

NUTRITION IN DIABETIC ULCER: AN AYURVEDIC APPROACH

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ABSTRACT

Diabetes influences on all stages of wound healing. Diabetic ulcer is a complex disorder and its healing depends on various factors. It occurs frequently in diabetic population. It causes personal, social and economic problems to the patients and remains as major risk factor for death. Etiopathological factors are biochemical, hygiene, structural deformity, dynamic pressure, nutritional, socioeconomic factors, reduced antibacterial activity, workplace influences etc. Though “Diabetic foot” is recent nomenclature, in ancient *Ayurvedic* literature, Sushruta had explained while describing *Prameha Pidaka* the prevalence of this condition in lower limb. The role of nutrition in wound healing in diabetic ulcer is crucial in treatment of the lesion. Nutrition is critical component of wound healing in diabetic ulcer as it influences immune function, glycaemic control, and malnutrition weight loss and weight maintenance. From ancient *Ayurvedic* texts to modern dietetics all have emphasized on role of nutrition in maintaining the health. Along with total calories of intake, form of food, rapidity of absorption and whether food is chewed properly also makes the difference in nutrition. Macro and micro nutrients from food helps in wound healing by tissue repairing, enhancing the immune response and modulating inflammatory responses.

Keywords: *Pramehapidaka*, Diabetic ulcer, Nutrition.

INTRODUCTION

In contemporary science, Sushruta has described the various aspects of *Prameha* in two headings as the main disease pathology with treatment and another one special attribution to *Prameha /Madhumehapidaka*. Due to

weakness of capillaries (arteriopathy? neuropathy?), in *Pramehi*, the *doshas* accumulates in lower limb. Though Diabetic Foot Ulcer (DFU) is recent nomenclature, in ancient *Ayurvedic* literature, Sushruta has explained

while describing *Prameha Pidaka* the prevalence of this condition in lower limb. *Madhumehapidaka* in lower limb is “diabetic foot”, an inflammatory process occurring in the foot due to DM. Pathology involves *Tridosha, vasa & meda* is suggestive of ischemia, insensitivity & favorable environment for infection¹. Charaka and Vagbhata were having opinion that *Paramaha pidaka* can occur without *Prameha* in debilitating subjects^{2,3}.

Diabetic ulcer (DU) is clinical condition in diabetes, characterized by various degrees of neuropathy, arteriopathy (ischemia) and infection⁴. Diabetic ulcers are complex chronic disorder which impact on morbidity and mortality and quality of the patient’s life. It causes personal, social and economic problem and are major risk factor for death of the patient or amputation. Individual who develops DU are at greater risk of premature death, myocardial infarction, and fatal stroke than those without diabetic ulcer⁵. Diabetes influences on wound healing in DU. Peripheral arterial occlusive disorder reduces distal tissue perfusion, and defective protein synthesis, while reduced immunity causes altered neutrophil function that are associated with diabetes and remain as long standing challenge for practitioner in treatment⁶.

The DFU has major impact on patients economic, social and psychological wellbeing as this patient may not be able to work for long term due to ulceration. It represents frequent occurrence in diabetic population, and many diabetics suffers from it once in their life time.

Etiopathology: Risk factors involved in development and maintenance of DFU are ischemia due to peripheral arterial occlusion disorder, neuropathy, cigarette smoking, structural deformity and reduced antibacterial activity⁷.

Diabetic neuropathy⁸: The presence of peripheral neuropathy with the consequent reduced or absent pain sensation may initiate and sustain the ulcer process.

Among the risk factors:

- Bone deformity that exposes the bone heads to an abnormal load, and of trauma,
- Autonomic nerve dysfunction causes severely damaged “Charcot foot”.
- It decreases sweating, causing thin, dry and fissured skin that breaks easily.
- Acute wounds during nail care or minor inadvertent injuries, due to concomitant nerve damage.
- Overheated water may cause burns or blisters that later develop into chronic wounds.
- Repeated impact of an ill-fitting shoe on one area of the foot.
- Rigidity of the joints creates an abnormal pressure and increases the risk of injury.
- Calluses are an independent cause of injury and are common to discover an ulcer beneath a callus.

Diabetic Arteriopathy⁹: It is a key risk factor for lower extremity amputation. Even in the absence of a poor arterial supply, microangiopathy contributes to poor ulcer healing; it commonly affects the tibial and peroneal arteries of the calf. Persistent hyperglycemic state,

smoking, hypertension, and hyperlipidemia causes endothelial cell dysfunction and smooth cell abnormalities in peripheral arteries.

Infection in DU¹⁰: Infections are because of neuropathy, vascular insufficiency, and diminished neutrophil function. Common infection in DU is cellulitis, myositis, abscesses, necrotizing fasciitis, septic arthritis, tendinitis, and osteomyelitis. Gram-positive bacteria, such as *Staphylococcus aureus* and beta-hemolytic streptococci, are the most common pathogens in previously untreated mild and moderate infection. Severe, chronic, or previously treated infections are often polymicrobial.

Assessment of ulcer¹¹: Patients with a DFU need to be assessed holistically. This should encompass a full patient history, medication, co-morbidities and diabetes status, history of the wound, previous DFUs, amputations and any symptoms suggestive of neuropathy or peripheral arterial disease.

Clinical diagnosis¹²: It is based on clinical signs and symptoms, and not just microbiological results. Assessment of ulcer should be done about site, depth, neuropathy, presence of infection, (tissue loss), sensation and presence of signs of lower-extremity ischemia, Excessive or abnormal plantar pressure, resulting from limited joint mobility, foot deformity.

Table-1- Clinical features of Diabetic ulcer adapted from¹³

Areas at risk for DFU: Fig-1

Management of Diabetic ulcer¹⁴: The essential components of management are

- Treating underlying disease processes
- Ensuring adequate blood supply

- Local wound care, including infection control
- Abnormal pressure is reduced with adequate biophysical support and
- Texture of the skin is reconstituted

Although frequently emphasized, the general condition of the patient, the strict control of his blood glucose and the general nutritional status are too frequently overlooked.

Management of *Pramehaidaka*^{15, 16}:

- In the stage of *purvarupa- Langhana, Kashayapana*, urine of goat, *nidanaparivarjana- Aargwadhadi and Salsaradi kashaya parisheka, Pippalyadi kashaya* and *Pathachitrakadi churna* for internal use.
- *Sotha* with *Raktamamsadushti-Sodhana, Raktamokshana*.
- Suppurated swelling- Surgical debridement with 60 *vranaupakramas*.

While describing special treatment for *Pramehaidaka* Sushruta has given caution that due to excess of *Medas* and *kleda*, *swedana* is contraindicated in these subjects. Due to complexity of pathology, the patient requires the *tikshna shodhana*.

In various stages of the disease *Dhanwantaram ghritam, Salsaradi lehyam, Loharishtam, navayas Louha* preparation has indicated. In later stage of the *madhumeha*, Sushruta has also praised the role of *Silajatu Rasayana* and *Tuvaraka rasayana* after *sodhana* therapy. *Bhaishajyaratnavali* also prescribes the use of *sarivadyasava* and *Gandhaka prayoga*¹⁷.

Nutrition aspect of ulcer management¹⁸:

Diabetes often causes delayed wound healing due to elevated blood glucose level. Due to hyperglycemia, peripheral arterial occlusion distal arterial wall becomes rigid and narrow, which in turn reduces blood flow and tissue perfusion. Lack of oxygen and other nutrients, prolong inflammatory phase, impaired cell migration, inadequate leukocyte function, and insufficient collagen synthesis are few causes of poor or delayed wound healing.

Proper and adequate nutrition plays key role in wound healing¹⁹. The best nutritional approach to DU patients is to evaluate patient's current nutritional need to achieve glucose, lipid control. Normally the healing of an ulcer requires energy supplied by lipids and glucose, but diabetics have difficulty in disposing of glucose inside the cells. The reconstitution of the skin requires the supply of proteins and other biological material that is usually reduced in the diabetic and must be supplied with adequate nutrition. The best nutritional approach to the diabetic patient is:

- i. Providing adequate calories
- ii. Encouraging weight loss(if needed)
- iii. Supplement of carbohydrate and proteins
- iv. Supplements of other micro nutrients such as vitamins, mineral and iron

Role of carbohydrate: During wound healing carbohydrates are main source of cellular energy. Adequate supply of carbohydrates prevents muscle wasting, oxidation of protein and fluctuation in blood sugar.

Role of Protein: Protein provides the foundation for tissue growth, cell renewal, and repair. It plays important role in wound healing. Im-

mune cells are comprised of proteins necessary to initiate inflammatory response & collagen synthesis in healing. Insufficient dietary protein can slow the rate and quality of wound healing. Arginine & glutamine are non-essential amino acids plays an important role in wound healing. Arginine also has a positive effect on wound healing, vasodilatation and stimulates immune function. Glutamine also has a spectrum of positive effects, notably stimulation of protein synthesis and support of immune function, collagen formation Glucosamine- promotes migration & mitosis of fibroblast during healing²⁰.

Another critical component is hydration, at least 2-3 liters of water per day. In addition to the maintenance of the circulation and the tissue exchange of metabolites, the water induced diuresis helps the body to get rid of the excess glucose.

Other supplements²¹: Additional supplement of trace substances, vitamin, mineral are absolutely needed for proper wound healing particularly Vitamin A, Vitamin C, E, with strong antioxidant properties to antagonize harmful oxygen radicals present in excess in chronic wound. Trace elements such as Copper, Magnesium, Iron, selenium, Zinc etc., are lost through wound exudates in chronic wound and essential for promoting proper healing.

- Vitamin A enhances early inflammatory phase & epithelial cell differentiation.
- Vitamin C is also needed for collagen formation, immune response, cell mitosis, monocyte migration into wound tissues.

- Zinc-It is co-factor for many enzymatic reactions involved in bio synthesis of protein, DNA and hence useful for proliferation of tissues.
- Iron- Co-factor of certain enzymes which are essential for synthesis of collagen for O₂ transportation to regenerate tissues. Role of fat and Fatty acid- The precise role of fat in wound healing is not known but they are important component of cell membrane and precursor to promote inflammatory responses.

Principles of diet²²: Diabetic should be kept on well-balanced diet providing enough calories to maintain ideal body weight

- Carbohydrates: The assumption that regulation of carbohydrates alone can control DM is erroneous. The rise of blood sugar after meal does depend on amount of carbohydrates but also on rapidity of absorption, form of food
- Fiber rich foods slow stomach emptying & delay intestinal transit so, reduce the rate of glucose absorption
- Fiber also contribute to satiety and consequent decreased food intake helps reduce weight
- Phytic acid in cereals have more dominant role in decreasing weight
- Protein: supplies essential amino acids needed to tissue repair
- It does not raise blood sugar during absorption as much as carbohydrate and does not supply calories as much as fat
- Fat: 20-25% of calories cannot be oxidized as readily as carbohydrates.

- Vitamins- Carbohydrates are not completely metabolized in deficiency of Vitamin B resulting in partial metabolism of carbohydrates, causes damage to nerve and peripheral neuropathy

It is also advised to supply vitamin A as liver may be damaged in diabetes. The form of food, (powder or whole), mode of preparation & whether food is well chewed or not also makes difference to GI. Even foods of high GI give lower rise of sugar if taken after a meal. Inclusion of food with low GI decreases requirement of Anti-diabetic drugs. Natural sorbitol, an alcohol of fructose is less sweet and absorbed very slowly in intestine so does not appreciably alter blood sugar level.

Table-2-Energy source and its effect

Nutritional aspect in DFU in Ayurveda²³:

Ayurvedic diet for Pramehi includes, most of cereals, pulses & other food are having low glycemic index and positive combination of carbohydrate, protein, and fat and vitamin minerals

JirnaSali, Sastik, yava, Godhuma, Syamak, Nivar- Carbohydrates source

Mudgadi, chanak, adhaki, uddalak, Kullattha, Trinadhanya, kodrava- Carbohydrate-Protein-fiber

Dhanvamamsa- protein & essential fatty acid, vitamin A, B,

Tiktasaka, sarshapa- fiber

Aamlaka, Kapittha, Tinduka, Jambu, Asmantaka- low glycemic index fruits, hypoglycemic effect, antioxidants

Nikumbha, Ingudi, Sarshapa, Atasitaila- reduces arteriopathy (?), source of fatty acids

Jirnasidhu- natural sorbitol(?)

Sushkabhakshya (brings satiety)

CONCLUSION

Diabetic foot ulcer is complex disorder due to characteristic etiopathogenesis of recombination of ischemia, neuropathy and hyperglycemia inviting infection. After various studies it has been proved that DU should be treated by holistic approach and not merely by local wound dressing. The role of the nutritional condition of the patient with a diabetic ulcer is crucial in wound healing. Nutrition is critical component of healing of diabetic ulcer particularly as it relates to immune function, malnutrition, glycemic control, weight loss and weight maintenance. The reconstitution of the skin requires the supply of proteins and other biological material, trace elements, vitamins and minerals. Ancient *Ayurvedic* texts to modern dietetics all have emphasized on well-balanced diet to maintain ideal body weight to control the disease process. Macro and micro nutrients from food fuel source component of regenerating cells, enhances immune response, modulate inflammatory response and ultimately helps in wound healing. Irrespective of which medical treatment will be used, the closure of a wound will be impossible unless nutritional aspects are followed.

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Table 1: Clinical features of Diabetic ulcer in various pathology (adapted from¹³⁾

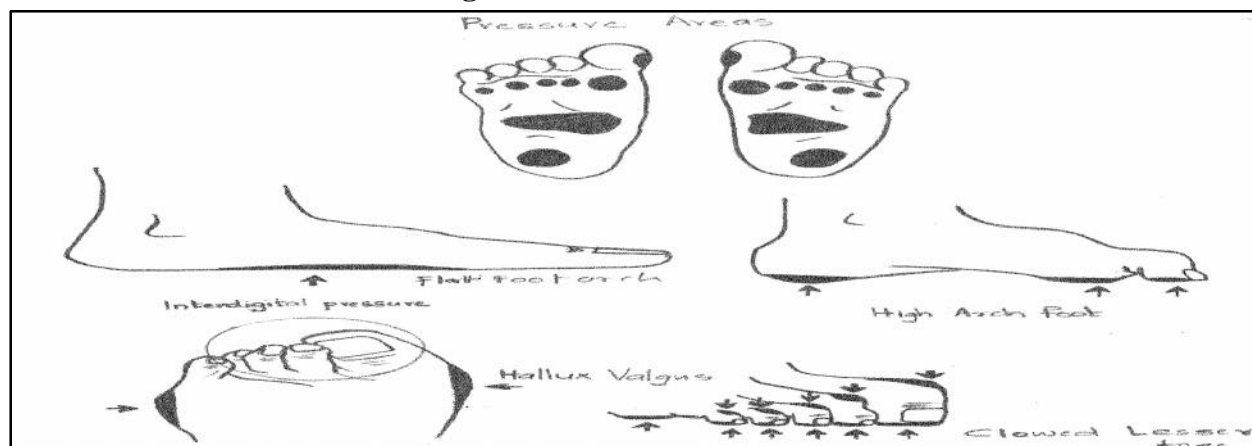
Feature	Neuropathic	Ischemic	Neuroischaemic
Sensation	Sensory loss	Painful	Varied sensoryloss
Callus	Thick Callus present	Necrosis common	Minimal callus, but Prone to necrosis
Wound floor	Pink and granulating and surrounded by callus	Pale & sloughy with poor granulation	Poor granulation in wound floor
Surrounding area temperature and pulses	Warm with bounding pulses	Cool with absent pulse	Cool with absent pulses

Other	Dry skin with fissuring	Delayed healing	High risk of Infection
Position of ulcer	Weight-bearing areas of the foot, such as metatarsal heads, the heel and over the dorsum of clawed toes	Tips of toes, nail edges and between the toes and lateral borders of the foot	Margins of the foot and toes

Table 2: Energy source and its effect

Energy source	source of diet	nutritional aspect	Wound healing
Carbohydrate	Old Sali, sastika, yava, trinadhanya (millets- kodrava, godhuma, nivar, syamak, Bajra, Jajwar, Ragi)	instant energy, more fibers(cellulose) arginin, millets control sugar, have Cu, Mn, Mg, P	Avoids ketoacidosis condition,
Protein	Mudga, chanak, uddalak, adhaki (tur dhal) Kullattha, Jangal mamsa rasa	In india combination of cereals & pulses high protein of biological value, essential amino acids	growth tissue repair, collagen synthesis , inflammatory response
Fat	Mamsa rasa, mustard oil, canola oil	Fatty acid- reduces cholesterol level	Component of cell membrane, inflammatory cell response
Vitamin	animal , fish fat, green –yellow vegetables	Vitamin A, Vitamin E, minerals like- iron, Vitamin C	Enhances early inflammatory phase & epithelial cell differentiation, collagen formation.

Figure1: Areas at risk for DFU



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