially oxidise carbohydrate instead of fatty acids for energy, insulin directly stimulates accumulation of fat into adipose tissue.

So in diabetic patient insulin resistance leads to disturbed fat metabolism in which the formed lipoproteins are changed in both quantity and quality. Insulin resistance has been indicated to be associated with high level of VLDL, high concentration of serum triglycerides and low serum HDL. The role of dyslipidemia in macrovascular complications and cardiovascular complications is well established.

There could be significant role of these lipid abnormalities in the causation of HTN. Hypercholesterolemia induced endothelial injury result in superoxide anion production. The resultant excessive degradation of nitric oxide which disrupts the endothelium dependent vasodilatation affects the peripheral vascular resistance[11] due to decreased lumen and elasticity of BV.

Insulin resistance has been shown to be an independent risk factor for IHD. Insulin resistance/ hyperinsulinaemia is one of the processes that cause HTN.[12] The chances of HTN in Diabetics is twice as compaered with non-diabetics.

In Ayurvedic medical science, no disease has described by the name of Uchharaktachapa/ HTN but their symptoms correlate with disease Raktagata vata.

Raktagata vata is described under Vata vyadhi by Acharyas. Acharya Charak in the context of vata vyadhi stated that the nidanas, vitiates only the vata dosha and not the other factors involved in disease like dushya, srotas etc. This vitiated vata dosha when flows with Rakta dhatu, which is in normal state and other factors like raktavaha srotas etc., leads to disease *Raktagata vata* and produce fallowing symptoms ; Ruja (pain, headache), Bhrama(Giddiness)

Spandana(Palpitation) Santapa (irritation), krashta (Weakness), stambha (Stiffiness) sparshanash(Numbness) paad daha(Burnning in feet), raaga/vivarnata (Discoloration) shotha (Oedema), klama (Tiredness without exertion). Ayurvedic scholers correlate this disease with Hypertension.[13,14,15,16,17]

The clustering of risk factors (HTN, Dyslipidaemia, Hyperinsulinaemia, Insulin resistance) in diabetic patients ultimately results in CVD and reduces life expectancy and increase morbidity and mortility in these patients.

The purpose of this study to detect lipid abnormalities and HTN associated with hyperglycaemia (DM Type-2).

AIM AND OBJECTIVES

1. To observe and evaluate the relationship between diabetes and lipid profile
2. To observe the incidences of HTN in diabetes patients.
3. To evaluate the relationship between dyslipidaemia, HTN and diabetes.

MATERIAL AND METHODS

In this study, the information of 30 patients were taken from the previous research work ‘‘ The Scientific Study Of Dyslipidaemia in DM Type-2 Patient By Dr. Ashok Kumar,2020, Roga Nidana evam Vikiriti Vigyana Deppartment, Government PG Ayurveda College and Hospital, Varanasi.

Diabetes was defined as fasting plasma glucose >125mg/dl or postprandial plasma glucose (2-hr plasma glucose) >200mg/dl. Dyslipidaemia was defined as Tg >150mg/dl, LDL >100mg/dl, HDL< 40mg/dl in males and in females <50 mg/dl, Toatal Chol. >200mg/dl. HTN was defined as SBP >140mmHg and DBP >90mmHg.

The fasting serum sample was taken for lipid profile and fasting glucose estimation and 2-hr after eating serum sample was taken for pp glucose estimation. HDL was determined by-*modified polyvinylsulfonic acid (PVS) and polyethyleneglycol-methyl ether (PEGME) coupled classic method*. Total chol. Was calculated by *dynamic extended stability CHOD-PAP method (with LCF),* Triglyceride was calculated by *dynamic extended stability with lipid clearing agent gpo- trinder method and* LDL is calculated. Fasting plasma glucose was determined by *Glucose Oxidase* method.

The obtained data were statistically presented in percentage(%).

OBSERVATION AND RESULT

- 13.3% patients were having chronicity of more than 10 years and 26.6% patients were having chronicity of between 5 to 10 years. 50% patients had chronicity between 1 to 5 years and 10% ptients were having chronicity of less than 1 year of Diabetes mellitus type-2.
- Out of 30 patients, 10%patients were having FBS <170mg/dl, 30%patients were having 170-200mg/dl, 53.3% patients were having 200-250mg/dl and 6.6% patients were having >250mg/dl.
- Max. no. of patients i.e. 36.6% shows serum chol. between 200-250mg/dl, max. 53.3% patients shows serum Tg between 200-499mg/dl, max.46.6% patients having HDL-C <35mg/dl and max. no. of patients i.e .43.3% patients having LDL-C >160mg/dl.
- 67% patients had SBP >140 mmHg and DBP >90 mmHg and 10% had high normal SBP and DBP value.

DISCUSSION

In our study the prevalences of dyslipidaemia in diabetic patients is 86% and prevalences of HTN is 67%. Most of the patients show combined form of dyslipidaemia. Diabetic patients who suffered from HTN mostly have High TG + Low HDL + High LDL. Second most common pattern of dyslipidemia in hypertensive patients is high TG + Low HDL and High LDL+ High total cholesterol. The most common abnormality is hypertriglyceridemia in these patients.

Disturbance in Total Cholesterol is more common in chronic patients and uncontrolled case of DM-2 while disturbances in Tg level in newly patients are seen. Reduced HDL-C and elevated LDL-C are seen in those patients who poorly controlled sugar level.

The quantitative changes in lipid profile is due to increased availability of glucose for VLDL synthesis and decrease in lipoprotein lipase to clear VLDL from circulation, results in the elevation of Tg. In diabetic patients, hepatic lipase activity is accelerated which is lead to fall in HDL level.[18]

The cases of hypertension with dyslipidemia in diabetics is increase with chronicity of diabetes. All diabetics with dyslipidemia donot have HTN. The percentage of HTN in those

patients who suffered from diabetes < 5 years is low when compare to patients who suffered from diabetes > 5 years.

The age and sex of the patients did not have much influence on serum lipid profile. The duration of diabetes and severity of diabetes had marked influence on lipid levels and HTN. HTN in turn can impair the glucose metabolism through various mechanisms. The exaggerated action of angiotensin II, inhibits insulin like growth factor-1 signaling pathway which in turn hampers the vasodilator and glucose transporting actions of IGF-1 and insulin. Inhibited IGF-1 and insulin can accentuated the vasoconstriction by diminishing endothelial nitric oxide synthase activity, impaired nitric oxide metabolism as well as the sodium pump functioning.[19] Thus DM and HTN worsen each other.

CONCLUSION

Dyslipidaemia is highly prevalent in diabetes and in particularly those with poorly controlled diabetes. In all these cases association of Co-morbid condition, Dyslipidaemia and HTN with Type-2 DM suggest that these patients may be at higher risk of developing cardiovascular complicatons. Hence the regular checkup should be done regularly in all patients with diabetes and all patients should be treated adequately with drugs, dietary and life style modification to achieve normal health. Further study should be done to evaluate the effect of other factors like Mansika Bhava, Aharaj and Viharaj factors, Deha prakriti on HTN and lipid profile in diabetic cases.

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