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Ayurvedic Concept Of Diabetic Peripheral Neuropathy

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ABSTRACT
Diabetes mellitus and its complication are going to a burden for human kind .Diabetes mellitus is a metabolic disorder where primary defect lies in glucose metabolism. As a result there is wide spread multi-organ damage that ultimately encompasses virtually every system of body and consequently every specialty of medicine. The complication of diabetes mellitus is categorized into acute and chronic. Chronic complication of diabetes mellitus includes predominantly nephropathy, retinopathy and neuropathy. Peripheral neuropathy is the most common form of diabetic neuropathy. In ancient classical text of *Ayurveda*, diabetic peripheral neuropathy (DPN) has not been described separately, rather than the most of neurological symptoms of diabetic neuropathy has may be considered as symptoms of various pathophysiological phenomena that occurs in *madhumeha*, i.e. *ojo visramsa* , *dhatukshaya* and *avarana*.

Key word - Diabetic peripheral neuropathy, *Madhumeha, Ojo visramsa , Dhatukshaya, Avarana*.

INTRODUCTION

Diabetes mellitus is defined as a group of metabolic diseases characterized by chronic elevation of blood glucose (hyperglycemia) that results from defect in insulin secretion, insulin action or both.[1] Diabetes and its complications are the major causes of mortality, morbidity and decreased quality of life. Diabetic neuropathy is one of such complication characterized by sensory abnormalities like paraesthesia, burning sensation, cutaneous hyperaesthesia and numbness and loss of tendon reflexes. Diabetic neuropathy may have following clinical manifestations

- Symmetrical sensory poly neuropathy (Distal)
- Autonomic neuropathy
- Somatic neuropathy
- Acute painful neuropathy
- Mononeuropathy and mononeurities multiplex
- Asymmetrical motor diabetic neuropathy

Peripheral neuropathy is the most common form of diabetic neuropathy ranging from mild dysesthesia to severe pain that can severely affect the quality of human life. Prevalence of diabetic peripheral neuropathy (DPN) among the patients having the history of diabetes for over 10 -15 yrs, is 60-70%. Risk increases with age and duration.[2] Nerve damage due to various causes like metabolic factors, chronic hyperglycemia and long duration of diabetes, mechanical injury, smoking and alcohol abuse also responsible for manifestation of diabetic neuropathy. Painful diabetic neuropathy affects approximately 30% of diabetic patient with neuropathy.[3] The neuropathic pain initiated or caused by a primary dysfunction in the nervous system and prevalence up to 26% of all patient with diabetic mellitus[4]. On the basic of extensive study in the filled of diabetic peripheral neuropathy (DPN) the precise mechanism responsible is still not clear.
Therefore exact patho-physiological mechanism of diabetic peripheral neuropathy is unclear and there also no established treatment available so far. It is a challenging fact for *Ayurveda* also. A standardized

Ayurvedic management protocol is mostly unavailable in most of diseases associated with modern era as well as DPN. An understanding of *Ayurvedic* concept of conventional diseases is an essential requirement to design any *Ayurvedic* therapeutics. Exploration of DPN in *Ayurvedic* parlance may explore the potential field to find out a solution. This review aims at scanning the *Ayurvedic* literatures to provide an *Ayurvedic* fundamentals behind the DPN.

CRITICAL ANALYSIS

Patho-physiology of DPN

Neuropathy is one of the commonest complications of diabetes. The earliest functional change in nerves is delayed nerve conduction velocity and the earliest histological change is segmental demyelination, caused by damage to schwann cells. In the early stages axons are preserved, implying prospects of recovery, but at a later stage irreversible axonal degeneration develops.[5] Most characteristic findings of the peripheral nervous system in diabetic patients are distal and sensory predominant nerve fiber degeneration, axonal loss and endoneurial microangiopathy.[6] Two following factors are thought to play an important role behind the development of DPN

- Metabolic disarrangements
- Vascular changes

Hyperglycemia is only one of the many key metabolic events known to cause axonal and microvascular injury. A chronic hyperglycemia, leads to peripheral nerve injury via an increased flux of the polyol pathway, enhanced advanced glycation end-products (AGE) formation, elevation of inflammatory markers, exaggerated oxidative stress, mitochondrial dysfunction as well as other factors.[7]

Metabolic disarrangements in Diabetes Mellitus, are thought to be responsible behind the development of vascular complications. Reduced endoneurial blood flow with impaired auto-regulation is likely to cause ischemic damage in the nerve. Such dual influences exerted by long-term hyperglycemia are critical for peripheral nerve damage, resulting in distal-predominant nerve fiber degeneration. More recently, cellular factors derived from the bone marrow also appear to have a strong impact on the development of peripheral nerve pathology.[8]

Clinical Manifestation of DPN

DPN has been defined by the Toronto Consensus Panel on Diabetic Neuropathy as a "symmetrical, length-dependent sensorimotor polyneuropathy attributable to metabolic and microvessel alterations as a result of chronic hyperglycemia exposure and cardiovascular risk covariates.[9]

Patients with DPN typically have numbness, tingling, pain, and/or weakness that begin in the feet and spread proximally in a length-dependent fashion (stocking and glove distribution). The symptoms are symmetric with sensory symptoms more prominent than motor involvement. DPN associated numbness often causes balance problems which can lead to falls. Patients with severe DPN are at risk for ulcerations and lower extremity amputations. Overall, diabetic DPN can severely affect quality of life, particularly in those with pain.

Neuropathic pain is one of the major disabling symptoms of patients with DPN.

Like other types of neuropathic pain, DNP is characterized by burning, electric, and stabbing sensations with or without numbness.[10]

DISCUSSION

Madhumeha is a disease known to mankind since Vedic period. In ancient text compiled by *Acharya Charaka*, *Acharya Sushruta*, *Acharya Vagbhatta* and many others, we get detailed description about this disease. *Mahumeha* has been described in ayurvedic compendia either separately or under the heading of *Premeha*. *Premeha* is a disease associated with altered urinary composition and is characterized in terms of *avila-prabhuta-mutra*[11] (excessive and contaminated urination). Among 20 types of *Premeha*, *Madhumeha* is concern with *ojo kshaya*. *Ojo kshaya* is the characteristic pathological phenomenon in *Madhumeha*.[12] *Madhumeha* is the disease in which the excretion is having quality concordant with *madhu* which have similarity with early concept of Diabetes mellitus. The term Diabetes denotes the excess passage of urine whereas the term Mellitus - a Greek word for honey. It was known for centuries that the urine of patients with diabetes mellitus was sweet.

Oja is the supreme *pranayatan* out of the ten *pranayatan*[13]. Here *pran* signifies the essence of vital parts of the body[14]. Destruction of *oja* leads to various diseases and decay of the body. *Oja* is the supreme essence of all the *dhatus* increasing the vitality of protective force of the body. *Oja* always tries to exhibit the immunological responses.[15] If the immunity is less of the diabetes affected person then *dhatu kshaya* takes place due to inappropriate metabolism where the function of *agni* is impaired. *Agni* is also the responsible factor for the vitality of the body. *Oja vistransa* is characterized by *sandhi vislesh* (loosness of joints), *gatra sadan* (numbness of limbs) and *dosha chyavan* (dislodgement of the deranged humour from their respective receptacles) and *kriya sannirodha* (immobility) are very much identical to that of diabetic peripheral neuropathy.[16] *Vayu* regulates the life span (*ayu*), immunity (*vala*) and also *vayu* is the sustainer of the body in the living humans[17].

Vayu is the prime *dosa* among other *dosas* and regulates the other two *dosas.* The *vyan vayu* moves very sweeftly and pervades the entire physic of a person. It always functions in the form of *gati* (motion), *prasaran* (extension), *vikshep* (sudden movements), *nimesh* (winking of the eyes) and all other movements[18]. *Apan vayu* is located in the *vrishan* (testical), *vasti* (urinary bladder), *medhra* (phallus), *nabhi* (umbilicus), *uru* (thighs), *vankshan* (groins), *guda* (anus and colon).[19] Practically it acts in excretory process. The above said physiological functions of *vyan and apan* are very much important in context with diabetic peripheral neuropathy as in the pathological state of these two *vayu* they afflicts the body with the disease specific to their location and function[20]. Diabetic peripheral neuropathy is categorically characteristics by pain, tingling sensations, numbness and burning sensation of the hands and feets. These very characteristics are also observed in the pathological state of *vyan* and *apan*. But numbness and burning sensation of hand and feet are the pre monitory symptoms of *prameha* (*paridaham cha suptata changeshu*)[21]. But these are also very much indicative of the complications of diabetes, specifically in diabetic peripheral neuropathy.

Vayu stimulates the digestive power. *Jatharagni* is the main and the proper function of *jatharagni* stimulates the function of *bhutagni* and *dhatwagni*.[22] Lipids are deposited in the liver which stored as triglycerides or degrades them into small compound (acetyl CoA) that can be used for energy for the synthesis of other lipids specially cholesterol. Lipids can be deposited in adipose tissue where they are stored as triglycerides that can be mobilised for energy or retained as heat insulation. Hyperglycaemia leads to increased formation of sorbitol and fructose in schwann cells, accumulation of these sugars may disrupt function and structure.[23] The disease diabetes is caused due to *dhatu kshaya* and *avaran.*[24] But at the optimum level it converts into *madhumeha.*[25]

The basic responsible factor for *prameha* is *kapha* and *meda.*[26] With the specific etiological factor the *kapha* gets immediately aggravated due to its pre exaggerated quantity. The aggravated *kapha* spread all over the body because of its later developed de-compactness. In the spreading state it afflicts the *medas* because of the increased quantity of *kapha* and decreased in the viscosity of *medas*. But *kapha* and *meda* are identical. The afflicted *kapha* and *meda* affects the muscle tissues and other liquid *dhatus* of the body. Because of the prior their pre exceeded quantity. In the onwards state the affected muscle tissues produce different types of abscesses and the liquid *dhatus* of the body are again passes through the urine.[27] In another way the pathogenesis of *madhumeha* reflects that the *vata* affects the *oja,* the fundamental immunity of body, comes down to reach the *vasti.* The unarrested state of *madhumeha* again affects the vital organs along with the joints. Occlusion pathology in terms of Diabetes can damage peripheral nervous tissue in a number of ways. The vascular hypothesis postulates occlusion of the vasa nervorum as the prime cause.[28]

Enzymatic action of *kshiti, apa* and *vayu mahabhutas* on *meda* gives rise to *ksharatwa* with the formation of *asthi* and along with the action of *ushma.* The formed pores of the one due to *vayu* is filled up with *sarakta meda* located in *suksmasthi* and *sthulasthi* is filled up with *majja.*[29] These bone marrow are also related to yellowish fatty bone marrow (*sthulasthi*). It is seen that the *ushma* is the utmost factor for the formation of any d*hatu* or *upadhatu* because of its transforming activity and *snehatwa* qualities identical to fatty substances is passed by *meda* and *majja.* Therefore the *snigdha guna* is definitely important for the formation of lipid from the ingested food materials. The *snayu* is the *upadhatu of meda*.[30] It is formed by *kshara paka* of *meda* and *sneha* .[31] In diabetic peripheral neuropathy there is a maximum aggravation of *vayu* leads to *lomaharsha* (Horripilation), *khanja* (limpimg), *gatrasuptata* (Numbness in the body), *spandan*, and simultaneously *kara pada daha*(Burning sensation in the hands and feets) is also found for which *vayu* is the responsible factor.[32] Though *vata* is incapable of causing any burning sensation itself but displacement

of normal *pitta* by *vata* causes all sorts of complication attributable to *pitta*.[33] In this context the Diabetic Neuropathy is the varied form manifested with *madhumeha* is considered as an incurable (*asadhya)* disease .so the continuous methodical treatment with presence of four basic therapeutic factors, may make the disease palliable .If the patient is not properly treated or found resistant to the treatment ,the complications appear by affecting various systems. This is possibly because of the *vatic* character of the disease or predominance of *vata* in the disease along with the vitiation '*oja'*. Gradual loss of *vyadhi kshamatva* or defensive mechanism of the body and in consequence the disease process affects different *srotas* or system and various complications are manifested .*Apara oja* is related with 8 *drops* of *para oja*, the site of which is *hridaya* is also the *chetana sthana* ; that is why at extreme stage of the disease, *para oja* is vitiated and the patient under goes *bhrama* and *murchha.*

CONCLUTION

According to *Ayurvedic* principle the symptoms of Diabetic peripheral neuropathy like paraesthesia, pain and tingling sensation are indicating involment of *vata dosa* whereas burning sensation is because of vitiation of *pitta dosa* .Hence drugs pacifying *vata* and *pitta dosa* are useful in the treatment of diabetic peripheral neuropathy.

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