KAPL
VAIDYAM
A QUARTERLY JOURNAL ON AAYURVEDA



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Founder of Kerala Ayurveda Pharmacy Limited

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Contents

1. Directorial 2
2. Neurological Complications of Diabetes 3
3. Rheumatological Association in Diabetes Mellitus 5
4. Dietetic aspects of Rheumatological Association in DM 7
5. Antidiabetic Drug "GLYMIN"-An Evaluation 12
6. The Flow of Life 20
7. Heritage Column 22
8. Students' Corner 23
9. Editorial 24

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Export Potentials of Aayurvedic Health Care Delivery

[Excerpt from Welcome Address Delivered on Founder's Day of KAPL 2003]

Dr. K. Anil Kumar

The Aayurvedic community of India is presently contemplating on the global proliferation of Aayurveda. There are echoes of certain reverberations of global deliberations on Aayurveda and other complementary systems of medicine. There is frustration in the modern medical world regarding the side effects and adverse reactions of the allopathic treatment and the steadily increasing monetary input demanded by the system. This has prompted the world to think seriously of some alternate system of medicine that could be at least complementary to the conventional system. All the global deliberations and negotiations on Aayurveda find this common platform, whether it is regarding intellectual property rights on traditional treatment methods, conservation of bio-diversity, export and import of herbs and herb based products, diversification of herbal medicine as a separate branch of medicine so on and so forth. Every seminar and symposium on Aayurveda held anywhere on the globe, discuss these topics seriously.

On the occasion of delivering the welcome speech, on the founder's day of KAPL, when we remember our great founder Aayurvedaacarya Vaidyan K G K Panicker with reverence to his ideals and visions, and rededicating ourselves to the vision and mission of the founder, it has become customary for me to think loudly on a topic of relevance. Quite naturally, this year, the topic that rushes into my mind is export potentials of Aayurvedic Health Care delivery.

Aayurveda is the exporter (from India) rather than an importer. This apparently puts Aayurveda and its stakeholders in a very comfortable position. But once we are viewing the plain truth without the euphoria in us, we soon identify that the task of globalization of Aayurveda is not with out any hurdles. In fact hurdles are piled on the track. Globalization of Aayurveda has three facets: 1) Export of practice 2) Export of medicines and accessories 3) Export of information.

In the export of practice, we should be careful to export only export quality physicians. If not, sooner or later not only the physicians but Aayurveda also will be rejected by the world. Export quality aayurvedic physicians are a real rarity. A good doctor should have sound theoretical knowledge and good practical wisdom. The practice of Aayurveda out side India essentially differs in that a xenophobic community in the foreign countries closely watches our therapeutic activities. We are answerable to them. Vague answers will not satisfy them.

Since Aayurveda is not an accredited system in almost all foreign countries, even highly qualified aayurvedic doctors with their post-graduation and doctorate diplomas immediately become quacks when they land on foreign soil. Attempts to regularize aayurvedic practice out side India are in vogue without positive results. The foreign countries are stipulating norms of modern medicine for Aayurveda. Acquiring a job visa in any foreign country is also very difficult for aayurvedic doctors. The aayurvedic academy of India caters to the needs of the Indian conditions as envisaged in the preamble of the syllabus and curriculum of the CCIM. This should be amended to meet the requirement of export of physicians. If we are not changing our syllabus and curriculum to meet the requirements of modern times, there will be mushrooming of courses in foreign countries. Short Aayurveda courses conducted and emerging in foreign countries will eventually nullify job opportunities of Indian doctors. Existing practitioners of Aayurveda and the young generation of doctors should also equip themselves with contemporary knowledge to meet this global challenge.

The problems of licensing modern pharmaceutical modes and classical formulations of Aayurveda in foreign countries are unlimited and the industry as well as the Government should pay top priority to tackle this severe problem. The export of classical aayurvedic products is not an easy task. Many are attempting to bypass the obstructions by exporting products based on single drugs. But remember! Any country can export herbal drugs. Only India could export authentic aayurvedic medicines. Aayurveda got its identity by using formulations that effectively reduced the side effects of single drugs.

Paramedical staff also is a very important component in treatment. This component is gaining attention now as more and more clinics of Aayurveda are emerging all over the world. But facilities for training paramedical staff properly are rare.

(contd. on page-19)

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Neurological Complications of Diabetes

Dr. **R.V. Jayakumar**, MD, DM, MNAMS, FRCP.

One of the long-term complications of Diabetes mellitus is the involvement of the nervous system. The resulting condition commonly referred to as Diabetic Neuropathy is very distressing to the patient and very difficult to treat. The Diabetic Neuropathy is classified as Diffuse polyneuropathy, Mononeuropathy and Autonomous Neuropathy. All these three have entirely different course.

Symmetrical Sensory Neuropathy

In this condition diffuse neuropathy affects peripheral nerves symmetrically, chiefly those of the feet and legs. It is almost always sensory, though motor involvement causing weakness and wasting occur rarely. Peripheral neuropathy is common in long standing diabetes, but in older patients it may be already present at the time of diagnosis. Neuropathy is usually without symptom and it may sometimes create problems as the patient does not take adequate care. In more advanced cases patient is aware of the sensory loss, usually numbness and some sensation of coldness that may progressively worsen until there is complete anesthesia, which is rare. Reduced sensation in the feet may result in unnoticed trauma from ill-fitting shoes, nails or stones, walking barefoot or burns from hot water bottles. Crude attempts at chiropody (cutting the nails), is dangerous as it can produce a wound, which may be the source of foot infection.

Diabetic Neuropathy rarely causes symptoms in the hands, and when it does the disease is already advanced in the feet and legs. Numbness and clumsiness of the fingers are thus very unsuasual and usually due to some other neurological disorder. Impairment of sensation is however enough to interfere with the day-to-day life of some diabetic patients engaged in special crafts. Paresthesia and numbness in fingers especially at night are usually due to carpel tunnel syndrome, which may be part of diabetic neuropathy and also seen in other endocrine conditions and pregnancy. It is easily and effectively relieved by minor surgical procedure.

Wasting of small muscles of the hand and feet like that of interosseous muscle is often seen in motor neuropathy due to diabetes. In ulnar nerve compression at the elbow level the typical sensory defects are seen in the forth and fifth nerves of the hand. It causes little diasability and there is no satisfactory treatment. Patients are advised not to lean on their elbows too much, thereby avoiding further damage to their ulnar nerve.

In diabetic neuropathy, pain is usually seen and at times disabling. It occurs in the thigh in patients with femoral neuropathy, in nerve root distribution in radiculitis and in both feet in symmetrical peripheral neuropathy. The pain causes exceptional distress because it is protracted and unremitting and may last several months to years. Constant burning sensations, paresthesia, or shooting pains occur, but the most characteristic symptom is a cutaneous hypersensitivity leading to acute discomfort on contact with the clothes. The pain leads to insomnia, depression and weight loss. The patients are distressed that, they may seek different consultations with many specialists.

Treatment of painful peripheral neuropathy is very difficult. The promise that the symptoms may remit eventually may sustain patients during the periods of their illness. Diabetic control should be optimal and insulin should be given if necessary by repeated injections. Regular analgesics are essential although drugs of addiction should be avoided. Antidepressants and hypnotic agents are also helpful. It is usual to use a combination of phenathiazine and tricyclic antidepressant. Vitamins, phenytoin, carbamazepime and anti-platelet drugs have been used with inconsistent results. Electrical stimulation using a cutaneous nerve stimulator at the site of pain may also help. Relatives must be taken into confidence, to increase the morale of the depressed patient.

Mononeuropathy

Another type of diabetic neuropathy is the rapid onset type of Diabetic Mononeuropathy. Its severity and eventual resolution contrast sharply with long-term nature and irreversibility of diffuse neuropathy. These two forms of neuropathy occur quite independently of each other. The different types of Mononeuropathy are as follows:

Diabetic femoral neuropathy: This is characterized by pain, with or without wasting of one or both thighs. The quality of the pain is similar to that in painful peripheral neuropathy, and management is along similar lines. The knee jerk is absent, and sensation on the thigh may be altered or impaired. Other neurological disorders must be considered and excluded. Full recovery usually occurs within about a year.

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Radiculopathy: Root involvement may cause the characteristic pain in almost any part of the body, notably the trunk. This can produce band like sensation around the trunk.

Cranial mononeuropathy: Third and sixth nerve palsies presenting with diplopia of sudden onset, are characteristics. Pain behind the eyes occurs sometimes in third nerve palsies. In diabetic third nerve palsies, the pupil is usually spread, and ptosis does not normally occur. Full examination and careful follow-up are needed, but extensive investigation is not normally required. Complete recovery occurs spontaneously in about three months.

Autonomic Neuropathy

In long standing diabetes, damage to both parasympathetic and sympathetic nerve occurs resulting in autonomic neuropathy. The disabling symptoms those result from this, are not very common and even when they do occur, they usually are intermittent. The common symptoms of autonomic neuropathy are diarrhea, postural hypotension, impotence, neurogenic bladder, gustatory sweating and abnormal heart rhythms.

Diarrhea is characterized by severe nocturnal exacerbation, watery diarrhea and fecal incontinence. These may be preceded by abdominal rumblings. These symptoms are intermittent with normal bowel action in between and even sometimes constipation. The diagnosis is usually established by the presence of peripheral neuropathy and excluding other causes of diarrhea. A short course of tetracycline may have a dramatic effect in some of the cases. Many of the cases of diarrhea require antidiarrheal agents like codeine phosphate.

Gastro-paresis is characterized by diminished gastric motility, delayed stomach emptying, and may cause symptoms like vomiting and belching. Many of these symptoms respond to metochlopromide.

Postural hypotension which is characterized by drop of blood pressure on assuming errect posture is a common presentation of autonomic neuropathy. It requires treatment only if it is troublesome. The patients with postural hypotension are usually advised to sleep with the head of the bed raised, and wear to the full length elastic stocking while up and about. The best results are obtained by increasing the salt intake and adding a small dose of flodrocortisone.

Impotence is another common neuropathy manifestation of male diabetic patients. Autonomic neuropathy causes errectile impotence which may be permanent and irreversible. Retrograde ejaculation is another manifestation of autonomic neuropathy in which the ejaculated semen goes to the bladder, instead of urethra.

Neurogenic bladder is another autonomic neuropathy manifestation, in which condition the urinary bladder looses its tone due to loss of autonomic nerve innervation. Urinary retention, which arises from this, usually results in severe infection and may lead to urinary tract infection. Treatment includes regular voiding with straining and abdominal pressure and also use of cholinergic drugs like bethenecol.

Gustatory sweating in which facial sweating occurs around the scalp, neck and shoulders, while eating tasty foods, is a symptom of autonomic neuropathy. When it is severe it is treated with anticholinergic agents.

Abnormal cardiac rhythms are manifestations of autonomic neuropathy of heart. Resting tachycardia, and loss of heart rate variability during deep breathing and during various phases of respiration, is a manifestation of autonomic neuropathy. This can be confirmed by taking ECG recording while taking deep breath and during different phases of respiration.

In conclusion it may be said that, the nervous system is one organ that will get damaged with different manifestations in long standing diabetic patients, especially if the diabetic control is not adequate. Eventhough it is possible to treat many of these neurological complications, the distressing painful diabetic neuropathy is still a Physician's nightmare.



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Rheumatological Association in Diabetes Mellitus - An Aayurvedic Perspective

Dr. N.V. Sreevaths M.D. Ay.

Relevance of the topic

Rheumatological syndromes and diseases are commonly found associated with Diabetes mellitus. They are a major cause of morbidity. They may be debilitating. An aayurvedic physician has a definite role to play in this context.

Rheumatological Syndromes

Adhesive capsulitis of the shoulder, Shoulder Hand Syndrome, Diabetic Hand Syndrome, Duputren's contracture, Neuro-arthropathy, Hyperosteosis, Stenosing tenosynovitis etc. may be the rheumatological syndromes associated with Diabetes mellitus.

Rheumatological Diseases

Osteoarthritis, Gout, Pseudogout, Osteopenia and Osteolysis are rheumatological diseases usually associated with diabetes mellitus.

Diabetes mellitus

Diabetes mellitus is the most common endocrine disease. It usually is associated with metabolic anomalies and long term systemic complications. Polyuria, polyphagia and polydypsia are three salient symptoms of diabetes mellitus and they are attributable to the hyperglycemia present in the disease and the resultant osmotic diuresis.

Classification of Diabetes mellitus

Diabetes mellitus may be primary or secondary. The primary Diabetes mellitus is of two types viz., Type I and Type II. Type I Diabetes mellitus is an autoimmune disease and may be insulin dependant (IDDM) or non-insulin dependant (NIDDM). Type II Diabetes mellitus is non-autoimmune disease and may manifest as IDDM, NIDDM or maturity onset diabetes of youth (MODY). Secondary Diabetes mellitus may be due to pancreatic disease, hormonal abnormalities, drugs chemicals, insulin receptor abnormalities and genetic syndromes.

Ayurvedic perspective

According to Aayurveda, PRAMEHA (diabetes) is one among the eight major diseases (MAHAA VYAADHI). It occurs in the middle track of diseases (MADHYAMA ROGA MAARGA). It is caused by all the three humors (TRIDOSHA A) and is manifesting specifically on bladder which is a vital area (VASTI MARMA AAS R TA). The disease is characterized by excessive urination (PRABHOOTA MOOTRATAA) and turbidity of urine (AAVILA MOOTRATAA).

According to Aayurveda, PRAMEHA may be congenital (SAHA A) or acquired (APATHYA NIMITTA A). Congenital (hereditary) diabetes is caused by the errors in the gametes of the parents (MAATR PITR BEE A DOSHA). Acquired diabetes is due to the exercise of unwholesome practices.

According to the predominance of the humor involved there are twenty types of PRAMEHA. Ten of them are caused by KAPHA. Six varieties are caused by PITTA and four varieties are by VAATA. Prameha caused by KAPHA is curable (SAADHYA), caused by PITTA is manageable (YAAPYA) and those caused by VAATA are incurable (ASAADHYA). Congenital PRAMEHA is always incurable.

Etiology and pathogenesis of PRAMEHA

Errors of the gametes (BEE ADOSHA), unwholesome food (MITHYAA AAHAARA) and unwholesome activities (mithyaa vihaara) are the etiological factors of PRAMEHA. Due to these etiological factors the three humors are vitiated and they further cause the vitiation of the following tissues and subsidiary factors viz. the body fluid (RASA), blood (RAKTA), flesh (MAAMSA), fat (MEDAS), reproductive tissue (S UKLA), lymph (LASEEKAA), water (AMBU), adipose tissue (VASAA), marrow (MA AA) and immunity (O US). The disease manifests through the urinary system represented by the bladder (VASTI) and thus produces the premonitory symptoms.

MADH -MEHA Diabetes mellitus

It is also called kshoudra-meha and ojo-meha. This condition is either due to the depletion of tissues (DHAATU-KSHAYA- ANYA) or generated by covering or blockage (AAVARAN<u>A</u>- ANYA). Here depletion occurs to watery tissues (SAUMYA DHAATU). In the case of blockage, VAATA is engulfed or covered by other humors (DOSHA) or tissues (DHAATU). The disease is characterized by the passing of sweet (MADHURA), astringent (KASHAAYA), pale (PAAN<u>D</u>URA) and cold (S EETA) urine. The disease is associated with symptoms (LAKSHAN<u>A</u>) of the aggravation (KOPA) of VAATA.

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Diabetic carbuncles (PRAMEHA-PIT AKAA) occur as complication (UPADRAVA) of the disease.

Treatment of MADH -MEHA

The following are the guidelines of treatment of MADHU-MEHA in Aayurveda:

- Avoidance of etiological factors (NIDAANA PARIVAR ANA)
- In strong patients purification therapy (S ODHANA) followed by palliative treatment (S AMANA).
- In weak and emaciated (KR S A) patients palliative treatment (S AMANA).
- Rejuvenation therapy (RASAAYANA CIKITSA) e.g. Shilajith.
- Wholesome diet and activities (PATHYA KRAMA).
- Different approach to rich (AAD HYA) and poor (DARIDRA) patients.

Acute Metabolic Complication of Diabetes mellitus

IDDM may cause diabetic ketoacidosis and NIDDM may cause hyperosmolar, non-ketonic coma as acute metabolic complication. These conditions present the clinical situations mentioned in intoxication (MADA), fainting (MOORCHA) and coma (SANYAASA) mentioned in aayurvedic classics.

Late Complications of Diabetes mellitus

Atherosclerosis, intermittent claudication, gangrene, organic impotency in male, coronary artery disease, stroke, cardiomyopathy, retinopathy, nephropathy, neuropathy, foot ulcers, infections, dermopathy, scleroderma, contractures, hyperlipidemia and hyperviscosity are late complications of diabetes mellitus.

Rheumatological Complications

Changes in the connective tissue, due to disturbances in the structural macromolecules of extra-cellular matrix and accumulation of glycation end products in the tissues (Maillard hypothesis) cause the rheumatological complications.

According to Aayurveda, as already mentioned, rheumatological problems are associated with MADHU-MEHA. Predominance of VAATA present in both accounts for the coexistence of Diabetes mellitus (MADHU-MEHA) and rheumatism (VAATA VYAADHI). The symptoms of rheumatic complication in Diabetes mellitus depend on the location of VAATA in the bone (ASTHI), marrow (MA AA), veins (SIRAA), ligament (SNAAYU), tendon (KAND HARAA) etc. These complications may manifest as continuation of premonitory symptoms (POORVA ROOPA), symptoms (LAKSHANA), sequelae (UPADRAVA), consequence (UDARKA) or combination of diseases (VYADHI-SANKARA).

Adhesive Capsulitis of Shoulder

This is marked by diffuse shoulder pain and loss of motion in all directions, but there is no intra-articular disease. It may recover spontaneously or may be severely debilitating. The condition is comparable to APABAAHUKA, AMSA-SANDHI-GATA-VAATA etc. detailed in Aayurveda.

Shoulder Hand Syndrome

This syndrome presents ACS with pain, swelling, tenderness, dystrophic skin and vasomotor instability. This is reflex sympathetic dystrophy syndrome confined to the upper extremity. Causalgia (burning sensation) may also be present due to nerve compression. Treatment is effective if begun early. The condition is comparable to APABAAHUKA and VISVAACEE with S OPHA.

Diabetic Hand syndrome

This is also called stiff hand syndrome. This is more common in long standing Type I Diabetes mellitus. In this dermal and subcutaneous sclerosis leads to loss of mobility of finger joints. Prayer sign is the inability to bring palms together due to flexor tendon contracture. This condition is comparable to VISVAACEE, KHALLEE, and contracture (SANKOCA) due to VAATA lodged in ligaments (SNAAYU GATA VAATA), muscles (MAAMSA GATA VAATA) and tendons (KAND HARAA GATA VAATA).

Dupuytren s contracture

This is focal flexion contracture with a thickened band of palmar fascia. This presents flexion deformities with tethering skin, pain and loss of movement. This is comparable to conditions discussed before.

Neuro-arthropathy

This is commonly known as Charcot's joints. Small joints of the foot are affected. Unilateral painless foot swelling or deformity, warmth and erythema are characters. Unsuspected fracture may be present. This is secondary to loss of pain and proprioceptive sensation in long standing cases.

Stenosing tenosynovitis, carpal tunnel syndrome and hyperosteosis are other complications.

Osteo-arthritis

This is the most common degenerative disorder. This is prevalent in young and middle aged diabetics. This is more common in obese and overweight people. The disorder is comparable to SANDHI GATA VAATA.

Other conditions

Gout, characterized by hyperurecemia and pseudogout characterized by calcium pyrophosphate deposition are other rheumatological conditions associated with diabetes. These are comparable with VAATA-RAKTA. Osteopenia and osteolysis are rheumatological conditions comparable to ASTI-MA AA -GATA-VAATA and ASTI-DHAATU-KSHAYA connected with diabetes.

Aayurvedic management

Unction (SNEHANA) and sudation (SVEDANA) as in VAATAVYAADHI are to be employed to combat the rheumatic manifestations. The following points are to be kept in mind during the management. The underlying diabetes (PRAMEHA) is to be given due importance. The role of covering (AAVARANA) by humors and tissues, the location of the aggravation of VAATA

(contd. on page-19)

KAPL VAIDYAM
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Dietetic Aspects of Rheumatological Association in Diabetes Mellitus

Lakshmy Priya S.

Diabetes mellitus or MADHU-MEHA as it is termed in Ayurveda, is an endocrine disorder widely prevalent all over the world affecting millions of people in both developed and developing countries. The number of adults with diabetes in the world is estimated to increase by 122 i.e., from 135 million to 300 million during 1995 to 2025 (The Hindu, 1999). India appears to have the largest number of diabetics in the world. Even though India is the world's second most populated country in terms of general population, it is the most populated country in terms of diabetic individuals.

As per reports there are more than 100 million diabetic people in the world today out of whom more than 20 million reside in India. Every seventh person in the world is an Indian but as far as diabetes is concerned, every fifth diabetic person is an Indian. So the total population affected by this disorder in India is estimated as 2.5 crores with a distribution of 2 residing in rural areas and 8 in urban areas (Paneer Selvam, 1996).

hat is Diabetes mellitus

The word diabetes is derived from the Greek word meaning to siphon' to pass through,' and mellitus comes from the Latin word for honey'. Thus two characteristic symptoms, namely copious urination and glucose in urine give the name to the disease.

Diabetes Dye-uh-beet-eez

Mellitus Mell-ih-tus or Mell-eye-tus

Diabetes mellitus is a group of diseases characterized by high blood glucose level resulting from:

- **Defect in insulin secretion insufficiency**
- **Defect in insulin action ineffective**
- **Or both.**

According to Whitney et al (1996), it is a metabolic disorder characterized by altered blood glucose regulation and utilization usually caused by insufficient or relatively ineffective insulin.

As per WHO criteria:

It is an elevation of blood glucose when fasting and following a standard oral glucose challenge.

Fasting Blood Glucose:

140 mg 100 ml of blood	**Diabetes**
11 140 100 ml of blood	**Impaired glucose tolerance**
11 mg 100 ml of blood	**Normal**

Glucose tolerance is the ability of the body to adjust to doses of dietary carbohydrate by bringing its blood glucose back down to normal state.

Causes

Changing demographic profile, technological progress, nutritional transition with sedentary life styles and unsuitable diets have contributed to a number of chronic degenerative health problems like cardiovascular diseases, diabetes, hypertension, liver and kidney diseases, cancer etc. Heredity and aging also play their role in this problem.

OBESITY: It is a strong predictor of high prevalence of diabetes. The people who eat too much food and had a sedentary life become over weight or obese. This condition reduces the sensitivity of tissues to the action of insulin in the glucose utilization. Lack of physical exercise and obesity increase the risk of development of diabetes in the later stages of life.

SEDENTARY LIFE STYLE: Individuals who are physically active with activities like jogging, swimming, dancing or engaging in similar exercise for about 20 minutes a day have only 40 chance of developing diabetes compared to inactive individuals.

ALCOHOL: Excessive alcohol consumption reduces glucose production by the liver and thereby interferes with the mechanism that corrects hypoglycemia.

SMOKING: It impairs glucose tolerance and insulin sensitivity, enhances serum cholesterol and triglyceride levels and raises blood pressure and heart rate. Nicotine in cigarette may cause an increase in blood glucose levels.

HEREDITY: It plays an important part in onset of diabetes. The closer the blood and the stronger the family history, the

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greater will be the chances of developing diabetes.

Management of Diabetes

Although it is not possible to cure diabetes completely, diabetics can lead almost a normal life if they follow certain do's and don'ts scrupulously. Cooperation of the patient is very important in the management of diabetes.

Ob ectives in the management of diabetes are to

- Reduce the sugar in blood and urine
- Maintain ideal body weight
- Treat the symptoms
- Reduce serum lipids
- Provide adequate nutrition
- Avoid acute complications
- Prevent vascular complications

The main modes of treatment of diabetes are

- Diet
- Exercise
- Drugs
- Education

Dietary Treatment

The primary dietary consideration for a diabetic patient is that he should be a strict lacto-vegetarian and take a low-caloric, low-fat, alkaline diet of high quality, natural foods. Fruits, nuts and vegetables, whole meal bread and diary products form a good diet for the diabetic. These foods are best eaten in as dry a condition as possible to ensure thorough ensalvation during the first part of the process of digestion.

Cooked starchy foods should be avoided as in the process of cooking the cellulose envelops of the starch granules burst and consequently the starch is far too easily absorbed in the system. The excess absorbed has to be got rid of by the kidneys and appears as sugar in the urine. With raw starchy foods, however the saliva and digestive juices in the small intestine regulate the quantities required to be changed into sugar for the body's needs. The unused and undigested portion of raw starchy foods does not become injurious to the system as it does not readily ferment.

The diabetic should not be afraid to eat fresh fruits and vegetables that contain sugar and starch. A mild diabetic can also consume honey in small quantities which is rich source of fructose. Fructose does not need insulin for its metabolism and is well tolerated by diabetics. Fats and oils should be taken sparingly, for they are apt to lower the tolerance for proteins and starches. Emphasis should be on raw foods as they stimulate and increase insulin production. For protein, home made cottage cheese, various forms of soured milk and nuts are best. The patients should avoid overeating and take four or five small meals a day rather than three large ones. A diabetic should consume fluids liberally. It is always good to eat raw salads sprinkled with lime juice. Citrus fruits have effective antioxidant function.

Diabetic diet need not be a complete deviation from the normal diet. The nutritional requirements of a diabetic are the same as the non-diabetic. Normal Indian diets are generally high in carbohydrate and low in fat, with carbohydrates providing 60 65 and fat providing 15 25 of total calories. The rest is derived from proteins. Thus even a normal Indian diet is ideal for a diabetic. However, the nutrient intake has to be tailor-made to the individual based on the age, sex, weight, height, physical activity and psychological needs of the patient. It is always better to consult a dietitian to prescribe and formulate a suitable individual diet. Both doctor and dietitian have a role in the dietary management of diabetics.

Dietary Prescription

Details regarding the patient's day-to-day activity including his/her current dietary history are first collected before preparing the dietary prescription. Based on the nature of physical activity, body weight and dietary history, the total daily requirements of calories are calculated.

Calories

The calculated calorie requirement should allow the patient to lose or gain weight as required and maintain body weight 10 lower than the ideal/desirable body weight.

Ho to calculate the ideal body eight

Ideal body weight (in kg) can be simply calculated for an individual by subtracting 100 from his/her height (in cm). For example, ideal body weight of a person with 160 cm height is 60 kg (160 100). It is always better for a diabetic to maintain the body weight 10 lower than the ideal body weight.

The ideal body weight will show whether the person is overweight (20 above his ideal body weight) or underweight (20 below the ideal body weight). Based on these, the daily calorie requirements of an individual per kg body weight can be worked out as 20 Kcal. / Kg. Wt./day for over weight 30 Kcal. / Kg. Wt./day for ideal weight and 40 Kcal. / Kg. Wt./day for underweight.

This calorie intake is intended for a person who does sedentary work. For person engaged in greater physical activity, the recommended calorie intake may be increased as per the needs. A person above 50 years of age requires 10 less calories for each decade. If the disease is mild, a diet restricted in calories and devoid of refined sugars such as sweets, jam and jellies, is adequate.

Pregnant omen

Pregnant women with diabetes are more prone to certain illness than non-diabetic women. Insulin requirements go up one to three times in pregnancy. In the absence of adequate insulin, glucose cannot be utilized and instead fat is used as

fuel, resulting in ketoacidosis. Pregnant women should be screened for ketones in urine. In addition to ketoacidosis, pregnant women with diabetes are more prone to preeclampsia, toxaemia, urinary tract infections and hydromnios.

Uncontrolled diabetes during the first three months of pregnancy increases the risk of abortions and congenital malformations in the fetus. Elevated blood sugar should therefore be adequately controlled by dietary means and treatment with insulin even before conception. Delivery should be conducted under medical supervision.

Neonatal hypoglycemia and respiratory distress are common in the newborn of a diabetic mother. The newborn is typically heavier than infants born to non-diabetic mothers. Although the newborn is heavier than normal, it is premature in development and functional capacity. Thus the neonate of a diabetic mother requires special attention after birth.

Pregnant woman has to eat not only to meet her physiological needs, but also to meet the requirements of her growing fetus. During pregnancy, the diabetic should be given 30 35 Kcal/ kg of the desirable body weight. The diet should provide 1.5 to 2.5 g of protein/kg body weight. The total gain in body weight during pregnancy should ideally not exceed 12 kg.

Blood sugar should be kept within normal limits throughout pregnancy to prevent diabetes-related complications in the mother and newborn.

Children

Nutrient requirements of children relative to their body weight are generally higher than those of adults to take care of their growth and developmental needs. Children suffering from Type I diabetes require the normal diet recommended for their age group. It can be calculated based on their age. Calorie requirements of children are 1000 basal calories plus 125 calories for boys and 100 calories for girls for every year of age up to 12 years. For example, a ten year old boy requires 2250 Kcal (1000 basal 1250 for 10 years).

Distribution of nutrients in the total calories

The total daily intake of calories from carbohydrates, proteins and fats in the diet should be distributed in the following way:

Distribution of calories

	Total calories
Carbohydrates	60 65
Protein	15 20
Fat	15 25

Carbohydrates

Our diet consists mainly of carbohydrates. One gram of carbohydrate provides 4 Kcals. of energy. Generally, in Indian diet, carbohydrates provide 60 70 of the total calories. Diabetics need not restrict the carbohydrate intake, but they can alter the type of carbohydrate in their diet. Cereals and pulses contain complex carbohydrates, which are broken down into simple sugars before they are absorbed from the gut. On the other hand, sugar, honey, jaggery and jam contain simple sugars that are directly absorbed. they are referred to as refined carbohydrates and are not recommended for diabetics, as they cause a rapid rise in blood sugar, which certain complex carbohydrates do not.

Sugars present in fruits and milk raise the blood sugar at a slightly slower rate. Fruits can be taken in the right quantity. It is recommended that a diabetic derive 60 70 of calories from carbohydrates of the desirable kind.

Distribution of carbohydrates in the diet

Since the blood sugar level depends mainly on the intake of carbohydrates, it is important to distribute the intake of carbohydrates in accordance with daily needs. The total amount of carbohydrates can conveniently be divided into 4 5 equal parts. One third (33) of the diet is served during lunch and another one third (33) during dinner. Of the remaining one third, 25 is served during breakfast and the rest (9) during evening tea or at bedtime. In insulin dependent diabetics, it may be necessary to give additional carbohydrates before the patient goes to sleep to prevent hypoglycemia if the patient is on slow-acting insulin. Individuals who take a heavy breakfast and dinner, and only snacks during lunch, must modify the dietary guidelines accordingly. Distribution of carbohydrates in food should be tailor made to individual habits.

Proteins

Proteins are essential for growth, development and tissue repair. When needed proteins also provide energy in the body. One gram protein provides 4 Kcal of energy. Proteins taken in the diet are broken down into amino acids before absorption into the blood. The Recommended Dietary Allowance (RDA) for protein is 1.0 g/kg body weight. Children, pregnant women and lactating women require more protein for growth and development and other physiological needs. In addition the requirement of protein goes up in conditions such as trauma and burns. Meat and meat products, pulses legumes and nuts are all rich sources of protein. In diabetics with associated renal problems, protein is restricted to 0.6 g/kg body weight. However this should be of high biological value, such as in egg and milk.

It is generally recommended that 15 20 of total calories be derived from proteins. In insulin dependant diabetic children, 1 1.5 g of protein/kg body weight is recommended. Proteins from vegetable sources are better than from animal sources as they do not contain cholesterol and also add roughage (fiber) to the diet.

ats

Fats are concentrated sources of energy. One gram fat yields 9 Kcal. Excess intake of fat thus increases body fat and leads to obesity. Fats contain essential fatty acids, and are also vehicles for fat-soluble vitamins such as vitamin A, D, E and K.

There are three types of fats. Ghee (clarified butter), butter,

vanaspati (hydrogenated oil) and coconut oil contain a high proportion of *saturated fats* which are likely to increase the serum cholesterol. They should be taken in small quantities. Vegetable fats such as sunflower and safflower oil contain *polyunsaturated fatty acids* (PUFA) which are considered good for health. *Monounsaturated fatty acids*, present in groundnut, palm oil and olive oil are not harmful to the body. Fats from above mentioned dietary sources are described as visible fats. We also get fats from cereals, pulses, milk and milk products, eggs, flesh foods and nuts and they are considered as invisible fats.

Both the total quantity, as well as the type of fat influence the serum lipids and could increase the risk of heart diseases. Since serum lipids are generally raised in diabetes, diabetics have to be careful with the amount and nature of fat they consume. Non-vegetarian diabetics can consume fish or chicken without the skin, instead of egg, mutton, liver and brain those are high in cholesterol. Fatty acids present in fish are particularly helpful.

It is recommended that 15 25 of the total calorie can be derived from fat. The saturated, mono and poly unsaturated fats can each provide approximately equal proportions of total calories. Diabetics can take 20 g visible fat/day. Persons with high serum lipids or obesity should restrict their fat consumption particularly of saturated fats.

Vitamins and Minerals

These are protective factors those in small amounts are essential for the body. They are found in green leafy vegetables, fresh fruits, milk and diary products, cereals, nuts, fish and egg. Daily intake of these foods can provide enough vitamins and minerals.

During infections and other complications, diabetics may require higher amounts of vitamins and minerals in the form of supplements.

Dietary iber

Dietary fiber is part of food that is not digested by the gut and is considered as unavailable carbohydrate. It is not a single entity, but consists of a wide range of complex carbohydrates. Fiber present in vegetables, fruits, legumes and fenugreek seed is soluble in nature and more effective in controlling blood sugar and serum lipids than the insoluble fiber present in cereals and millets. However long-term consumption of insoluble fiber also improves glucose tolerance.

Diets containing high dietary fiber can reduce blood sugar, serum cholesterol and relieve constipation. In addition, dietary fiber is beneficial in the prevention and treatment of several diseases such as cardiovascular diseases and colon cancer. Excess dietary fiber may cause gastrointestinal symptoms such as flatulence (gas formation) and diarrhea. It may also interfere with the absorption of minerals such as iron, calcium and zinc. Intake of 25 g of dietary fiber with every 1000 calories is considered to be optimum for a diabetic. High-fiber foods have a low caloric value and low glycaemic index, and therefore diabetics should consume such foods liberally.

Dietary guidelines

In general, all foods can be classified into the following three categories for diabetics:

1. *Foods that can be used freely*
- Vegetables
- Green leafy vegetables
- Spices
- High-fiber food

2. *Foods to be used in moderate amounts*
- Fats
- Nuts
- Cereals / Roots / Tubers
- Pulses
- Fruits
- Milk products
- Meat products
- Eggs
- Artificial sweetners

3. *Foods to be avoided*
- Sugar
- Sweets
- ams and ellies.
- Cakes and pastries
- Sweetened juices and soft drinks
- Honey

Dietary alterations in associated disorders

Frequently, diabetics are likely to have other disorders and the diet has to be altered accordingly. For example, dietary fat intake has to be limited in diabetics with high serum lipids or with heart disease. Similarly, protein intake in those with renal disease and salt intake in patients with hypertension and renal disease has to be restricted.

Alcohol

It is well known that alcohol in excess is not good even for a normal person, although it is the most common drug of abuse. It may lower the blood sugar below certain critical normal limits and result in hypoglycemia. Alcohol should never be taken with some anti-diabetic drugs as it may make the diabetic very ill. Diabetics on continuous consumption of alcohol are likely to develop peripheral neuropathy. Alcohol provides empty calories, as it doesn't contain protein or fats. The extra calories may make a diabetic overweight or obese. Further, alcohol stimulates the appetite and dietary regulations are generally not complied with.

The diabetic should avoid alcohol. When a diabetic cannot avoid alcohol, as at parties, he should only take a small quantity just before or along with food, provided his diabetes is under control.

Artificial s eeteners

There are several artificial sweetening agents such as saccharin and aspartame (Sugar Free). Diabetics are generally advised to use artificial sweeteners in place of sugar. However recent researches seem to show that it is better to restrict the use of these kinds of artificial sweeteners. Diabetics can use artificial sweeteners instead of sugar in beverages.

Exercise

Exercise improves the general wellbeing not only of normal but also diabetic subjects. Exercise, when combined with reduced food intake, helps the reduction of body weight and other risk factors for heart disease. It also improves peripheral circulation. In addition, exercise enhances the action of insulin and thus helps to reduce the dose of anti-diabetic drugs.

Persons with diabetes should therefore exercise regularly. This can include brisk walking, jogging, bicycling, swimming and playing badminton and tennis. It should become a part of daily routine. In addition, exercise is known to reduce stress and strain and enhance the quality of life. The place and duration of exercise however can be decided depending on the age and physical fitness of the subject. Diabetic patients on insulin before engaging in strenuous exercise should seek medical advice to prevent hypoglycemia. They may require extra carbohydrates before, during and after exercise.

Drugs

In most patients with Type II diabetes, diet exercise and weight reduction, are enough to control high blood sugar. When the symptoms of diabetes persist, and blood sugar continues to be high, despite alterations in diet and exercise for 6 8 weeks, one has to depend on drugs as prescribed by the doctor. Depending on the type of diabetes, the doctor decides either on insulin injections or oral tablets in individual cases.

Insulin

Persons with Type I diabetes produce very little or no insulin and they, therefore, need insulin injections. Conventionally available insulins are prepared from bovine or porcine pancreas. In some individuals, conventional insulins may produce insulin resistance or allergy, or atrophy or hypertrophy at the insulin injection site. Recently newer, highly purified semi-synthetic insulins have been developed by genetic engineering. Patients having adverse effects with conventional insulins can use much purified insulins.

The doctor will decide the type of insulin that the diabetic requires. A person on insulin has to be more careful with the timing of his meals. The quantity of carbohydrate also should match the dose of insulin given. In addition to a regular schedule of breakfast, two meals and evening tea, patients with IDDM may require bedtime snacks to prevent hypoglycemia during the night. Patients with NIDDM also require insulin in certain circumstances, such as inadequate response to oral hypoglycemic agents and during ketoacidosis, acute infections, surgery and pregnancy.

Oral Drugs

In NIDDM, the body produces insulin, but this may be less effective in controlling the high blood sugar levels. Anti-diabetic tablets, in such patients either enhance the production of insulin by the pancreas, or improve the action of the insulin produced in the body. Two groups of drugs are generally used in NIDDM, sulphonylureas and biguanides. Some common sulphonylureas are tolbutamide (Rastinon), chlorpropamide (Copamide, Diabinese), glibenclamide (Euglucon, Dionil, Betanase) and glipizide (Glynase). Important biguanides are metformin (Glyciphage) and phenformin (DBI). As the mode of action of these two groups of drugs is different, the doctor will decide the type of drug suitable to the particular person with diabetes.

Education

Education of the patient is an integral and important component in the management of diabetes. The diabetic should be educated on the nature of the disease(s) they have and the possibility of development of acute and long-term complications of the disease, if blood sugar is not kept under control. Adequate basic information on diabetes enables the diabetic to comprehend and improve their psychological acceptance of the disease. In addition, the importance of following the doctor's instructions regarding diet, exercise and drugs should be explained.

Diabetics should be aware of the importance of monitoring urine and blood sugar and serum lipids at regular intervals to ensure overall wellbeing. They should learn self-monitoring of urinary sugar, either using commercially available strips or the Benedict's test. Patients who require insulin injections should know how to measure the insulin dose and to give their own injections. Diabetics should be informed about the symptoms of hypoglycemia and the immediate need for taking sugar in such conditions. Similarly they should be aware of the acute complications such as ketoacidosis and the danger of infections which require immediate medical attention. Diabetic patients should maintain a diary to keep a record of their urinary and blood sugar results, body weight and alterations in their diet and drugs.

During Illness

When diabetic fall sick, they should be careful with their activities, diet and drugs. Exercise should be avoided and rest should be taken. If usual meals cannot be eaten, at least enough liquid diet at frequent intervals should be taken. Urine or blood should be tested more often and the dose of anti-diabetic drugs prescribed by the doctor should be taken. During infections or other acute complications, a changeover to insulin may be required and the doctor should be consulted.

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Antidiabetic Drug L MIN An Evaluation

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Introduction

Diabetes mellitus comprises an etiologically and clinically heterogenous group of hyperglycemic disorders. It is continued to be defined as disorder of carbohydrate and lipid metabolism characterized by hyperglycemia and glucosurea. It may be due to the deficiency of insulin resulting from either deficiency of its supply or diminution in its effect. Different extracts of plant materials reported as anti diabetic have shown significant change in carbohydrate and lipid metabolism alone. Search for a new medicine continued for the treatment of diabetes mellitus that could significantly affect both lipid and carbohydrate derangement.

Kerala Ayurveda Pharmacy Limited had prepared a medicine by combining different plant materials that are well reported to be hypoglycemic and hypolipidaemic when used alone. The aim of this study was to focus their effect in combination. Glymin, the combination drug of KAPL has been studied for its hypoglycemic and hypolipidaemic effect. The results are reported here.

Part I. lymin an antidiabetic agent

Composition

Each uncoated tablet is prepared out of

Salacia oblonga Wall.	(EKANAAYAKAM)	300 mg
Tinospora cordifolia Miers.	(GUD OOCEE)	600 mg
Emblica officinalis Gaertn.	(AAMALAKEE)	100 mg
Curcuma longa Linn.	(RA ANEE)	100 mg
Gymnema sylvestre (Retz.) R.Br.	(MESHAS R N'GEE)	400 mg
White bitumen	(KARPOORA S ILAA ATU)	30 mg

Ingredients

1 Salacia oblonga all. (Celastraceae) is commonly known as EKANAAYAKAM or PONKORANT I in Malayalam. The root bark is golden colored and hence the name PONKORANT I. In traditional Indian medicine, the root bark of the plant is used in gonorrhea, rheumatism and skin diseases. The aquous extract of the root bark has shown hypoglycemic activity. Root bark boiled in oil or as decoction or powder is used for the treatment of rheumatism, gonorrhea, itches, asthma, thirst and ear disease. Oral administration of root bark powder showed anti-inflammatory activity.

2 Tinospora cordifolia Miers (Menispermaceae) is a well-known tonic in Indian medicine. Leaves, bark and the roots of the plant are generally used in pharmaceutics. The drug has tonic, stimulant, aphrodisiac, antiperiodic, hypoglycemic and diuretic activity. Its medicinal quality is said to be at its best during the summer months.

Experiments conducted in rabbits indicate that the aqueous and alcoholic extracts caused reduction in fasting blood sugar and increase of glucose tolerance but deterioration in tolerance occurred after a month of treatment. It has been suggested that the action of the drug is due to its effect on the endogenous insulin secretion, glucose uptake and inhibition of peripheral glucose release. The aqueous extract is very effective for toning the liver in jaundice, anemia, hepatosplenomegaly, diabetes mellitus and immuno-deficiency. The study states that Tinospora exhibits immuno-stimulant activity rather than anti-microbial action.

3 Emblica officinalis aertn. (Euphobiaceae) is referred to in ancient texts as the best medicine to prevent aging. It is a very strong rejuvenator, which is believed to be rich natural source of vitamin C.It is also effective for respiratory complaints.

4 Curcuma longa Linn. (ingiberaceae). The essential oil and the pigment curcumin cholagog and choleretic acid are antibacterial . Sci.Industr. Res., 1956,15C,256 . The rhizomes are bitter, acrid, thermogenic, emollient, anti-inflammatory and antiseptic. Soudamini et al showed that the oral administration of curcumin significantly lowered the increased lipid peroxidation in the rat tissues like liver, lung, kidney and brain produced by carbon tetrachloride. Administration of curcumin was also found to lower significantly the serum and tissue cholesterol level in rats.

ymnema sylvestre Ret . R. r. (Asclepidaceae) leaves of this plant are used as an anti-diabetic agent. The leaves have active principles having blood sugar reducing properties. (Current Med. Pract., 1959, 3 (5) 227 Biol. Abstract 1959,14336) The leaves contain two resins, one insoluble in alcohol forms the major part and another one soluble in alcohol.

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Other major ingredients are albuminous and coloring materials, Calcium oxalate, paranabin, glucose, carbohydrate, tartaric acid, glucoside ($C_{32}H_{56}O_{12}$) and gymnemic acid. The leaf material stimulates insulin secretion though it has no component having direct action on blood sugar (Congr. Luso-espan. Farm., 1952,3,197 Chem. Abstract 1954, 11728). Oral administration caused inhibition of hypoglycemic response of anterior pituitary.

Part II. lymin - Clinical Trial

Materials and Methods

The study group consisted of 26 diabetic females and 6 normal females. All the diabetic patients selected for the study were attending to the District Aayurveda Hospital, Kottayam, Kerala. There were about 150 patients for diagnosis and 95 of them were diagnosed as diabetic before attending our clinic. Of them we selected only the Non-Insulin Dependent Diabetes Mellitus (NIDDM) cases. From the selected group, 26 females were willing to take the medicine and ready for fortnightly analysis of blood sugar and other parameters for our study.

Selected patients are of the age group 45 50. Normal group samples were collected from the staff of the same institute as well as from the staff of School of Medical Education.

Before administration of the drug, the patients' samples were analyzed for different parameters for our study. The patients were stabilized for 3-4 days until the steady state concentration of the drug was attained by administering the drug initially with lower concentration and then gradually increased the concentration to the prescribed dose.

Blood samples were collected from the study subject in fasting state in EDTA vials and heparinized tubes. The plasma were separated by centrifugation and used for the measurement of glycosylated hemoglobin, glucose, urea, creatinine, ALP, AST, ALT, bilirubin, amylase, sodium, potassium, bicarbonate and chloride. Low-density cholesterol and very low-density cholesterol were calculated by using Firedwald formula.

Groups:

Group I Normal group, consists of normal persons of aged 42 to 48 years.

Group II Before Administration of Drug (BAD), consists of diabetics for the treatment. The plasma of these patients was analyzed for different parameters.

Group III After Administration of Drug (AAD), consists of diabetics for the treatment. The plasma of these patients was analyzed for different parameters after the administration of the drug for 120 days.

Table 1

asting values are mean SD of 26 female patients

Groups	Glucose (mg/dl serum)	Hemoglobin (g/ dl serum)	Glycosilated Hemoglobin
I	78.83 _1.73	13.36	7.02 _0.052
II	182.87 _1.44	8.50 _0.04	
III	137.40 _3.12	11.23 _1.07	8.02 _0.042

Group III was compared with Group II and, both group II and III were compared with group I. All values have significance of p 0.001.

Table 1a

t' between Groups	Glucose	Hemoglobin	Glycosilated Hemoglobin
III II	10.04	13.40	13.75
II I	66.53	54.51	15.54
III I	21.99	25.10	2.616

Table 2

Lipid profile

Values are mean SD of 26 female patients

Groups	Cholesterol (Mg/dl serum)	HDL (Mg/dl serum)	Triglycerides (Mg/dl serum)	LDL (Mg/dl serum)	LDL/HDL
I	170.20 _1.8	42.93 _0.93	98.46 _1.54	107.57 _2.18	2.52 _0.45
II	236.56 _6.4	30.73 _0.66	128.09 _2.60	185.60 _6.18	8.62 _1.68
III	228.55 _5.4	33.21 _0.99	122.90 _2.10	175.70 _4.28	6.78 _2.37

Table 2a

t' values to Table 2

Groups	Cholesterol	HDL	Triglycerides	LDL	LDL/HDL
III II	6.257	6.02	6.39	5.75	3.385
II I	42.89	71.38	32.07	18.63	53.52
III I	35.50	68.90	22.31	3.87	47.38

Table 3

Values are mean SD of 26 female patients

Groups	Anion gap	t' between the groups	t' value of groups
II	15.82 _0.22	III II	0.386
III	15.97 _0.13		

Results:

1. Concentration of asting lood lucose and lucose Tolerance

The concentration of blood glucose in serum of diabetic patients and alloxan treated rats were significantly higher to that of normal control groups. Treatment with Glymin in diabetic patients significantly lowered the fasting blood glucose.

2. Concentration of Hemoglobin lycosylated Hemoglobin

The concentration of serum hemoglobin in the diabetic patients was significantly lower to that of normal control groups. Treatment with Glymin in diabetic patients significantly increased the serum hemoglobin as compared to diabetic controls.

The concentration of serum glycosylated hemoglobin in diabetic patients was significantly higher to that of normal control groups. Treatment with Glymin in diabetic patients significantly lowered the serum glycosylated hemoglobin as compared to diabetic controls.

3. Concentration of liver cholesterol LDL C and triglycerides in the serum of diabetic patients

These values were significantly higher in diabetic patients when compared to that of normal individuals. Glymin significantly lowered the values after treatment.

4. Concentration of HDL C

Concentration of serum HDL (C) in the diabetic patients was significantly lower to that of normal control group. Treatment with Glymin to diabetic patients significantly increased the serum HDL (C). as compared to that of the group before treatment.

. Anion gap

The calculated anion gap does not show any significant change in the diabetic patients before and after the administration of the drug.

. The activity of amylase

The activity of amylase enzyme does not show any significant change in the diabetic patients before and after the administration of the drug.

. LDL HDL ratio

The concentration of serum LDL/HDL in the diabetic patients was significantly higher to that of normal control groups. Treatment with Glymin to diabetic patients significantly lowered the serum LDL/HDL as compared to diabetic controls.

Part III. lymin Animal Trial 1

Materials and methods:

In clinical practice, oral glucose tolerance test is conducted both as a diagnostic tool and as an index for the amelioration of diabetes in diabetic patients. This is also followed on experimental animals to assess the anti-diabetic effect of drugs.

Male albino rats of Sprague Dawley Strain, weighing 160 180g were used for the study. To induce diabetes in rats, Alloxan was injected intra-peritonially (155 mg/kg body weight) and monitored for 4-5 days to stabilize the diabetic condition. Blood sugar was measured in blood samples collected from the orbital plexus. Administration of drugs and saline was conducted through a stomach tube. Half an hour time is given for the drugs to be absorbed. After 30 minutes a solution of glucose (100 mg/ 10 ml) at a dosage of 3g/kg bodyweight was given to each of the rats with a stomach tube. Afterwards blood glucose was determined at every 30 minutes by Glucose Oxydase method.

Dose Effect Relation of Glymin

The dose effect relation of Glymin on oral administration was studied on normal rats. The results are tabulated below:

Table 4

Groups	Dose (mg/kg Body weight)	Maximum percentage fall			Maximum blood glucose reduction as of glibencla-mide (Diaonil)
		1 hr	2 hr	3 hr	
I. Glymin	100	11	17.5	13.5	60.3
II. Glymin	150	11.5	17.5	14	60.3
III. Glymin	200	13	18	15	62
IV. Diaonil	2	16	29	26	

Maximum hypoglycemic effect of Glymin and Diaonil were found at 2 hours after administration of each dose. Therefore only the second hour blood sugar values were considered for the dose effect relation.

Effect of the drug

There were six groups of rats in this study. They are:

roup I **Normal control** Normal saline was administered to them.

roup II **Diabetic control** Rendered diabetic with Alloxan induction.

roup III **Diabetic lymin** Rendered diabetic with Alloxan induction, with glucose level above 200 mg/dl. Glymin was administered to them at the dose of 100 mg/kg body weight.

roup IV **High diabetic lymin** Rendered diabetic with Alloxan induction, with glucose level above 400 mg/dl. Glymin was administered to them, on the third day of Alloxan induction, at the dose of 200 mg/kg body weight.

roup V **lymin control** Normal (non-diabetic) rats. Glymin was administered to them at the dose of 100 mg/kg body weight.

roup VI **Diabetic libenclamide Diaonil** Rendered diabetic with Alloxan induction, with glucose level above 200 mg/dl. Glibenclamide was administered to them at the dose of 2 mg/ kg body weight.

The animals were fasted for 18 hours and fasting blood sugar was estimated every fortnight in order to monitor the diabetic state. Glucose Tolerance Test (GTT) was conducted after the induction of Alloxan to confirm the diabetic state of the animals. GTT was repeated after 15 days of drug administration also to confirm the improvement of the animals.

The animals were fasted for 12 hours before sacrifice. They were stunned by a bow at the back of the neck and killed by decapitation.

Results

1. The concentration of **fasting blood glucose** in the serum of diabetic rats was significantly higher than that of normal controls. Treatment with Glymin and Glibenclamide in diabetic rats significantly lowered the fasting blood glucose level. (Table 5. t values in Table 5a.)

Blood glucose level one hour after glucose load increased significantly in diabetic controls whereas in groups treated with Glymin and Glibenclamide the level showed significantly lower values, compared to those of diabetic controls.

2. The concentration of **glycogen in liver** in the alloxan diabetic rats was lower to that of normal control group. Treatment with Glymin and Glibenclamide in diabetic rats increased the liver glycogen as compared to diabetic control. (Table 5. t values in Table 5a.)

Table 5

asting lood lucose and hepatic glycogen

(values are mean __SD of four rats in each group)

Groups	Fasting blood glucose (mg/dl)		fall or rise in blood glucose	Hepatic glycogen (g/100g wet tissue)
	Initial	Final		
I. Normal control	75_.63	79 _2.13	( ) 5.33 _ 0.1	2.80 _0.06
II. Diabetic control	238_7.3	298 _8.8	( )25.21	0.87 _0.04
III. Diabetic Glymin	230 _6.9	131 _4.4	(-)43.04	1.49 _0.05
IV. High diabetic Glymin	450 _11.1	290 _7.4	(-)35.55	1.38 _0.05
VI. Diabetic Diaonil	238 _8.61	1 44 _5.1	(-)39.49	1.51 _0.09

Group II is compared with Group I. Group III, IV VI compared with Group II.

Table 5 a

t values to table

t' between groups	Fasting blood glucose	Hepatic glycogen
II I	49.103	4.77
III II	33.940	3.21
IV II	11.390	2.63
VI II	30.315	3.39

3. The concentration of cholesterol, triglycerides and LDL cholesterol in Alloxan induced diabetic rats were significantly higher to that of normal control groups. Treatment with Glymin and Glybenclamide in diabetic rats significantly lowered the cholesterol, triglycerides and LDL cholesterol levels as compared to diabetic control. (Table 6, 6 a 7)

4. The concentration of serum HDL cholesterol in Alloxan induced diabetic rats were significantly lower to that of normal control group. Treatment with Glymin in diabetic rats significantly increased the serum HDL cholesterol as compared to the diabetic control. (Table 6 6 a)

Table 6

Concentration of Cholesterol in serum HDL LDL serum VLDL fractions

(Values are mean _ SD of four rats in each group)

Groups	Total cholesterol (mg/100 ml serum)	HDL cholesterol (mg/100 ml serum)	LDL VLDL cholesterol (mg/100 ml serum)
I	75.92 _1.89	40.7 _0.487	35.22 _2
II	200.78 _2.64	80.2 _0.237	120.58 _1.2
III	102.1 _0.56	53.4 _0.188	48.7 _2.5
IV	125.38 _1.09	60.2 _0.28	65.18 _2.2
V	77.55 _1.025	41.3 _0.225	36.25 _1.09
VI	149.33 _0.915	71.1 _0.5	78.23 _2.1

Group II is compared with Group I. Group III, IV VI are compared with Group II. All values p 0.001

Table 6a

t' between groups	Total cholesterol	HDL cholesterol	LDL VLDL cholesterol
II I	132.48	79.75	73.58
III II	101.44	53.14	52.08
IV II	73.14	30.36	46.94
VI II	48.363	25.7	8.47

Table 7

Serum Triglycerides in Alloxan diabetic rats

(Values are mean _ SD of four rats in each group)

Groups	Serum triglycerides mg/dl	t' between groups	t' value
I	28.8 _1	I II	60.38
II	75.9 _1.2		
III	43.5 _0.8	III II	44.53
IV	58.5 _0.7	IV II	25.05
VI	45.6 _0.3	VI II	49.02

All values p 0.001

5. The concentration of **cholesterol and triglycerides in hepatic tissue** of Alloxan induced diabetic rats were significantly higher to that of normal control groups. Treatment with Glymin and Glybenclamide in diabetic rats significantly lowered the cholesterol and triglyceride level as compared to diabetic control. (Table 8 8a)

Table 8

Cholesterol triglycerides in hepatic tissue

(Values are mean _ SD of four rats in each group)

Groups	Cholesterol (Mg/100 g wet tissue)	Triglycerides (Mg/100 g wet tissue)
I	336.25 _3.317	342.1
II	526.12 _1.75	444 _1.375
III	338.75 _1.44	335 _1.15
IV	385.35 _2.375	345 _1.25
V	339.75 _2.315	339 _1.50
VI	440.5 _2.50	419 _1.60

All values p 0.001

Table 8a

t values to table

t' between Groups	Cholesterol	Triglycerides
II I	169.42	157.97
III II	166.84	120.02
IV II	142.16	106.45
VI II	88.07	24.51

Group II is compared with group I, and group III, IV and VI are compared with group II

6. The **activity of glucose phosphatase in hepatic tissue** of Alloxan induced diabetic rats were significantly higher to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly lowered the activity of the enzyme as compared to diabetic control. (Table 9)

7. The **concentration of glutathione content in the hepatic tissue** of Alloxan induced diabetic rats was significantly lower to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly increased the concentration of glutathione as compared to diabetic control (Table 10)

Table 9

lucose phosephatase activity in the liver of Alloxan diabetic rats

(Values are mean _ SD of four rats in each group)

Groups	Glucose 6 phosphatase Activity of liver (Umole of Phosphate lib/min/mg protein)	t' between groups	t' value
I	0.0393 _0.0018	II I	92.605
II	0.0098 _0.0019		
III	0.0340 _0.0010	III II	70.030
IV	0.0460 _0.0070	IV II	70.030
V	0.0470 _0.0040		
VI	0.0460 _0.0070	VI II	65.234

Group II is compared with group I, and group III, IV and VI are compared with group II. All values p 0.001

8. The **activity of glutathione reductase and catalase en yme in hepatic tissue** of Alloxan treated diabetic rats was significantly lower to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly increased the activity of the enzyme as compared to diabetic control. (Table 11 11a)

Table 10

lutathone content in the liver of treated and untreated Alloxan diabetic rats

(Values are mean _ SD of four rats in each group)

Groups	HMG CoA reductase	t' between groups	t' value
I	226.00 _1.337	II I	131.53
II	115.45 _1.025		
III	156.04 _0.530	III II	73.71
IV	154.14 _0.697	IV II	60.34
V	216.66 _1.183		
VI	154.48 _1.240	VI II	61.10

Table 11

Activity of glucathine reductase and catalase in the liver in the treated and untreated Alloxan diabetic rats

Groups	Glutathione reductase (u moles of NADPH oxidized/minute/mg protein)	Catalase (values x 10 3 units /mg protein)
I	5.53 _0.03	65.80 _0.165
II	2.54 _0.044	41.30 _0.315
III	3.55 _0.04	62.00 _0.50
IV	3.25 _0.05	56.00 _0.40
V	5.15 _0.05	65.28 _0.14
VI	3.45 _0.017	46.38 _0.44

Unit velocity constant /sec. All values p 0.001

Table 11 a

t values to table 11

t' between groups	Glutathione reductase	Catalase
II I	131.53	195.68
III II	73.71	97.50
IV II	60.34	84.13
VI II	70.50	25.96

9. The **concentration of thiobarbuturic acid reactive substances T ARS** in the hepatic tissue of Alloxan induced diabetic rats was significantly higher to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly decreased the concentration of thiobarbuturic acid reactive substances as compared to diabetic control. (Table 12)

Table 12

Groups	TBARS (U moles/ mg protein)	t' between groups	t'values
I	0.84 _0.017	II I	101.43
II	2.38 _0.026		
III	0.96 _0.012	III II	99.97
IV	1.22 _0.024	IV II	64.61
V	0.93 _0.01		
VI	1.01 _0.0087	VI II	68.92

Group II is compared with group I, and group III, IV and VI are compared with group II. All values p 0.001

10. The concentration of **insulin and Apo A1** in the hepatic tissue of Alloxan induced diabetic rats were significantly lower to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly increased the concentration of insulin and Apo A1 as compared to diabetic control. The concentration of **Apo** in the hepatic tissue of Alloxan induced diabetic rats was significantly higher to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly decreased the concentration of Apo B as compared to diabetic control. (Table 13 13A)

Table 13

Concentration of insulin Apo A1 and Apo in the liver of treated and untreated Alloxan diabetic rats

Groups	Insulin (IU/ml)	Apo AI (mg/dl)	Apo B (mg/dl)
I	96.81 _1.50	5.150 _0.05	11.72 _0.17
II	36.95 _1.60	13.770 _0.07	63.40 _0.25
III	65.89 _1.34	4.825 _0.04	12.32 _0.26
IV	38.92 _1.60	4.200 _0.05	26.25 _0.38
V	92.88 _1.43	6.100 _0.01	10.15 _0.10
VI	82.81 _1.20	4.300 _0.05	24.25 _0.75

Group II is compared with group I, and group III, IV and VI are compared with group II. All values p 0.001

Table 13 a

t value of Table 13

t' between groups	Insulin	t' values Apo A1	Apo B
II I	54.91	32.86	54.91
III II	27.82	23.98	27.82
IV II	10.47	11.62	10.47
VI II	46.32	14.13	46.32

11. The **total lipolytic activity** in hepatic tissue of Alloxan induced diabetic rats was significantly lower to that of normal control group. Treatment with Glymin and Glybenclamide in diabetic rats significantly increased the activity of the enzymes as compared to diabetic control. (Table 14)

Table 14

Total lipolytic activity in the liver of treated and untreated diabetic rats

(Values are mean _SD of four rats in each group and are expressed in U moles of FFA liberated/hour/mg protein)

Groups	Lipolytic enzyme	t' between groups	t'values
I	104.50 _0.69	II I	20.23
II	90.18 _0.65		
III	108.60 _1.10	III II	28.77
IV	108.60 _1.10	IV II	15.88
V	106.18 _0.81		
VI	80.12 _0.12	VI II	30.48

Group II is compared with group I, and group III, IV and VI are compared with group II. All values p 0.001

Part IV. lymin Animal trial - 2

Aim

To assess the antidiabetic effect of Glymin by glucose tolerance test after the administration of Glymin and Glybenclamide (Diaonil) for 15 days.

Materials and methods

Male albino rats of Sprague Dawley Strain weighing 160 180 g and diabetic by induction of Alloxan were used to the study the effects of Glymin and Diaonil on Glucose tolerance after 15 days of administration of the drugs. Glymin was administered 250 mg/kg and Diaonil was administered 2 mg/ kg of body weight. Before starting the experiment the rats were fasted for 18 hours and their blood glucose was estimated. Half an hour time was given for the drugs to be absorbed. After 30 minutes a solution of glucose (100 mg/10 ml) at a dosage of 3 g/kg body weight was given to each of the rats with a stomach tube. Afterwards blood glucose was determined at every 30 minutes for three hours by glucose oxidase method.

Groups

Group I : Diabetic rats treated with Glymin (250 mg/kg body weight).

Group II : Diabetic rats treated with Glybenclamide (2 mg/ kg body weight).

Group III : High diabetic rats treated with Glymin (250 mg/ kg body weight).

Result

High diabetic rats showed an increase of only 18 in their blood glucose concentration after the first hour of GTT. Diabetic rats treated with Glybenclamide showed 49.3 increase while diabetic rats treated with glymin showed an increase of 22.2 after the first hour of GTT. (Table 15)

Table 15

TT of Diabetic rats after 1 days of drug administration

Groups	Fasting blood glucose (mg/dl)	Blood glucose after one hour (mg/dl)
I	135	165
II	295	350
III	144	215

Part V. Discussion

Oral hypoglycemic drugs play an important role in the treatment of Type II Diabetes mellitus (NIDDM). The evaluations of anti-diabetic drug Glymin' has been carried out in animals as well as in humans.

Sulfonylurea and biguanides are two groups of anti-diabetic agents available for clinical use in NIDDM. Sulfonylurea acts on the pancreatic β cells to stimulate insulin secretion so as to lower hyperglycemia. Clinical trial in 26 drug treated female patients showed a decrease in their blood sugar level after four months' study. Animal trial also showed a similar result after the administration of drug in alloxan diabetic rats for a period of two weeks.

In our study, insulin showed an increase in its serum level after the administration of Glymin. This is possible only if the drug acted as an insulin secretagogue i.e., stimulates the β cells of pancreas for insulin secretion. But the extend of this increased release of insulin in Glymin treated rats was much lower when compared to Glybenclamade administered rats.

In long term administration of the drug basal insulin level has been increased. This increase in the insulin level may not be effective in decreasing the concentration of glucose in hyperglycemic conditions, since hyperglycemia resist insulin in liver and adipose Nawano (Mar. 2000) et al . Therefore the result suggests that the decrease in the concentration of glucose showed is the direct effect of drug itself and not by increasing the concentration of insulin only.

In diabetic condition, hemoglobin concentration is decreased. Peterus (1977) et al suggest that the glycosylation of the red blood cell membrane reduce the erythrocyte survival time by 15 . The increased hemoglobin level observed in drug treated patients might be due to the improved survival of erythrocytes that suggests reduced glycocylation of RBC.

Arthurson (1974) et al reported that glycosylation of hemoglobin blocks the reduction of 2,3,DPG with positively charged residues on the B chain causing a slight, but clinically insignificant increase in oxygen affinity. Our results showed a decreased concentration of glycosylated hemoglobin. The result obtained suggests improvement in the diabetic condition of the patient and may be an index of the efficiency of the drug.

De Fronzo (1977) et al in their work explained that the glycosylation of insulin receptors could contribute to their reduced sensitivity to insulin that occurs in chronic hyperglycemia. Therefore increased level of glucose concentration in the system may increase not only the glycosylation of hemoglobin concentration but also the glycosylation of insulin resceptors of the cells.

Glucose 6 Phosphatase is a key gluconeogenic enzyme which showed an increased activity in diabetic animals. Cohn et al suggest that glucose 6 phosphatase is a multifunctional enzyme that possesses several hydrolytic and synthetic activities. It has a Krn value for glucose remarkably higher than that of glucokinase so that it prevails over glucokinase in diabetic condition and activates the gluconeogenic function.

The glucose uptake of liver is not proportionate to glucokinase activity alone, but due to difference between glucokinase and glucose 6 phosphatase activities. Our results showed a decrease in the activity of enzymes for gluconeogenesis. Thus by an increased insulin response observed in drug treated animals, the uptake of glucose is increased. This in turn results in an increased storage of glucose in the form of glycogen.

It is reported that there is derangement in the lipid metabolism of diabetes mellitus. Our results showed an increase of cholesterol in the serum of diabetic patients and Alloxan treated rats. HMG CoA reductase activity in our study showed a decrease in Alloxan treated diabetic rats. Thus the increased cholesterol in the diabetic rats may be due to the decreased removal of LDL cholesterol that may be due to the glycosylation of LDL receptors which causes decreased turn over of LDL as suggested by other. Triglycerides level in blood of diabetic patients are another significant parameter that is deranged in diabetic condition. Niklli (1973) et al reported that the poor control of diabetic patients results in hyper triglyceridemia due to increased hepatic production of VLDL coupled with delayed removal of VDL and chylomicrones. In our study the diabetic patients show a high value of triglycerides and FFA in the serum.

Goldstein further reported that the production of VLDL is secondary to increased lipolysis and elevated FFA.

There is increased lipolysis observed in diabetic patients Shiu (Aug. 1999) et al . Our results showed a decreased activity of lipoprotein and increased FFA and triglycerides in the serum of diabetic rats suggesting an increased activity of hormone sensitive lipase causing an increased lipolysis in the adipose tissue resulting in increased FFA in the serum Chu B.Y.(Aug. 1999) .

FFA that escape oxidation to ketone are re-esterified to triglycerides and secreated by the liver as nascent VLDL that causes the increased production of LDL in diabetic patients that also support the increased cholesterol in the serum of diabetic rats.

A significant increase in the activity of lipoprotein lipase is observed in the administration of the drug. This may help in

the lipolysis of tryglycerols in VLDL and chylomicrones that provides a continuous flux of free cholesterol and phospholipids to the remodeling of the surface mono layer of the lipoproteins. There is also a flux of phospholipids from cells to plasma HDL (Blelichi et al). Decreased HDL cholesterol had been observed in the diabetic rats. Our results showed an increase in HDL cholesterol in the serum of Glymin treated rats. This increase may be due to the increased activity of lipoprotein lipase LCAT and CETP. Tan K.C. (Aug. 1999) et al . The transfer of cholesterol esters from HDL to TG rich lipoproteins and LDL involves exchange of TG into HDL mediated by cholesterol ester transfer proteins.

In this connection Biesbrock (1982) et al reported that the composition of HDL is abnormal in NIDDM due to the glycosylation of HDL which causes increased clearance of glycosylated HDL. The drug may also decrease the glycosylation of HDL as the result observed in the glycosylated hemoglobin which causes the decreased clearance of HDL.

The study showed an increase of Apo A1 in the serum of the treated groups. Apo A1 activates the LCAT that helps in the removal of free cholesterol as esterified cholesterol in the blood. Decreased HDL in the serum may be a possible cause of increased cholesterol in the diabetic patients. In our study increased level of Apo A1 showed a corresponding increase in the HDL concentration in the blood also.

Administration of the drug showed a decrease in the concentration of the Apo B. Apo B proteins are mainly present in chylomicrons, VLDL and LDL. Therefore increased level of Apo B may be attributed to the increased levels of LDL, VLDL and chylomicrons in Diabetes mellitus. The decrease in the concentration of LDL and VLDL observed on administration of drug may be due to the non-availability of Apo B.

The results on hepatic lipogenic enzyme assay in our study showed a decreased lipogenic activity in diabetic conditions. The increased activity of lipogenic enzymes like Malic enzyme and glucose 6 phosphate dehydrogenase in Glymin treated rats suggests an increased production of reducing equivalent for the lipogenesis. This is possibly mediated by improved insulin action in Glymin treated rats.

Karpen (1982) et al observed an elevated level of lipid peroxides in the plasma of diabetic rats and lipid peroxidation is one of the characteristic features of chronic diabetes. Fellet (un. 1999) et al suggest an increase in the level of Thiobarbituric acid reactive substances (TBARS) in experimental diabetic rats. The increased level of Thiobarbituric acid reactive substances (TBARS) in the liver of diabetic rats may thus be due to the increased lipid peroxidation. In Glymin treated rats, the decreased TBARS may be due to the antioxidant activity of its ingredients namely Emblica officinalis, Curcuma longa Osawa (Sept. 1995) et al and Salacia oblonga K.T.Augusti et al .

In diabetic condition a decrease of Glutathione (reduced) is reported. The increased Glutathione (reduced) in the liver of Glymin treated rats may be due to the increased scavenging action of antioxidants in the drug and also due to increased activity of the antioxidant enzymes.

Catalase and Glutathione reductase are two important scavenging enzymes that remove toxic free radicals in-vivo. Administration of the drug increased the activity of both the enzymes.

From the results given above it is very clear that Glymin can be an effective anti-diabetic agent.

...Export Potentials of Aayurvedic Health Care Delivery *(contd. from page-2)*

It is a relief that some private firms are training paramedical staff. But we should maintain unanimity of course and curriculum and the Government should step forward stipulating syllabus, curriculum and minimum standards. There should be stipulations on minimum educational qualifications, standards of functioning and emphasis should be laid on proper behavior and manners of the paramedical staff. For this we should have special courses. The HRD Department of KAPL is presently engaged in conducting such courses to train paramedical personnel.

On the whole, the global aayurvedic scenario is not very promising. If this state of affairs is permitted to escalate, foreign countries will discard Aayurveda as a useless humbug. To avoid this tragedy we may earnestly try to do the following:

1) Try to equip aayurvedic doctors to meet the global standards.

2) Instead of marketing clinical medicines as food supplements, in the longer run we should attempt to license them as medicines.

3) Properly train the paramedical staff.

4) Advocate to the foreign governments to approve Aayurveda.

5) Try to propagate correct and valid information to the mass.

...Rheumatological Association in Diabeted Mellitus An Ayurvedic Perspective *(contd. from page-)*

(STHAANA of VAATA KOPA), the depletion (KSHAYA) or increase (VR DDHI) of the bone tissue (ASTHI DHAATU), the interdependence (AASRAYA-AASRAYI BANDHA) of VAATA and bone tissue (ASTHI DHAATU) and the management of fracture (BHAN'GA CIKITSAA) are to be kept in mind.

Conclusion

Ayurvedic therapeutic intervention is relevant in rheumatological association in patients with Diabetes mellitus. An individualistic, judicious, holistic management of both the diseases is necessary to offer relief.

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The lo of Life

Dr. C. R. Agnives

Life is a flow. The flowing nature of life is well appreciated by Aayurveda. The Sanskrit term for life is AAYU, from the root I' meaning to go' or to flow', and it denotes the transitional nature of life. According to Caraka Samhitaa, an authentic text of Aayurveda, NITYAGA' is a synonym for AAYU and this term denotes the constant transit or passage of life (NITYA constantly GA to go).

We all want the flow of life to be streamlined so that life is cozy, peaceful and pleasurable. We also want to perpetuate the pleasure. We do not want the flow of life to be hurdled with troubles or to be arrested completely by total blocks. But the flow of life often becomes turbulent or even stops forever. The turbulence is disease and total arrest is death.

Turbulence in a flow is due to obstruction. Innate inertia as well as external forces may pose obstructions to a flow. Of the external influences, the nature of the channel or path of flow itself is the most important and potent cause of obstruction. A blocked pipeline may partially or completely interrupt the flow of water through it. Perhaps the block may be generated by the pollution of water running through the pipe. Similarly, the flow of life depends on the quality of life itself and the circumstances confronted by life.

Life, though too common in all living things, is the most inexplicable phenomenon known to us and is a point to ponder for the philosophers. But the history of thinking has not made us any wiser about it, in spite of the volumes of recorded contemplation on the topic. Aayurvedic philosophy also has attempted to explain life from various functional and structural perspectives as all such explanations are of significance to a science that deals with life itself. Aayurveda uses the analogy of flow to illustrate the nature of life and we find that many aayurvedic terms such as channel SROTAS and blockage RODHA are based on this conception.

Aayurveda, as per its classics, is advised for the benefit of those who want to live. Life itself is of four types, viz. pleasurable life, miserable life, wholesome life and unwholesome life. What we want to attain and perpetuate is happy and wholesome life. Longing to have an unhappy or unwholesome life itself is morbid. Happiness or pleasure occurs when there is no misery. It is interesting to note that in Aayurveda, the term pleasure or happiness SUKHAM is synonymous with health AAROGYA and misery or pain is synonymous with disease DUH KHAM . The most common Sanskrit term for disease is ROGA' and it literally means pain. Pain may be of three types according to its source: celestial, biogenic and autogenic. Celestial or providential pains are natural calamities like flood draught landslide and avalanche. To a greater extend they are not under our control and hence are considered as divine. Biogenic pain is the effect of the deeds of other living things. Thus diseases caused by bites and stings and by assault of wild animals etc. are biogenic. Diseases caused by wrong deeds and thoughts of individuals come under autogenic pain. The patient himself is responsible for Autogenic pain.

The Sanskrit term for happiness or pleasure is SUKHAM. This term is comprised of two elements, the prefix SU and the word KHAM. KHAM means space and SU means good. Hence happiness is good space. Space symbolically represents channels, pathways, passages or any structure for conduction. Thus nerve fibers are passages for nerve impulses, blood vessels are passages for blood to flow and the alimentary canal is the passage for food. All these are channels and physically or functionally represent tubes for conduction. A tube has a lumen (KUSH) and a wall (STHAM) and because of this feature all tubular and baglike structures of the body are termed KOSHTAM as they have lumens and walls. It is true that nerves have no lumen. But nerves are also functional tubes as they conduct the nerve impulses. It may be noted that the term NAAD EE meaning tube is used to denote nerves too.

Thus the term SUKHAM implies the well being of channels. A good channel ensures the optimal flow through it. It should neither obstruct nor unduly accelerate the flow through it. A channel is at ease when the conduction through it is optimal. We would stress the term optimal. It is what is exactly needed, not an iota more or less. A channel should not also inadvertently push the material that passes through it. Such additional enthusiasm from the part of the channel is pathognomonic. Passage of anything through a channel is at a specific pace in par with the purpose of the passage. For example passage of food through alimentary canal is for digestion and assimilation. Hence the pace of passage of food through the alimentary tract should be optimal for these physiologic activities. An enhanced pace of passage of food through alimentary canal will not be conducive to proper digestion and absorption. Lack of digestion and absorption, especially lack of absorption of water, results in diarrhea. On the contrary, slowness of passage of the contents of the lumen of the alimentary canal and their stagnation are

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also pathognomonic. It causes constipation and various resultant problems.

Milliards of transportation are taking place in a living system. Some of them are gross and many are very minute. For every such transit there are specific channels. Some channels are macroscopic but many are microscopic pores as on the cell wall or nuclear wall. Some channels are not at all anatomical. They are mere biological pathways such as pathways for glucolysis and neoglucogenesis. The ease of transit in all these channels contribute to the ease of flow of life. When we lack it we have disease. Thus lack of disease is a matter of good space.

It goes without saying that disease is a matter of bad space. The term DUH KHAM means bad space and as already mentioned it is synonymous to disease DUH bad . A bad space inadvertently interferes with its content. It poses obstruction to its content and at times it unduly pushes its content. In either case the content of the space is exposed to stress and distress and the content is not permitted to exercise its free will. It cannot stand on its own. The concept that both excessive flow and obstruction, including retention, are diseases and all flows should be optimal seems to be very old. In Adharva Veda we find the two terms ROGAM and AASRAAVAM used together at many instances. Here ROGAM means RODHAM or obstruction and AASRAAVAM means excessive secretion. For example retention of urine and painful obstructed micturition is ROGA whereas polyurea as in diabetes mellitus is AASRAAVA. Similarly constipation in hemorrhoids is ROGA and diarrhea is AASRAAVA. Almost all disease conditions can be thus explained simply as various stages of retention or excessive flow. This simple approach has aided Aayurveda to group syndromes into a handful of diseases.

It is noteworthy that a healthy person is termed SVASTHA in Aayurveda. This means one who stands on his own SVA oneself. STHA to stand, to stay . He is not dependent on anything else. He is an independent individual. In other words, health is freedom it is liberation. It is freedom to stand on one's own. Captivated in a cell no individual can enjoy freedom, as the cell does not provide ample space for movement. In solitary confinement we feel not at ease as we have no vent for communication. Hence freedom is not a mere physical experience. It is equally mental and social. True freedom occurs from proper bondage, one's relations with others in the society. This bondage prompts our duties and liabilities. To an untrained mind this may seem to be a paradox. How an individual can experience freedom when he is under liabilities and bonds

This is where Indian philosophy differs considerably from its western counterpart. Freedom or liberation is considered as the ultimate purpose of life and it is an out come of the harmony of three opposing factors namely DHARMA, ARTHA and KAAMA. This triad is known as TRIVARGA. In each and every activity, we are expected to harmonize these three so that our actions will not generate pain. According to Aayurveda, liberation or MOKSHA is a state of complete harmony of this triad. DHARMA is what is rightfully expected of an individual by virtue of his being himself. ARTHA implies the means for life and KAAMA embodies the longings of an individual and their gratification. We should strive to attain all the three and in this strife we should see that none of the three is ignored or given over emphasis. All the three has due importance for the free flow of life. Life without sufficient means is miserable. Life devoid of gratification is worthless and painful and life without responsibilities is unwholesome. DHARMA dictates the wholesomeness of our life. The channel or path of life will permit free flow of life only if this triad is harmonized. If any three of the triad is in excess or is deficient, it will cause misery and pose obstruction to the free flow of life. In other words, man should not be a slave of the triad, he should be the master of it. He should be wise enough to operate in the space allotted to him. Unfortunately diseases handicap man by stealing his ability to harmonize the triad.

Thus we are coming to a three dimensional space, a space that has physical mental and social dimensions. Stricture in any of the three dimensions blocks the smooth flow of life. If all the three dimensions of the living space of an individual are optimal his flow of life will be uninterrupted and streamlined. As mentioned, free flow of any thing depends on its innate inertia and fluidity. Life should be innately mobile and devoid of inertia to have a free flow. But life is very prone to be polluted, as it has a tendency to pick up and accumulate filth. The filth accumulated may be physical, mental or social. To prevent and cure social filth the society has formulated ethical codes. We are expected to abide by the stipulations of the ethical codes. To keep the mind unpolluted, our mind should be devoid of the mental pollutants viz. RA AS and TAMAS. To attain this we should practice control of mind. To keep the body unpolluted, we should see that the physical pollutants or the humors viz. VAATA, PITTA and KAPHA are in equilibrium. We should avoid etiological factors that pollute the humors and use the factors conducive to their harmony. Foods, drugs and activities that are conducive to health are to be utilized and foods, drugs and activities conducive to disease should be avoided.

Aayurveda is the science and art of medicine that advises what is wholesome and what is unwholesome during health and disease. It logically modulates our foods, drugs and activities so that the physical space is kept at an optimum. Aayurveda also tunes our minds for correct thought and to eliminate mental pollutants. It also stipulates ethical norms for social well being. Thus Aayurveda attempts to normalize all the three dimensions of the path of life to ensure free flow of life. This uniqueness of Aayurveda renders it a status higher than that of a medical science and we prefer to state that Aayurveda is a way of life rather than a treatment system. Yes, Aayurveda is a way, path or channel of life that permits free flow of life.



वङ्गसेनसंहिता सटीक

श्रीमद्भिषग्वर्यवङ्गसेनविरचित

जोकि समस्त वैद्यकग्रन्थोंकी शिरोभूषण है

लखनऊ

The Van'gasena Samhitaa is one of the important classical treatises of Aayurveda. Though not esteemed at the level of the treatises of Caraka, Sus ruta and Vaagbhat a, the book surely enjoys popularity and reverence among practicing aayurvedic physicians, especially of North and East India. The period of original texts (Samhitaas) in the history of Aayurveda was followed by the period of compilations (san'grahaa). This book can be considered as an important compilation of updated aayurvedic information at the time of its emergence. The book is a manual of treatment and is practically oriented. Hence theoretical contemplation is minimal in it. The book is also named as the compilation of the essence of treatment' (cikitsaa Saara San'graha) and this name does justice to the content.

Another name for the book is Agastya Samhitaa, attributing the authorship to Agastya. It is mentioned in the book itself that the book was originally named as Agastya samhitaa and it was Van'gasena who redacted the classic to its new form and hence the name Vang'asena samhitaa. If this is true, the original book is from the period of Caraka Samhitaa. Caraka Samhitaa has referred Agastya as a reputed R shi who participated in the conference of seers at the Himalayas, that consequently deputed a delegation to heaven to fetch the science of life to the human world. More over there are formulations ascribed to Agastya such as Agastya Rasaayana. Agastya, incidentally happens to be the originator of the Siddha system of medicine too and is usually referred to as the seer source of many South Indian sciences. It seems that Agastya has brought scientific information from Sanskrit to Tamil and vice versa.

Though Van'gasena hails from Bengal, it is stated that he has migrated to South India. From the intrinsic evidence in the book we can understand Van'gasena is the son of Gadaadhara who migrated from Kaantikapura of Bengal. It is estimated that Van'gasena Samhitaa is written somewhere between 11th and 13th century CE. It is very probable that the book was written about 1210 CE. The book, though it simulates Vr nda Maadhava and Cakradatta, has many unique features. It has included plenty of mineral preparations. Though opium is not mentioned in the book, cannabis is included in some formulations of the book. The book also mentions about S an'kha draava, a chemical formulation for the treatment of ascitis (UDARA)

The title page given here is of a copy of Van'gasena Samhitaa published from Lucknow in 1904. The book, belonging to the collection of heritage materials of KAPL, is about to celebrate its centenary in a few months. The book also contains Hindi commentary by royal physician iyaram Sastri, son of Ravidatta, resident of Berinagar of Rohitaka. The book was printed at the press of Munshi Navalkishore (C.I.E.) and published by Babu Prayag Narayan. Bookseller Pandit Narayan Sastri sold the copy from Mumbay.

Here is a place for the aayurvedic students to dot down what they want. It is also a corner from where they can pick up some useful tit bits. The corner also serves as a doubt-clearing cell. Hence, dear student of Aayurveda! Knock, the corner will be open unto you. Occasionally the corner may turn quizzical and puzzle you with quizzes. At times it may preach or teach. All these are important, as today's student is tomorrow's practitioner, scientist and teacher. In this inaugural issue we are serving the ball to you. It is now your turn to strike it back as the ball is in your court.

-Editor

Sayings that go ithout saying

Here are some common maxims used in Sanskrit literature. They usually find application in Aayurveda. Maxims (axioms, aphorisms or sayings) are termed NYAAYA in Sanskrit. The term NYAAYA usually means logic. It also means method, manner, way, rule, system or plan. It also implies law, justice, virtue, equity, righteousness and honesty. NYAAYA also denotes fitness. Maxims are termed NYAAYA as analogies denoting common or scientific rules.

1. ANDHA-CAT AKA-NYAAYA by mere chance. Once upon a time a blind man caught a sparrow! ANDHA the blind CAT AKA sparrow

2. ANDHA-PARAMPARAA-NYAAYA ritual following without thought. From the train of blinds following a blind leader. PARAMPARAA sequence

3. ARUNDHATEE-DARS ANA-NYAAYA explaining the unclear with the help of what is clear. Pointing to the weak star ARUNDHATI by first coming to a bright star in the constellation polar bear' (SAPTARSHI). DARS ANA seeing

4. AS OKA-VANIKAA-NYAAYA by random selection. God knows why Raavana lodged Seeta in the garden of Saraca trees. He could have very well put her somewhere else. AS OKA Saraca asoca (Roxb.) de Wilde. S.indica auct. non Linn. VANIKAA garden

5. AS MA-LOSHT A-NYAAYA relative merit. In comparison with cotton wool, a clod of earth may be considered as rock in hardness. AS MA rock LOSHT A clod of earth

6. KADAMBA-KORAKA(GOLAKA)-NYAAYA simultaneously. All buds in the globose head (inflorescence) of Kadam tree burst open simultaneously. Kadam tree Neolamarckia cadamba (Roxb.) Bosser Anthocephalus chinensis (Lam.)Rich. Ex Walp. A. cadamba (Roxb.) Miq. KORAKA bud GOLAKA globose - head' inflorescence

7. KAAKA-TAALEEYA-NYAAYA. unexpected, accidental consequence. A crow sat on the palm and then fell the palm fruit! KAAKA crow TAALEEYA pertaining to TAALA palmyra palm The fruit was ripe enough to fall.

8. KAAKA-DANTA-GAVESHANA-NYAAYA impossible, unprofitable and unwanted task. Searching the teeth of crow! DANTA tooth GAVESHANA research

9. KAAKA-AKSHI-GOLA-NYAAYA bivalent application. From the belief that the crow has only one useful eye and it shifts between left and right as needed. The maxim is used to denote phrases that can be connected to clauses on either side. AKSHI-GOLA eye ball

10. KOOPA-YANTRA-GHAT IKAA-NYAAYA diversified by destiny. When some pots are full, others are empty. Some go up while others go down. See the fate of the row of buckets attached to the wheel of the well. KOOPA well, YANTRA machine, equipment, GHATIKAA pot . As to a particular pot, it is sometimes empty and sometimes full. Also sometimes it goes up and sometimes goes down, all not at its will.

11. GHAT T A-KUT EE-PRABHAATA-NYAAYA ending up in the hated. Like the one who roamed all night through the bye-roads to avoid toll! But in the morning he is at the tollbooth! GHAT T A KUT EE tollbooth PRABHAATA morning

12. GHUNA-AKSHARA-NYAAYA by mere chance. Like the white ant that ate away the wood to write a letter! GHUNA white ant, worm. AKSHARA letter of alphabet

13. DAND A-AAPOOPA-NYAAYA implication. When rats ate the stick of cakes, it goes without saying that the cakes are also eaten. DAND A stick AAPOOPA cake/ sweet bread

14. DEHALEE-DEEPA-NYAAYA bivalent. A lamp over the threshold lights up both the sides. DEHALEE threshold DEEPA lamp

15. NR PA-NAAPITA-PUTRA-NYAAYA innate fondness towards ones' own. The king asked the barber to select the most beautiful child in his kingdom. The barber selected his own child! NR PA king NAAPITA barber PUTRA son

16. PANKA-PRAKSHAALANA-NYAAYA prevention is better than cure. Why bother to wash the mud if you wont' tread on it PANKA mud PRAKSHAALANA washing, cleaning

17. PISHT A-PESHANA-NYAAYA unprofitable deed, such as unwarranted repetition. Grinding the ground dough. PISHT A dough, flour PESHANA grinding

18. BEE A-AN'KURA-NYAAYA mutual causation. Seed causes the sprout. It in turn produces the seed. BEE A seed AN'KURA sprout

19. LOHA-CUMBAKA-NYAAYA close affinity. As iron is attracted to a magnet. LOHA iron CUMBAKA magnet

20. VAHNI-DHOOMA-NYAAYA invariable concomitance. Where there is smoke, there is fire.

These are some examples. Now here is your homework. Find out similar maxims from aayurvedic texts and send to us with explanatory notes. Selected entries will surely find their place in this corner. Ed.

KAPL VAIDYAM
A QUARTERLY JOURNAL ON AAYURVEDA

When wisdom is lost in knowledge and knowledge is lost in information,[1] it is time to revalue the source and path of knowledge. We are living in an era of information bombardment. Avalanche after avalanche of information is showered upon us from all possible sources. But are we getting any wiser True, the vedic singer has rightly pointed out that when the ignorant falls in dark ditches, the one devoted to knowledge falls into the depths of darker and deeper pits[2] . It is a sorry state of affairs to know!

All information collected by us should lead us to correct knowledge and correct knowledge is that which augments our wisdom. For this our antennae to collect information should be normal and choosy. The information gathered by them should be processed and validated. Information those cannot be validated should be promptly rejected. The accepted information, designated as knowledge, should be clear and lucid to impart wisdom to us. This is what aayurvedic philosophy teaches us.

As physicians, all the medical information we collect from journals, books, discs, internet web sites and veterans should lead us to better clinical wisdom, aid us in developing our skills and help us to serve humanity better. Unfortunately, this is not happening. Information simply adds up to confusion of the recipients and in this confused state they tend to confuse others. This prompts the necessity for authentic information. KAPL VAIDYAM is a humble effort towards providing authentic medical information.

This inaugural issue is focussing on the Founders day of KAPL, 2003 and hence naturally contains papers presented at the scientific seminar on Rheumatological Association of Diabetes Mellitus' conducted on the day. Further, this focus reminds us the vision and mission of the founder of KAPL, Aayurvedaacarya Vaidyan K G K Panicker and fills humility in us at the auspicious start of this new venture.

The bringing out of this journal, a humble beginning, marks the actualization of a long cherished ambition of KAPL to enter into the field of aayurvedic publication and is the pilot of much more to follow suit. This English version of KAPL Vaidyam' is mainly targeting the physicians of various systems of medicine whereas a Malayalam variant, to be published shortly, is to cater for the needs of general public. Ideas of different books on Aayurveda, for various readership levels such as students, foreigners, lay men, doctors of modern medicine and aayurvedic physicians are already on the anvil. Print media is not the only avenue of our publications. We are working with the electronic media too. In fact we have already launched an online awareness program on the philosophy and practices of Aayurveda through our special academic site www.ayurvedaacademy.com.

All these ambitious projects can be actualized only with your assistance, patronage and guidance. Please feel free to comment. Each word in this journal is intended for you and your opinion is valuable to us. We are trying to spread light. But, if we are not shedding enough light, even a pen torch in your hand can add up to the luminance.

Dr. C.R.Agnives
Editor-in-Chief

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[1] *Courtesy, .S. Elliot (he Rock 192)*

[2] *Ees ovaasya panishad -9*

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