IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

A review On complications and Management of DIABETIC MALITUS

Anjali B. Bargal 1, Jasvanti B. Patil 1 , Mayuri K. Lonare2 and Deepak S. Musmade3

1.Department of Nandkumar Shinde collage of pharmacy vaijapur, Maharashtra, 423701, India(MS).

2.Department of Quality Assurance, Pravara Rural College of Pharmacy(For Women) chincholi ,Nashik,A/P-Sinnar,Dist-Nashik 422103, India(MS).

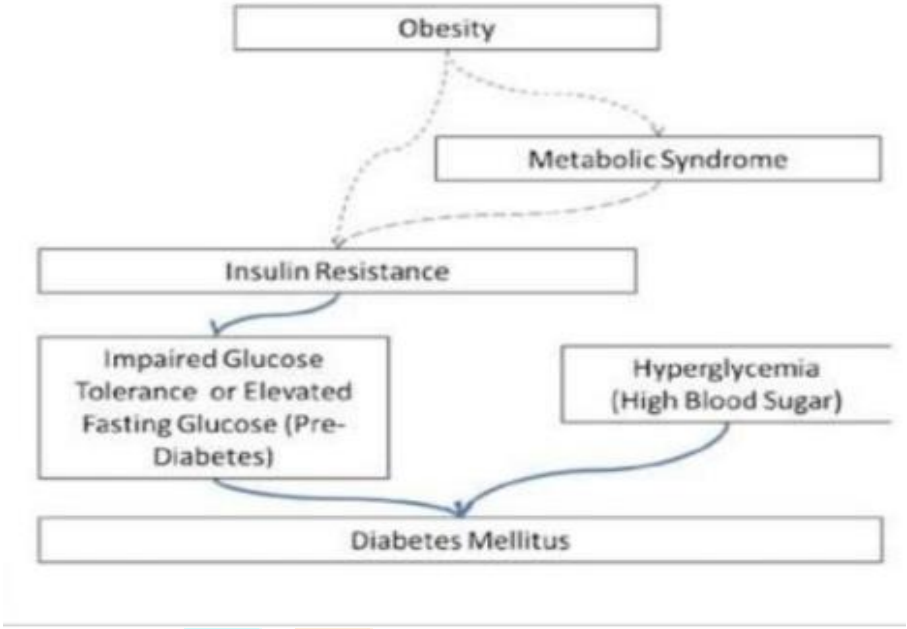
3. Department of pharmaceutical chemistry Nandkumar Shinde collage of pharmacy vaijapur, Maharashtra, 423701, India(MS).

ABSTRACT:-

Diabetes mellitus (DM), belongs to the class of metabolic diseases which the main symptom associated with this disease is the high sugar levels in blood for a long period. It can be categorized to the world's major diseases considering that affects high population in earth and presents two main types I and II.Diabetic is a chronic disease which affects global population from long time. A widespread pathological change is thickening of capillary basement membrane, increase in vessel wall matrix and cellular proliferation resulting in vascular complications like lumen narrowing, early atherosclerosis, sclerosis of glomerular capillaries, retinopathy, neuropathy and peripheral vascular insufficiency.This review is an update on unknown complications, causes and management of this disease and also provides summary on diabetic malitus disease through various strategie

KEYWORDS:- Diabetic malitus, T1DM,T2DM,DM

Introduction:-



Diabetes mellitus (DM) is one of the very oldest diseases and was mentioned three thousand years ago in Egyptian literature.[1]The terms "Diabetes" and "Mellitus" are derived from Greek language. "Diabetes" denotes "a passer through, a siphon" whereas the "Mellitus"means "sweet" It is believed that Greeks entitled it such way, due to the exaggerated urine proportions produced by diabetic patients which attracted flies and bees[2,3,7]. Around 1500 B.C the physicians in India noticed the sweetness of urine of the diabetic people and called it as"Madhumeha". Ebers papyrus, the oldest literature was written around the same time by Egyptians and it was also the first document that describes a condition of frequent emptying of urine.[4,5]. Prolonged hyperglycemia leads to a complex disorder called diabetes mellitus which is associated with loss of glucose through urine and formation of harmful compounds known as keton bodies. Diabetic patients are successfully treated with insulin therapy [6].Classification of diabetes mellitus is based on its aetiology and clinical presentation. As such, there are four types or classes of diabetes mellitus viz; type 1 diabetes, type 2 diabetes, gestational diabetes, and other specific types[7]. Hyperglycemic state (diabetes mellitus) arises when the blood glucose (sugar) levels are higher than 180 mg/dl (10 mmol/l) [14].

CLASSIFICATION OF DIABETIC MALITUS:-

Diabetic malitus is most comman metabolic endocrine disorder of pancreas.it leads to increase in blood glucose levels. In children, such a condition is called insulin dependent diabetic malitus /Type I (IDDM) and the other form of diabetic is non insulin dependent diabetic malitus/ Type II (NIDDM). It is caused due to failure to facilitate movement of glucose into the cell.reduced sensitivity of insulin is called insulin resistance[8]. gestational DM which is diagnosed in 2nd or 3rd semester of pregnancy. Type I DM cannot be avoided Type II can be avoided with good health, exercises and healthy diet.In Type II DM complications occur in several body parts heart,nerves, kidney, eyes and so on[9]. Diabetic Malitus are categorised into following types are as follows:

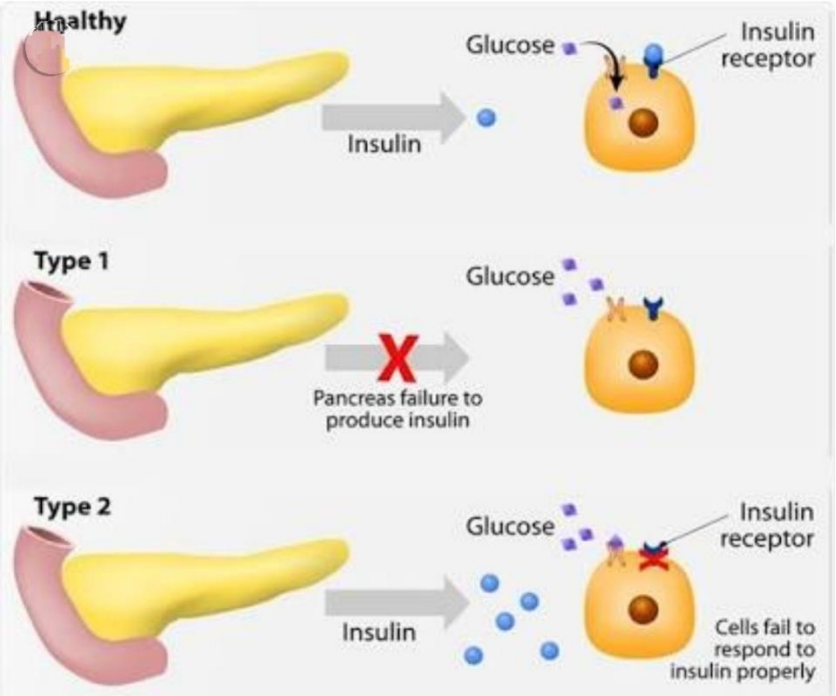
TYPE I DIABETIC MALITUS:-

The type 1 diabetes mellitus (T1DM) is a multifactorial autoimmune disease characterized by chronic hyperglycemia and by the development of specificvascular alterations. Autoimmune destruction of β-cell by T-cells, is responsible for T1DM which results in severe insulin depletion. It is also known as juvenile diabetes[10,11]. In this form of diabetes, the rate of beta-cell destruction is quite variable, being rapid in some individuals (mainly new born and children) and slow in others (mainly adults). Some patients, particularly children and adolescents,may present with ketoacidosis as the first manifestation of the disease. Others have modest fasting hyperglycemia that can rapidly change to severe hyperglycemia and/or ketoacidosis in the presence of infection or other stress. Still others, particularly adults,may retain residual b-cell function sufficient to prevent ketoacidosis for many years; such individuals eventually become dependent on insulin for survivaland are at risk for ketoacidosis. At this latter stage of the disease, there is little or no insulin secretion, as manifested by low or undetectable levels of plasma C-peptide. Immune-mediated diabetes commonly occurs in childhood and adolescence, but it can occur at any age, even in the 8th and 9th decades of life[12].

TYPE II DIABETES MALLITUS:-

Type II diabetes is the most typical DM. In this type, the body is capable of producing insulin but becomes so resistant that the insulin is ineffective.By the time, insulin levels could subsequently turned out insufficient[15].The cause of type II diabetes are unknown, some significant risk factors being pointed out. The most significant ones include: excess body weight, physical inactivity and poor nutrition. Other factors which impacted are ethnicity, family history of DM, past history of gestational diabetes and advancing age[16,17]. Noninsulin-dependent diabetes mellitus(NIDDM), maturity onset diabetes mellitus:There is no loss or moderate reduction in β cell mass; insulin in circulation is low, normal or even high, no anti-β-cell antibody is demonstrable; has a high degree of genetic predisposition; generally has a late onset (past middle age). Over 90% cases are type 2 DM. Causes may be:

- Abnormality in gluco-receptor of β cells so that they respond at higher glucose concentration or relative β cell deficiency.
- Reduced sensitivity of peripheral tissues toinsulin: reduction in number of insulin receptors, 'down regulation' of insulin receptors. ManyMany hypertensives are hyperinsulinaemic,but normoglycaemic; exhibit insulin resistance associated with dyslipidaemia (metabolic syndrome). Hyperinsulinaemia per se has been implicated in causing angiopathy.
- Excess of hyperglycemic hormones (glucagon,etc.)/obesity: cause relative insulin deficiency—the β cells lag behind[13]



3. GESTATIONAL DIABETIC MALITUS (GDM):-

Gestational diabetic malitus is a type of DM determined in the second or third trimester of pregnancy that is not clearly overt diabetes. GDM is a provisional disorder that happens in pregnancy and brings enduring danger of type II diabetes [18,19]. Gestational diabetes mellitus refers to glucose intolerance with onset or first recognition during pregnancy.

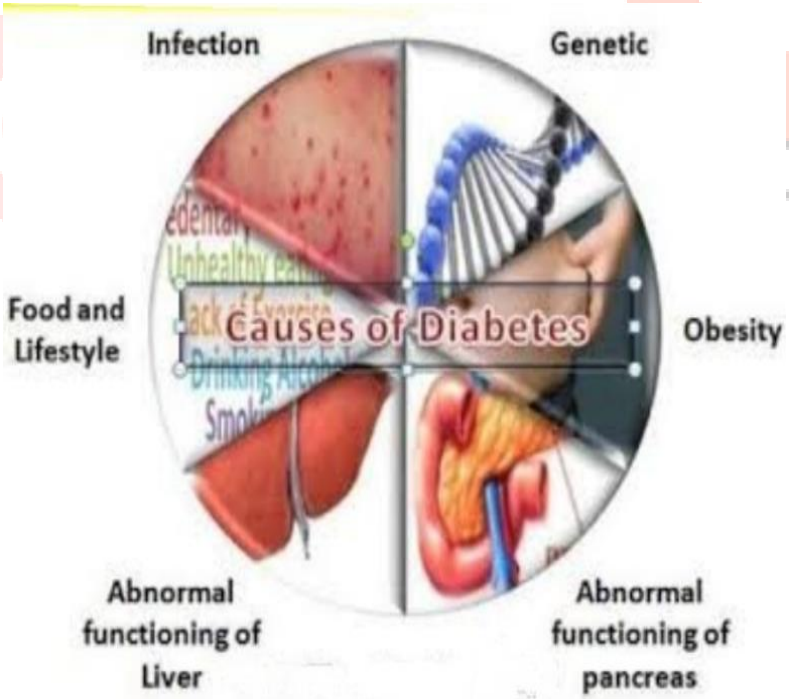
GDM occurs in approximately 7% of pregnancies and there is a greater risk of morbidity and mortality to mother, fetus and subsequent neonate. Intensive monitoring and treatment is necessary for GDM. Women with the history of GDM have a significantly increased risk of T2D and of cardiovascular diseas during the next years after delivery [20,21]. Diagnosis of type 2 diabetes before or during pregnancy is based on criteria mentioned before. Fasting plasma glucose ≥ 126 mg/dL (7.0 mmol/L) or 2-h plasma glucose ≥ 200 mg/dL (11.1 mmol/L) after a 75 g oral glucose load. However, gestational diabetes has been diagnosed at 24-28 wk of gestation in women not previously diagnosed with diabetes using two approaches: the first approach is based on the "one-step" International Association of the Diabetes and Pregnancy Study Groups (IADPSG) consensus and recently adopted by WHO[23].

4.OTHER SPECIFIC TYPE:-

Other specific types include a wide variety of relatively uncommon conditions, primarily specific genetically defined forms of diabetes or diabetes associated with other diseases or drug use Etiologic. Classification of Diabetes Mellitus[22].

FACTORS CAUSING DIABETES MALITUS:-

T1DM is mainly triggered by environmental factors. The main factors that contribute to the development of insulin resistance (T2DM) include obesity [24], physical inactivity, and smoking. The prevalence of DM is increasing due to urbanisation, westernisation and their associated lifestyle changes (nutritional habits, lack of adequate dietary intake and low physical activity) accompanied by obesity, and low socioeconomic level[25,26].



COMPLICATION:-

DM may induce several complications or can co-exist with other diseases. In everyday clinical management of diabetic patients, doctors battle with diabetic complications which are very common and come in broad spectrum of manifestations .The complications are divided in microvascular and macrovascular. The macrovascular, which are more severe, are coronary disease, stroke and peripheral neuropathy[32].The

microvascular are sneakier and in long-term maylead on macrovascular complications are diabetic retinopathy, diabetic nephropathy and diabetic foot.The increased blood glucose in diabetes mellitus leads to many complications such as metabolic changes, increased oxidative stress, cardiovascular and renal diseases.[27,28]. Diabetes is root cause for several serious complications such as cardiovascular diseases, cerebrovascular diseases, renal disorders, inflammation and immunity, and obesity [29]. Diabetic complications include hypertension, retinopathy, end-stage renal disease, neuropathy, peripheral vascular disease, electrolyte imbalance, immune suppression, erectile dysfunction, and complications of pregnancy [30]. A research study mentioned that people with less physical activity are more prone to metabolic syndrome and hypertension[31].
Thus the hazard ratio of all-cause mortality for diabetes was found to be
higher compared to nondiabetic subjects. The study also showed that mortality in diabetic subjects due to cardiovascular (52.9%) and renal disease (23.5%) was greater than the mortality due to cardiovascular (24.2%) and renal disease (6.1%) in the nondiabetic subject. A study showed that migration from the village to the city in India led to obesity, glucose
intolerance, and dyslipidemia.[33]

1.Glaucoma :-

Glaucoma is a condition in which increase in fluid pressure inside the eye leads to optic nerve damage and loss of vision. A person with diabetes is more prone to get glaucoma compared to others.

2.Diabetic maculopathy :-

Diabetic maculopathy is most commonly seen in T2DM whereas macular ischemia is more frequently seen in T1DM. Diabetic maculopathy consist of macular edema and ischemia.

3.Cardiovascular complications of diabetic malitus:-

Coronary artery disease is a major health concern and the leading cause of death in individuals with diabetes. Endothelial dysfunction and alterations at the coronary vessel wall resulting in the formation of atherosclerotic plaques are at the foundation of this cardiovascular disease. Hyperlipidaemia, hypertension, hyperglycemia, obesity and other factors interact to induce the development and instability of atherosclerotic plaques.[34]Enhanced oxidative stress appears to be a key factor in promoting high risk of atherosclerosis in diabetes.[35]

4.Diabetic nephropathy :-

Diabetic nephropathy is a kidney disease or damage that occurs in people with diabetes.Diabetic nephropathy is one of the most important causes (61%) of end-stage renal disease that requires renal replacement therapy. In people with diabetes, the nephrons thicken and slowly become scarred over time. The kidneys begin to leak and protein (albumin) passes into the urine.
People who have more severe kidney disease may have a poor appetite, feel tired most of the time,and have a general ill feeling. Headache, nausea and vomiting, swelling of the legs, and many other symptoms may also occur. Clinical progression to diabetic nephropathy is not apparently seen in T2DM
as it is in T1DM, because of the difficulty in determining the acute onset of diabetes itself. Sometimes it is difficult to differentiate minimal change nephritic syndrome (MCNS) and membranous nephropathy (MN) from diabetic nephropathy, especially in middle to advanced aged patients with
T2DM because it does not cause hematuria [36]. Diabetic nephropathy was the leading cause of end stage renal disease (ESRD) (61%) in patients with Intradialytic hypotension (IDH) [37].

5.Osteoporosis and osteoarthritis :-

Diabetes is a higher risk factor for bone and joint disorders. Osteoporosis is a thinning of the bones that weakens them and increases the risk of fractures. Osteoarthritis is a joint disorder caused by the degeneration of the joint cartilage between bones resulting in joint pain, swelling and stiffness.Individuals diagnosed with T1DM are at an increased risk of developing osteoporosis, while those withT2DM are at an increased risk of osteoarthritis. Incidence of osteoporosis and T2DM is known toincrease in prevalence with aging. Expression of insulin in pancreatic β cells as well as of adiponectin in adipocytes is increased by osteocalcin [38,39].

MANAGEMENT OF DIABETIC MALITUS:-

The management of diabetes is so important for diabetics to understand because it helps in Dealing with or controling the disease and also in avoiding complications. Maintenance of normal blood glucose levels suppresses the onset and progression of vascular and neurological complications in T1DM patients.Strategies such as diet, exercise and stress management have been strongly recommended and adopted to control T2D [40].It is recognized as being an essential part of diabetes and cardiovascular disease prevention. Meta-analyses demonstrate that lifestyle interventions, including diet and physical activity, led to a 63% reduction in diabetes incidence in those at high risk. Lifestyle modification programs have demonstrated encouraging improvement in risk factors for diabetes; however, the effect on diabetes incidence has not been reported[7]. When the diet and exercise are
unable to achieve the required glycemic control, oral glucose-lowering drugs and insulin injections are initiated. Oral antidiabetic drugs (OADs) are usually
introduced when lifestyle modifications fail to satisfactorily control hyperglycemia. These oral antidiabetic drugs are commonly given to the diabetic patients which act on the organs such as pancreas, liver and
skeletal muscle. They are very useful for managing high blood glucose especially in the early stages of the disease, achieving typical HbA1c reductions of 0.5% to 2%. The commonly used OADs are Sulphonylureas (SU) eg. Glibenclamide, Biguanides eg. Metformin, α glucosidase inhibitors eg. Acarbose and the Thiazolidinediones (TZD) eg. Pioglitazone, Rosiglitazone. The sulfonylureas and the biguanides are the major groups of oral hypoglycemic agents widely used in the treatment of diabetes, the other OADs are insulin sensitizer (Troglitazone), dipeptidyl peptidase-4 inhibitors (Sitagliptin,Saxagliptin, and Vildagliptin), incretin mimetics (glucagon-like peptide (GLP-1), Thiazolidinedione, Alpha-glucosidase inhibitors (Acarbose). normally OAD s are initiated when the fasting blood glucose level is more than 140mg/dl, postprandial blood glucose is level is 160mg/dl or above
and the Glycosylated hemoglobin level exceeds 8.0%.[41] It is recommended to organize educational programs in hospitals for teaching diabetic patients hygienic carediet, and compliance to physician's instructions regarding nutrition, exercise and medication[42,43,44].

CANCLUSION:-

The above review has given the information of diabetic,it's causes, complications and management. This review helpful to future reasercher in the field of diabetic malitus.

REFERENCE:-

1. Ahmed AM. History of diabetes mellitus. Saudi Med J 2002; Apr 23(4):37therapy

2. Southgate TM. De medicina. JAMA 1999; 10:921.

3. Patlak, M. New Weapons to Combat an Ancient Disease: Treating Diabetes. FASEB J. 2002, 16 (14), 1853.

4. Papapyros NS. The history of diabetes mellitus. In: Verlag GT, ed.
Stuttgart: Thieme 1964; 4

5. Polonsky, KS. The past 200 years indiabetes. N Engl J Med 2012; 367: 1332-40.

6. NCERT biology textbook class XI pp 338

7. M.N. Piero, G.M. Nzaro, J.M. Njagi. Diabetes mellitus – a devastating metabolic disorder. Asian Journal of Biomedical and Pharmaceutical Sciences; 04 (40); 2014 ,1-7.

8. Maharashtra state Breua textbook of 12th science frist edition:2020 pp:215

9. Wu, H.; Walker, J.; Damhuis, R. A.; Brewster, D. H.; Wild, S. H. Metformin and Survival of People with Type 2 Diabetes and Pleural Mesothelioma: A Population-Based Retrospective Cohort Study. Lung Cancer 2016, 99, 194–199.

10. Ribeiro C, de Alencar Mota CS, Voltarelli FA, de Araújo MB, Botezelli JD, et al. Effects of Moderate Intensity
Physical Training in Neonatal Alloxan- Administered Rats. J Diabetes Metab. 2010; 1:107.

11. Ragheb R, Medhat AM. Mechanisms of Fatty Acid-Induced Insulin Resistance in Muscle and Liver. J Diabetes Metab. 2011; 2:127.

12. 2013 by the American Diabetes Association, diagnosis and
Classification of diabetic malitus.

13. Tripathi KD of essential medicinal pharmacology book, sixth edition , New Delhi, India: Jaypee brother medical publisher (p) LTD pp:272

14.Uppu RM, Parinandi NL. Insulin Sensitization and Resistance Interrelationship Revisited with a Quantitative Molecular Model Approach. J Diabetes Metab. 2011; 2:106

16. Olokoba, A. B.; Obateru, O. A.and Olokoba, L. B. Type 2 Diabetes Mellitus: A Review of Current Trends. Oman Med. J. 2012, 27 (4), 269–273.

17. Standards of Medical Care in Diabetes—2015 Abridged for Primary Care Providers. Clin. Diabetes 2015, 33 (2), 97–111.

18. Piero, M. N. Diabetes Mellitus – a Devastating Metabolic Disorder. Asian J. Biomed. Pharm. Sci. 2015, 4 (40), 1–7

19. Standards of Medical Care in Diabetes--2014. Diabetes Care 2014, 37 (Supplement_1),S14–S80.

20. 7. Lemos Costa TMR, Detsch JM, Pimazoni-Netto A, de Almeida ACR, Sztal-Mazer S, et al. Glycemic Variabilityand Mean Weekly Glucose in the Evaluation and Treatment of Blood Glucose in Gestational Diabetes Mellitus;Evidence for Lower Neonatal Complications. J Diabetes Metab. 2011; 2:137.

21. Alina S, Barbara R, Krzysztof G, Barbara G, Marek G, et al. Elevation of sE-Selectin Levels from 2-24 MMonthFollowing Gestational Diabetesis Associated with Early Cardiometabolic Risk in Non-Diabetic Women. JDiabetes Metab. 2011; 2:138

22. Diabetes Canada Clinical Practice Guidelines Expert Committee Zubin Punthakee MD, MSc, FRCPC, Ronald Goldenberg MD, FRCPC, FACE, Pamela Katz MD, FRCPC

23. Diagnostic criteria and classification of hyperglycaemia first detected
in pregnancy: a World Health Organization Guideline. Diabetes Res Clin Pract 2014; 103: 341-363 [PMID: 24847517]

24. Kablan A, Saunders RA, Szkudlarek-Mikho M, Chin JB, Bosio RM, et al. Prieurianin Causes Weight Loss in Diet-Induced Obese Mice and Inhibits Adipogenesis in Cultured Preadipocytes. J Diabetes Metab. 2010; 1:101.

25. Taloyan M, Saleh-Stattin N, Johansson SE, Agréus L, Wändell P. Differences in Cardiovascular Risk Factors in Swedes and Assyrians/Syrians with Type 2 Diabetes: Association with Lifestyle-Related Factors. J Diabetes
Metab. 2010; 1:110.

26. Belmokhtar F, Belmokhtar R, Dali-Sahi M, Charef M. Risk
FactorsAssociated With Type 2 Diabetes Mellitus in West Region of Algeria, Maghnia. J Diabetes Metab. 2011; 2:148.

27. Latha S, Vijayakumar R, Senthilkumar B.R, Srikumar R, Bupesh G. Synergetic hypoglycemic and hypolipidemic effect of herbal formulation of flax seed, fenugreek and jamun seeds in streptozotocinnicotinamide induced diabetic rats. IJPT, June-2016, Vol. 8, Issue No.2, 12671-12684.

28. Latha S, Vijayakumar R, Senthil Kumar B.R, Srikumar R. In vivo anti oxidative effect of polyherbal formulation of Flax seed, fenugreek and jamun seed on streptozotocinnicotinamide Induced diabetic rats. Int J Pharm Bio Science, 2016 Oct;7(4): (B) 607 – 611.

29. Uppu RM, Parinandi NL. Insulin Sensitization and Resistance Interrelationship Revisited with a QuantitativeMolecular Model Approach. J Diabetes Metab. 2011; 2:106e

30. Li YW, Aronow WS. Diabetes Mellitus and Cardio vascular Disease. J Clinic Experiment Cardiol. 2011; 2:114

31. Mohan V, Gokulakrishnan K, Deepa R, Shanthirani CS, Datta M. Association of physical inactivity with components of metabolic syndrome and coronary artery disease – The Chennai Urban Population Study (CUPS No. 15). DiabetMed2005; 22: 1206-11.

32. 23. McMillan, T.; Girgis, R.and Sellers, E. A. C. Neonatal Diabetes and Protein Losing Enteropathy: A Case Report. BMC Med. Genet. 2016, 17 (1), 32.

33. Misra A, Pandey RM, Devi JR, Sharma R, Vikram NK, Khanna N. High prevalence of diabetes, obesity and dyslipidaemia in urban slum population in northern India. Int J Obes Relat Metab Disord 2001; 25: 1722-9

34 . Cheng AY, Leiter LA. Diabetes and cardiovascular disease: the role of glycemic control. Curr Diab Rep 2009; 9:65-72.

35. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature 2001; 414:813-20.

36. Soma J (2011) Minimal Change Nephrotic Syndrome Superimposed on Type 2 Diabetic Glomerulosclerosis. J Nephrol Therapeutic 1:e101.

37. Pavan M, Ranganath R, Chaudhari AP, Aiyangar A, Upadhayaya KL, et al. (2011) Incidence and Measures ToPrevent Intradialytic Hypotension in Patients on Maintenance Hemodialysis In a Tertiary Care Centre in India. J
Nephrol Therapeutic 1:101.

38 Kanazawa I (2011) Osteocalcin Possesses Hormonal Function Linking Bone to Glucose Metabolism. J Diabetes Metab 2:105e.

39. Lee NK, Sowa H, Hinoi E, Ferron M, Ahn JD, et al. (2007) Endocrine regulation of energy metabolism by theskeleton. Cell 130: 456-469.

40. Saini A, Devidayal, Verma S, Bhalla AK (2011) Comparative Efficacy of Once Daily Insulin Glargine with Twice Daily NPH Insulin in Children with Type 1 Diabetes. J Diabetes Metab 2:124

41. DeFronzo RA. Pharmacologic therapy for type 2 diabetes mellitus. Ann Intern Med 199;131:281–303

42. Ali ZH. Health and Knowledge Progress among Diabetic Patients after Implementation of a Nursing Care Program Based on Their Profile. J Diabet Metabol. 2011; 2:121

43 .Patra SR, Jahnavi G (2011) An Improvement in Compliance for Foot Care in Persons with Type 2 Diabetes with a Teaching Session. J Diabetes Metab 2:130.

44. David SK, Upadhayaya N, Siddiqui MK, Usmani AM (2010) Knowledge Discovery Technique for Web-Based Diabetes Educational System. J Health Med Informat 1:102