wjpmr, 2017,3(8), 58-62

SJIF Impact Factor: 4.103

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Review Article

ISSN 2455-3301

WJPMR

COMPLICATIONS OF DIABETES MELLITUS AND AYURVEDA

Kadam Krishna Namdeo[1]* and Jadhav Viraj Vilas[2]

[1]Ph.D. Scholar, Asst. Professor, Department of Roganidan and Vikriti Vigyan, Government Ayurved College, Nanded, Maharashtra, India.

[2]Associate Professor, Department of Rachna Sharira, SAM College of Ayurvedic Sci., Bhopal, India.

***Corresponding Author: Kadam Krishna Namdeo**

Ph.D. Scholar, Asst. Professor, Department of Roganidan and Vikriti Vigyan, Government Ayurved College, Nanded, Maharashtra, India.

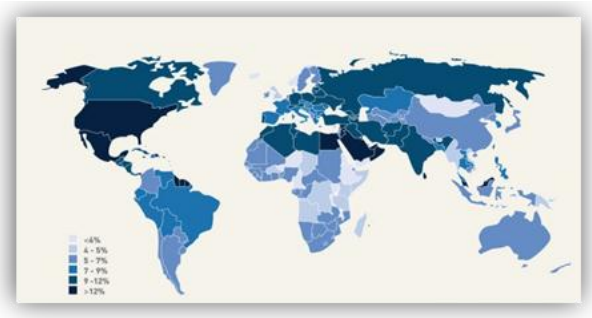
Article Received on 28/06/2017 **Article Revised on 19/07/2017** **Article Accepted on 09/08/2017**

INTRODUCTION

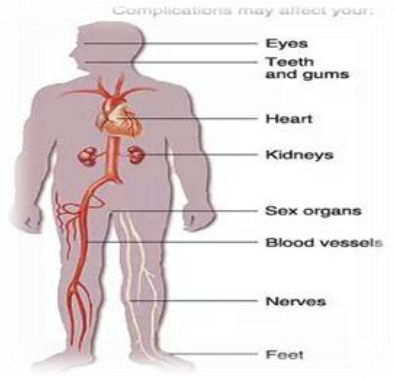
Diabetes is a metabolic disorder characterized by hyperglycaemia due to absolute or relative deficiency of insulin. In this, a person has high blood sugar either because the pancreas does not produce enough insulin or because cells do not respond to the insulin that's produced. This high blood sugar produces the classical symptoms of- Polyuria (frequent urination), Polydypsia (increased thirst), Polyphagia (increased hunger).

Long standing metabolic derangement in diabetes frequently associated with functional and structural changes in the cells of the body. Among them most characteristically affected are the eye, the kidney and the nervous system, well defined clinical entities, the so called "complications of diabetes." Approximately more than 50% of individuals suffer from these complications. But there is no satisfactory treatment in modern medicine for this condition. Thus this is the high time to have an ayurvedic approach regarding its pathological condition and to establish a comprensive treatment plan by revalidation of concerned contexts in the classics.

Prevalence (%) estimates of diabetes (20-79 years), 2030



At present, India has 35million diabetics, which is likely to reach 80million by 2030. Every 5th patient visiting a physician is diabetic. Fortunately 90 to 95% are Type 2 Diabetics not needed insulin for survival. Among them 80% of Type 2 diabetics are obese. .



Factors determining the complications

1. Genetic susceptibility
2. Duration and degree of hyperglycemia
3. Maintenance of hypertension below 130/80mm of Hg
4. Obesity and fat levels
5. Hyper Insulinemia

Complications includes

- Long term complications relate to damage to blood vessels
- Cardiovascular disorder (CVD)
- IHD (angina and MI)
- Peripheral vascular disease
- Diabetic retinopathy
- Diabetic nephropathy
- Diabetic neuropathy
- Diabetic foot ulcers

Causes includes

- The onset of type I DM is unrelated to lifestyle
- Type II DM is due primarily to lifestyle factors and genetics

Comprehensive causes

- Genetic defects of β-cell function
- Maturity onset diabetes of the young
- DNA mutations
- Genetic defects in insulin processing/insulin action
- Defects in pro insulin conversion
- Insulin gene mutation
- Insulin receptor mutations
- Exocrine pancreatic defect

a. Chronic pancreatitis
b. Pancreatectomy
c. Pancreatic neoplasia

Pathophysiology

The complication will generate because of following abnormality in metabolism

1. Decreased anabolism
2. Increased catabolism

Diabetic Neuropathy

Axonal, degeneration of both myelinated and unmyelinated fibres.

Early – axon shrinkage, late – Axonal fragmentation.

Thickening of Schewnn cell basal lamina.

Micro thrombi - These in turn result functional derangement i.e,

Reduced sensory and motor conduction velocities

Features of diabetic poly neuropathy includes.

Distal sensory loss, loss of ankle reflex, abnormal position sense, pain usually present and worsens at night, poly rediculopathy - severe disabling pain in the distribution of nerve roots. Diabetic amyotrophy - Muscular weakness and contractile pain in legs.

Diabetic Ketoacidosis (DKA)

It may be precipitated by stress or other illness. The history usually reveals polyurea, polydipsia, polyphagia and weight loss for a variable period. The striking features of salt and water depletion appear with loss of skin turgor, furred tounge and cracked lips, tachycardia, hypertension. Breathing may be deep sighing. High plasma acetone level imparts fruity odour to breath. Fever strongly suggests infection, but leucocytosis may be present without infection.

Non ketotic Hyperosmolar Coma or Hyperglycaemic Hyperosmolar State (HHS)

It is an acute catabolic complication of type 2 DM analogous to diabetic ketoacidosis in type1 DM; although not completely sparing type1 DM. It is characterized by extreme hyperglycaemia and dehydration without significant ketoacidosis. It usually affects elder age group. Up to 20% cases account for previously undiagnosed cases of diabetes. HHS is less common than DKA but mortality rate is high up to 50%. The most common precipitating factors include pneumonia and UTI accounting for 30%-50% of all cases. Others are recent myocardial infarction, sepsis, stroke, trauma, surgery that provoke release of counter regulatory hormones.

Certain medications that cause DKA can also precipitate HHS including glucocorticoids, thiazide diuretics, phenytoin and beta blockers.

Hypoglycaemia

Hypoglycaemia is defined as blood glucose level below 50mg /dl. However glucose thresholds for hypoglycaemia induced symptoms and physiological responses vary widely.

Clinical diagnosis is based upon Whipple's triad:

1. Symptoms consistent with Hypoglycaemia
2. A low plasma glucose concentration
3. Prompt relief of symptoms after glucose administration

Causes

a. In all diabetic patients Hypoglycaemia can be caused by excess exogenous insulin administration and use of some oral agents in type 2 Diabetes,
b. Lack of appropriate food intake and reduced physiological defences against falling blood glucose, due to lack of ability to recognize due to autonomic neuropathy.
c. In the face of inadequate carbohydrate replacement or insulin excess an acute exercise bout can cause post exercise hypoglycaemia.
d. Advanced age is significantly increased risk for hypoglycaemia
e. Insulin sensitivity changes over the course of night increasing from 1AM to 3AM. Combined with different insulin regimens this change can contribute to nocturnal Hypoglycaemia.
f. On the other hand insulin requirements have been to shown to increase by more than 20% in order to maintain same glycaemia level in Type1 Diabetes Mellitus patients during early morning hrs. This effect is called **dawn phenomenon**, is thought to occur due to spurt in growth hormone in early morning and cortisol leading to early morning hyperglycaemia.
g. Bedtime snacks that vary in composition and amount depending on bedtime blood glucose may also prevent nocturnal hypoglycaemia. The continuous glucose monitoring system can prevent undermined nocturnal Hypoglycaemia
h. Moderate ethanol consumption because of negative interaction with diabetic causing Hypoglycaemia.

Microvascular complications

Microangiopathies

Basement membrane thickening of small blood vessels and capillaries of different organs and tissues such as skin, skeletal muscle, eye and kidney and non vascular tissues such as peripheral nerves, renal tubules,

Bowman's capsule is histopathologic hallmark of microangiopathy of diabetes. The pathogenesis of diabetic microangiopathy and peripheral neuropathy is believed to be due to recurrent hyperglycaemia resulting in increased glycosylation of hemoglobin and other proteins.

Diabetic Nephropathy

End stage renal disease (ESRD) is leading cause of death and disability in diabetes. Nephropathy is defined as persistent albuminuria of 30 mg to 299 mg per day in the absence of other renal disease. Overt proteinuria occurs when protein excretion exceeds 300mg per day. Diabetic nephropathy occurs more frequently in IDDM (30%) than NIDDM (20 %), males being more involved than females. Diabetic patients with albuminuria are at 20 times more likely to die of cardiovascular disease.

Diabetic Retinopathy

Diabetic retinopathy is most dreaded complication of ocular manifestations of diabetes and leading cause of blindness associated with Diabetes mellitus. Prevalence is more in IDDM (40%) as compared to NIDDM (20%). Most important risk factor for development of Diabetic retinopathy is duration of diabetes and degree of glycemic control. It is rare for Diabetic retinopathy to develop within 5 yrs. of onset of Diabetes mellitus. Retinopathy can be proliferative or non proliferative. Progressive basement thickening in retinal precapillary arterioles might lead to gradual closure of these vessels and cause non perfusion of capillaries they supply.

Cardiovascular system involvement in Diabetes

Cardiovascular disease is increased in both type1 or type2 Diabetes Mellitus. Type 2 diabetic patients without a prior myocardial infarction posess similar risk for coronary artery disease as non diabetic individuals who had a prior Myocardial infarction. The absence of chest pain (silent ischemia) is common in individuals with diabetes. Risk factors for macrovascular disease in diabetics include dyslipidemia, hypertension, obesity, reduced physical activity and cigarette smoking.

Additional risk factors specific to diabetic population include microalbuminuria, gross protienuria, elevated serum creatnine, and abnormal platelet function. Insulin resistance is reflected by elevated serum insulin levels and is associated with increased risk of cardiovascular complications in individuals with or without diabetes.

- ***Dyslipidemia:*** Individuals with Diabetes Mellitus may have several forms of dyslipideamia; the most common of which is hypertriglyceridemia and reduced HDL cholesterol levels.
- ***Hypertension:*** Hypertension can accelerate other complications of Diabetes particularly Cardiovascular disease and nephropathy.

Gastrointestinal manifestations

Main oral pathologies include periodonitis and diffuse alveolar atrophy. In diabetes mellitus lower gatroesophageal sphincter tone is reduced leading to reflux esophagitis. Further altered insulin: glucagon ratio with relatively increased glucagons affects lower gatro esophageal sphincter tone and decreased gastro intestinal motility resulting in delayed gastric emptying which is known as *gastroparseis diabeticorum,* clinically manifesting as feeling of fullness after meals and anorexia. Due to delayed gastric emptying absorption of food and oral hypoglycaemic drugs may become erratic leading to brittle diabetes characterized by frequent hypoglycaemia and hyperglycaemia. The gastric atony in diabetes mellitus is due to autonomic neuropathy, probably due to diminished vagal tone. Diabetic diarrhoea occurs in long duration of diabetes. . Diarrhoea is marked in nights showing exacerbations and remission. 20% of diabetics with neuropathy may be constipated. Long standing diabetes mellitus may have mega sigmoid colon with atony as a result of autonomic neuropathy.

Pregnancy and Diabetes

Pregnancy may unmask the impaired glucose tolerance state or cause deterioration of pre existing diabetes. Factors for screening for diabetes mellitus in pregnancy:

a. Strong family history of diabetes mellitus
b. H/O Delivery of large babies over 4 kg
c. Recurrent abortions
d. Mother delivering infant with multiple congenital anomalies.
e. Unexplained stillbirth
f. Recurrent candidal vaginitis or Urinary Tract Infection
g. Obesity over 20%
h. Polyhydramnios
i. Hypertension with pregnancy

Erectile Dysfunction

Erectile dysfunction is common problem in diabetes associated with autonomic neuropathy and endothelial dysfunction. Endothelial dysfunction is characterized by decrease in bioavailability of endothelial nitric oxide which is most potent vasodilator and is secreted by endothelium. There is inverse correlation between erectile dysfunction and glycemic control.

Diabetic foot

The breakdown of Diabetic foot was traditionally considered to result from peripheral vascular disease, peripheral neuropathy and infection. More recently other contributory causes such as psychosocial factors and abnormalities of pressure and loads under the foot is implicated. There is also no compelling evidence that infection is direct cause of ulceration, it is likely that infection becomes established once the skin break occurs and thus a consequence rather than a cause of ulceration.

Infections

Individuals with Diabetes Mellitus have greater severity and frequency of infection due to incompletely understood defects in cell mediated immunity and

decreased vascularization. Hyperglycaemia aids the growth and colonization of variety of organisms (Candida and other fungal species). Many common infections like pneumonia, UTI, skin and soft tissue infections are severe in diabetics whereas several rare infections like malignant or invasive otitis externa are seen almost exclusively in diabetics.

Cutaneous manifestations

The most common skin manifestations of Diabetes Mellitus are protracted wound healing and skin ulcerations. Diabetic dermopathy sometimes termed pigmented pretibial papules or diabetic skin spots begins as an erythematous area and evolves into an area of circular hyperpigmentation. These lesions result from minor mechanical trauma in pretibial region and more common in elderly men with Diabetes Mellitus. Lipotrophy and lipohypertrophy can occur at insulin injection sites but unusual with use of human insulin. Xerosis and pruritis are common and is relieved by common skin moisturizers.

General Principles behind Ayurvedic treatment of diabetes-

1. For sthula diabetic- Sanshodhan therapy (purification mechanism)
2. For krish diabetic – Brihan therapy (plan of medicine)

Kashaya

निशा कतकादि कषाय; कतक खदिरादि कषाय; असनादि क्वाथ; वरुणादि क्वाथ; मञ्जिष्ठादि क्वाथ कोकिलाक्ष्यादि क्वाथ

Asava and Arishta

लोध्रासव; मध्वासव; देवदार्वाद्यरिष्ट

Guggulu

कैशोर गुग्गुलु; अमृत गुग्गुलु; त्रयोदशांग गुग्गुलु; योगराज गुग्गुलु; महा योगराज गुग्गुलु; चन्द्रप्रभा वटि

महा माष तैल; अश्वगन्धा तैल; नकुल तैल; हिम सागर तैल; गुडूचि तैल; आमृतादि तैल; पिण्ड तैल; बला तैल; शतापुष्पादि तैल; विषगर्भ तैल; शतावरी तैल; क्षीरबल तैल; प्रसारिणी तैल

शतावरी घृत; शतधौत घृत; अमृतप्राश घृत

त्रिवंग भस्म; वंग भस्म; स्वर्ण भस्म; रजत भस्म; अभ्रक भस्म; स्वर्ण माक्षिक भस्म; गोदन्ति (कुमारि भावित); गैरिक; कासीस

आहर – विहार

यव – Predominant diet

व्यायाम- Predominant activity

Jambul fruit

The seeds of jambul fruit should be dried and powdered. Take one teaspoon of this powder mixed in 1cup of tea or water or half a cup of curd twice day.

Bitter gourd

Take the juice of about two or three bitter gourds (karelas) every morning on an empty stomach.

Water

Put one cup of water into a copper vessel at night, and drink the water in the morning.

Goose berries

Take a tablespoon of Indian gooseberry juice mixed with a cup of bitter gourd juice, daily for two months.

Do's and dont's

DO'S	DONT'S
Have regular medical checkups to monitor the disease & adjust treatment as needed	Avoid refined carbohydrates, saturated fats
Eat meals & snacks at the same time every day or as close to the same time as possible	Avoid alcohol, soft drinks and sugary desserts
Eat fiber rich food such as vegetables , whole legumes & whole wheat products	Do not walk bare feet. Decreased sensations can result in injury
Drink 8-9 glasses of pure water daily	
Lose weight if necessary	
Take extra care of feet, gums and teeth to prevent probs that may develop because of high blood sugar	
Exercise regularly	
Wear medical identification at all times so that, in case of emergency, health professionals can see that they have diabetes	