

An Ayurvedic Approach in Muscular Dystrophy in Children

Dr. Minaj Dosani¹, Dr. Harish Kumar Singhal²

¹PG Scholar, P. G. Department of Ayurveda Pediatrics, Dr. S. R. Rajasthan Ayurved University, Jodhpur, Rajasthan

²Associate Professor & Head, Post Graduate Institute of Ayurveda, Dr. S. R. Rajasthan Ayurved University, Jodhpur, Rajasthan

Corresponding Author: Dr. Minaj Dosani

DOI: <https://doi.org/10.52403/ijhsr.20240318>

ABSTRACT

Duchenne muscular dystrophy stands out as the most prevalent and severe form of childhood muscular dystrophy, impacting approximately one in every 5200 male births. It results from dystrophin deficiency, a condition inherited through X-linked recessive traits due to a missing or altered dystrophin protein encoded by the DMD gene located on chromosome Xp21. Unfortunately, this myopathy is currently incurable, often leading to mortality between the ages of 20-25. The primary pharmaceutical intervention for Duchenne muscular dystrophy involves corticosteroids, though they come with long-term negative consequences. In the realm of *Ayurveda*, Duchenne muscular dystrophy falls under the classification of *Medomamsa dushti*, attributed to *Vata doshas* and stemming from *Bheejabagahaavyava dushti*. *Ayurvedic* strategies for management emphasize the promotion of regeneration in neuromuscular illnesses, employing a combination of *Ayurvedic* oral drugs and *Panchakarma* therapies.

Key words: DMD, Dystrophin, Muscular Dystrophy, *Medovaha Srotodushti*

INTRODUCTION

A category of disorders known as muscular dystrophies produce unrelenting, gradual muscular weakening and muscle mass loss.¹ The most prevalent of these illnesses is Duchenne muscular dystrophy (DMD), an inherited, X-linked recessive progressive myopathic disability by the Xp21 chromosome's DMD gene. DMD, the most prevalent dystrophinopathy, is characterised by the complete absence of dystrophin from its normal location on the subsarcolemmal surface of the muscle fibre. Males are the primary victims and females are the carriers, however they are very infrequently get minor illness. One in every 5200 males born has Duchenne muscular dystrophy², according to statistics a

large proportion of the genetic abnormalities are deletions with frameshift and point mutations accounting for the rest. Approximately 2/3rd of the cases is familial and the remainder represent new mutations. In the affected families, females are carriers³. The DMD gene consists of 79 exons, encoding at least seven isoforms of the dystrophin protein by use of different promoters that are expressed in different tissues. The actin cytoskeleton and extracellular matrix are connected by an essential protein called dystrophin, which stabilises the highly deformable muscle fibres' muscular membrane. Dystrophin deficiency causes a degenerative process characterised by myofiber necrosis, muscular fibrosis, and a

lack of regeneration ability. It also causes the integrity of the muscle fibres to fail. Over time, fibrosis and fatty replacement, two traditional elements of descriptions of dystrophic muscle, intensify. Clinical symptoms of DMD appear at the age of five. By the ages of 10 to 12, it causes dependence on a wheelchair, and it gets worse from there. Ordinarily, proximal weakness is visible at the moment of presentation. Even at a young age, proximal muscle weakness is evident in difficulty in climbing stairs, hopping or arising from the floor. Gowers' move, which is always present in DMD, serves as a typical illustration of the latter patients stand up from the floor by using their hands to climb up their thighs. Other manifestations of DMD include muscular growth, which Duchenne first referred to as pseudohypertrophy and linked to increased fibrosis and fatty replacement rather than muscle fibre hypertrophy.⁴

Science was a fundamental component of the ancient *Vedas*, which also explained how diseases progress genetically. In the *Rigveda*, genetics had its earliest mention.⁵ As recounted by *Acharya Sushruta*, *Adibala Pravritta Vyadhi* describes the classification of diseases in the *Vyadhisamuddeshiya* chapter of *Sutra Sthana* and mentions the inherited disorders.⁶ According to *Sushruta* and *Dalhana*, *Vatadi Doshas* are the primary factor in the vitiation of *Shukra* and *Shonita*. *Dalhana* further indicated that *Matruja vikaras* arise from the mother's *Shonita* (ovum) and *Pitruja vikaras* from the father's *Shukra* (sperm). Additionally, *Acharya Charka* demonstrates the significance of *Beeja* (genetic material) and clarifies the fundamentals of genetics, stating that "the part of the foetus is affected with morbidity in genes, wholly or partially of which the *Dosha* are vitiated *Beeja* (ovum/sperm), *Bheejabhaga* (Chromosome), and other genetic components⁷."

AIMS & OBJECTIVES

AIMS

To evaluate the efficacy of *Ayurvedic* herbs and *Panchkarma* procedures in the management of DMD.

OBJECTIVES

1. To reduce spasticity in DMD affected children.
2. To improve quality of life of DMD affected children.
3. To assess the efficacy of oral medicine and *panchakarma* procedures on various *Ayurveda* and modern parameters.

MATERIAL AND METHODS

Materials for present manuscript were collected from ancient classics of *Ayurveda* like *Charaka Samhita*, *Sushruta Samhita*, *Astanga Sangraha*, *Ashtanga Hridaya*, *Kashyapa Samhita*, *Harita Samhita*, *Ayurved Pharmacopeia of India (API)* and *Ayurved Formulary of India (AFI)* along with modern textbooks like *OP Ghai*, *Nelson Textbook of Pediatrics*, etc

To explore the neuroprotective activity, nootropic activity, anticonvulsant activity, neuro-regenerative activity, analgesic activity of various *Ayurveda* Herbs and utilization of various *Panchakarma* procedures in the management of DMD various search engines like *Ayush research portal*, *PubMed*, *Medlar*, *Open Med*, *In Med*, *Scopus*, *Google Scholar* were studied from 1960 to till date.

Etiopathology according to Different

Authors

Numerous publications on the *Ayurvedic* etiopathogenesis of Duchenne muscular dystrophy are accessible, some of which have the same description and others which have various descriptions of the DMD's etiopathogenesis. Duchenne muscular dystrophy cannot be directly associated with any one *Ayurvedic* condition, claim *Trivedi Bharvi B et al*. Due to *Beeja Dushti* and

Adibala Pravritta, this illness falls under the *Vata vikara*. *Aatma Karmaja* and *Beeja Dosha* can be used to group the significant causal elements. These elements lead to *Khavaigunya* at *Mamsa Dhatu* levels results in *Vata* vitiation, which impairs *Bhutagni*⁸. According to Ashutosh Chaturvedi et al, pathogenesis is brought on by *Beejabhagaavyava Dusti*, which causes *Vata Parkopa* to take *Sthana samshraya* in *Mamsa* and vitiate the *Medo Dhatu* and depletes them (muscle tissue x-linked progressive degenerative disease). The involvement of the *Dhatvagni mandhya*, which results in *Kshaya*, is clearly indicated in the *Ansha-Ansha Kalpana* of the *Dhatu*s. *Ama* is created as a result of the *Agnimandya* that the *Dhatu*s level causes. *Srotodushti* was defined by *Madhavkara* as a kind of *Ama*. While *Srotorodha* causes hypertrophy in the targeted area, it also exhibits *Vata Parkopa* before depletion, which is caused by *Vata*. The progressive wasting and necrosis of muscle fibres are in fact caused by this intricate diversity of pathophysiology.⁹ Dr. Mukesh Meena has described the importance of *Dhatu Avarna* in the aetiology of DMD, stating that vitiated *Vata Dosha* is a significant factor in muscular dystrophy that is created after a genetic mutation occurs because the *Vata Dosha* is more aggravated and obstructed (*Avrita*) by *Rasa*, *Raktadi Dhatu*s, and as a result, mainly *Mamsa* and *Meda* are affected because they make up the majority of our body (as the skeleton and muscles, which are the primary sites of *Vata Dosha*, support the body of a human being). This leads to the development of *Meda* and *Mamsa Dhatu Kshaya* as well as *Virddhi* (degeneration and regeneration of muscular fibres, especially in the calf muscles, which causes tongue hypertrophy and pseudohypertrophy in the calf muscles) occurs, by which nervous tissues supplying to those affected parts lacks proper nutrition and gets deactivated. This pathophysiology leads to Muscular Disorders¹⁰. A different correlation of

Paurasadini Jataharini (in which delivered child dies before 16 years of age) one of the *Asadhya Jataharini* as mentioned in *Kashyapa Samhita* can also be correlated with DMD¹¹.

CLINICAL MANIFESTATION

Infant males with DMD are rarely symptomatic at birth or in the first few months of life, though some are moderately hypotonic. Early gross motor abilities, like as rolling over, sitting, and standing, are typically attained at the right ages or may be somewhat delayed. Because facial muscle weakening occurs later in life, distinct facies are not an early feature; nevertheless, a "transverse" or horizontal smile may be noticed in later childhood. Walking is usually accomplished by the age of 12 months, but hip girdle weakness can be noticed as early as the second year. When standing, toddlers may adopt a lordotic posture to compensate for gluteal weakness. An early Gowers' sign can be noticed as early as 3 years old, although it is almost always visible by 5 or 6 years old. A Trendelenburg (waddling) gait is also common at this time. Toddlers commonly show with delayed walking, frequent falling, toe walking, and difficulty running or walking upstairs, developmental delay, and, less frequently, malignant hyperthermia following anesthesia. The amount of time a patient with DMD can walk varies substantially. Patients may experience greater ambulation difficulties as a result of proximal lower extremity weakness, which is exacerbated by growing ankle contractures and toe walking. The age of complete ambulation ranges between 10 and 14 years. With the introduction of clinical care guidelines suggesting the use of corticosteroids (e.g., prednisone or deflazacort) in boys with DMD, the age at which loss of independent ambulation occurs has increased over time. Most people can walk until the age of 12 years with orthotic bracing, physiotherapy, and sometimes minimal surgery (Achilles tendon lengthening).

Upper extremity strength falls more as the condition develops in the adolescent years, and patients may experience increased difficulty putting hands to mouth independently, tiredness with writing, and developing contractures, including in the hands and fingers. Involvement of respiratory muscles typically shows as a weak and inefficient cough, recurrent lung infections, and a decline in respiratory reserve. Snoring and sleep apnea are common early pulmonary symptoms.

Calves enlargement (pseudohypertrophy) and thigh muscle atrophy are common symptoms. The enlargement is generated by hypertrophy of some muscle fibers, fat infiltration of the muscle, and collagen proliferation. The tongue is the second most common location of muscular enlargement after the calves, followed by forearm muscles.

Myopathy, including persistent tachycardia, myocardial fibrosis, and cardiomyopathy, occurs in a majority of patients with DMD. In patients with DMD, the progression of cardiomyopathy typically occurs after loss of independent ambulation.

Although approximately 20-30% of patients have an IQ of 70 or higher, intellectual decline affects the majority of patients. The severity of intellectual disability varies, with some individuals requiring specialized education and having difficulties reading and writing, while others may only require some additional tutoring or assistance. Epilepsy is significantly

more common in DMD patients than in the overall juvenile population, although it is not a distinguishing trait. Some patients may develop autism-like behavior. Dystrophin is expressed in the brain, retina, striated and cardiac muscle, however at a lesser level in the brain than in the muscle¹².

MANAGEMENT

The management of DMD is focused on providing symptomatic relief. The general management of *Vatavyadhi* can be adopted. The line of treatment of *Vatavyadhi* comprises of *Snehana*, *Swedana*, *Mriduvirechana* (~mild purgation) and *Basti* (enema) along with other oral medication¹³. Likewise, *Virechana*, *Niruha Basti* (Medicated enema mainly comprises of decoction of medicinal plant for *Vatavyadhi* etc) and *Shamana* (palliative) therapies are indicated in the management of *Mansagata Vata*.¹⁴ For *Dhatukshayajanya Vatavyadhi*, the main treatment includes *Snehana*, *Swedana*, *Basti* along with *Ayurvedic* oral medications. Thus following the textual line of management *Abhyanga*, *Parisheka Swedana*, *Shalishashtika Pinda Swedana*, *Udwartana* and *Basti* can be planned along with oral medication. For *Avrita Vata Rasayana* are indicated.¹⁵

The oral medication of *Ayurveda* which contains properties like muscle strengthen, analgesic, anti-spasmodic, nootropic viz. some of them are described as below.

TABLE 1. DEPICTING ACTION OF SINGLE HERBS

Sr. no.	Ayurvedic herb	Latin name	Action	Reference
1.	<i>Ashwagandha</i>	<i>Withania somnifera</i>	Muscle strengthens	Wankhede S et.al
			Analgesic	Twaij HAA et.al
			Cardiorespiratory strengthen	Choudhary, D et.al
2.	<i>Shatavari</i>	<i>Asparagus racemosus</i>	Analgesic	Karmakar UK et.al
			Antihepatotoxic	Zhu X et.al, Muruganadan S et.al
3.	<i>Amalaki</i>	<i>Emblica officinalis</i>	Muscle strengthens	Ekta Singh et.al
			Cardio protective	Chatterjee A et.al, Bhattacharya SK et.al
			Hepato protective	Srirama R et.al
4.	<i>Gokshura</i>	<i>Tribulus terrestris</i>	Analgesic	Heidari MR et.al
			Cardio protective	Phillips OA et.al
5.	<i>Guduchi</i>	<i>Tinospora cordifolia</i>	Hepato protective	Karkal YR et.al
6.	<i>Kebuk</i>	<i>Costus speciosus</i>	Analgesic	K. Binny et.al
			Anti spasmodic	Bhattacharya S. K et al
7.	<i>Pippali</i>	<i>Piper longum</i>	Analgesic	Vedhanayaki G et.al
			Respiratory stimulant	Kulshresta VK et.al
8.	<i>Shankhpushpi</i>		Nootropic	Kaushik R et.al

		Anti-convulsant	Siddiqui N. A et.al
		Analgesic	Agarwal P et.al
		Spasmolytic	Amin H et.al

PANCHKARMA PROCEDURES

Panchakarma therapy primarily aims at cleansing the body of its accumulated impurities, toxins or stagnant *Malas* and nourishing the body tissues. In person, who has undergone the purificatory regimen, the digestive power increases, his disorders disappear and his health returns to normal and the individual acquires strength, vigor and virility¹⁶.

ABHYANGA

Abhyanga is a process by which the body surface i.e. the integument undergoes manual pressure by various techniques and various substances to provide not only relaxation of the body but also the pacification of several types of diseases¹⁷. *Abhyanga* with *Bala Ashwagandha Taila*, *Kshira Bala Taila* etc. is *Vata* pacifying oil can be used to nourish and strengthen the muscles. The name "*Ashwagandha*" gives reference to the strength of a horse and is known to rejuvenate both the muscular and nervous systems. "*Bala*" literally means "strength," inferring the herb's potential to build muscle mass and provide energy. Also, base of all *Taila* is *Tila Taila* which pacifies *Vata* and also penetrates deep into the skin. Sesame oil pacifies *Vata dosha*¹⁸.

SWEDANA

Svedana may broadly be defined as a process of external heating by which the body is induced to produce sweat to mitigate several localised and systematic disorders. The main function achieved by *Svedana* is rise in temperature, which is responsible for increased metabolic activity, increased blood flow, stimulation of neural receptors in the skin or tissues¹⁹.

Sweating treatment is administered as a standard treatment for diseases of *Vata* and *Kapha* treatment. In this diseases *Snigdha-*

Ruksha Sweda is administered. After administration of *Swedan* there is complete recovery from pain, stiffness and heaviness of the body²⁰.

Parisheka Sweda (sprinkling) is indicated in *Vata/Vata-Kapha* disorders. Here, hot liquids are sprinkled over the body parts or whole body. Pots with small holes at the bottom, bamboo pipes, *nala* (phragmites *karka trin* etc.) are to be filled up with lukewarm decoction (*Kashaya*) of root etc. of *Vata/Vata-Kapha Hara* drugs²¹.

SHASHTIK SHALI PINDA SWEDANA

Acharya Charak has classified the *Sweda* into two groups one is *Sagni* & another one is *Niragni*.²² In *Ayurveda*, *Swedana* procedures are done in the form of *Pinda Sweda* (sudation using bolus) in which *Kizhi/Pinda/Pottali* (bolus) containing drugs is used for *ekanga* and *sarwanga Sweda*. *Shashitka Shali Pinda Sweda* is a variety of *Snigdha Sankara Sweda* which comes under *Sagni Sweda*²³ In this procedure we use *Shashti* rice (a special type of rice harvested in 60 days) processed in herbal decoctions like *Bala mula* etc. and milk tied in a bolus to rub against the whole body or afflicted part of the body so as to provide heat to the pain afflicted joints, muscles or body parts. It is a strengthening therapy. It gives nutrition to the tissues which are undergoing depletion and degeneration. It is a time-tested treatment administered to those ailing from musculoskeletal and neuromuscular diseases. It is basically a strengthening and nutritious treatment. It also has an extraordinary relaxing and analgesic effect²⁴.

UDAVARTANA

Application of medicated oil as indicated and dusting of herbal powder on the body followed by massage with some pressure in the direction opposite to that of hair is the main method

adopted in *Udvardana*²⁵. *Udvardana* with *Kolkulattadi Churna* is indicated in neurological condition and also in accumulated subcutaneous fat²⁶. *Udvardana* by *Yava, Masa* coarse powder also very effective to treat hypertrophy of calf muscles and strength of muscles due to its *Medo-Kaphar*²⁷. properties and stimulates nerve ending, relax muscles and relieves pain²⁸. *Udwardana* opens the minute channels and improves blood as well as lymphatic circulation. *Udwardana* is *Kapha, Vata Hara* and removes *Aavarana* or *Srotorodha*. It provides a platform for further procedures like *Abhyanga, Swedana* and *Basti*. *Udwardana* is beneficial in reducing the spasticity.³⁰

BASTI

Basti, the prime treatment in *Shodhana* is considered as one of the most important treatments for many diseases according to associated with arthritis and other painful conditions as mentioned in *Ayurvedic* classics. It is the best treatment modality for all types of *Vata* diseases (neurological disorders). The type of *Basti* where decoction is the major part is called as *Asthapana Basti* or *Niruha Basti*. *Basti* in which major part is oil or other *Sneha* (oleaginous substance) is called as *Anuvasana Basti*²⁹. *Anuvasana Basti*, after the use of *Niruha Basti* gives soothing effect on the purified *Marga* or *Srotas* (Micro Channels) and increases the body lustre and strength. Oil (*Tilodbham*) is the best remedy for *Vatarogi* with its *Snigdha, Guru* and *Ushna* Properties. It gives instant tranquilizing effect and potentiates i.e. *Veerya, Bala, Varna, Agni* and *Pushti*³⁰. The *Basti* which can be administered at any time and it increases lifespan of the patient, called *Yapana Basti*^{31,32}. It is made of *Mridu* drugs like milk, Ghee and *Mamsarasa* etc. It is specially indicated for children and elder persons.

DISCUSSION

Breaking down the pathogenesis of the disease, means removing the *Srotorodha* and pacifying *Vatadosha*, will be the main aim of this treatment. This study reports that *Ashwagandha* supplementation is associated with significant increases in muscle mass³³. Monitoring creatine kinase in the current investigation revealed that *Ashwagandha* administration resulted in faster recovery from muscle injury. The sooner recovery could be due to a variety of mechanisms, or more likely their synergistic effects, as mediated by the various extract components, such as antioxidant effects to combat free radical damage at both the muscle and central nervous system levels, anti-inflammatory and analgesic effects, and lactic acid and blood urea nitrogen reduction^{34,35,36}. *Ashwagandha* (1000 mg/kg/oral) demonstrated considerable analgesic effect in a rat with thermal analgesia elicited by the hot plate method. At the second hour of dosing, *Ashwagandha* had a maximal analgesic effect of 78.03 percent. The role of pain mediators such as prostaglandins and 5-hydroxytryptamine in *Ashwagandha*'s analgesic action was investigated. Pretreatment with paracetamol (100 mg/kg, ip) and cyproheptadine (10 mg/kg, ip) was used. Cyproheptadine considerably increased the analgesic efficacy of *Ashwagandha*, whereas paracetamol had little effect, indicating that serotonin, rather than prostaglandins, is involved in the analgesic activity of *Ashwagandha*³⁷. Alcoholic extract from the roots of *A. racemosus* has been demonstrated to dramatically lower the elevated levels of alanine transaminase, aspartate transaminase, and alkaline phosphate in CCl₄-induced hepatic damage in rats^{38,39}, demonstrating the plant's potential to be antihepatotoxic. Using a mouse model of acetic acid-induced writhing, the *A. racemosus* crude ethanol extract's analgesic efficacy was tested. In acetic acid induced writhing in mice, the ethanol extract exhibited significant inhibition of writhing

reflex 67.47% ($P < 0.01$) at dose of 500 mg/kg body weight⁴⁰. Muscle strengthen activity of *Embilica officinalis* has been proven as it enhances protein synthesis, which is why it is good for strengthening muscles and building lean muscle mass⁴¹. Analgesic activities of *Tribulus terrestris* were studied in male mice using formalin and tail flick test. The study indicated that the methanolic extract of TT at a dose of 100 mg/kg produced analgesic effect⁴². Tribulosin treatment resulted in a significant reduction of malondialdehyde, aspartate transaminases, creatine kinase, lactate dehydrogenase activity, and myocardial apoptosis rate⁴³. which proves its cardioprotective activity. A clinical study has shown that *Guduchi* plays an important role in normalization of altered liver functions (ALT, AST)⁴⁴. The antihepatotoxic activity of *T. cordifolia* has been demonstrated in CCl₄ induced liver damage, normalising liver function as assessed by morphological, biochemical (SGPT, SGOT, serum alkaline phosphatase, serum bilirubin) and functional (pentobarbitone sleep time) tests. *T. cordifolia* revealed hepatoprotective action in goats⁴⁵. Analgesic effect of *Costus speciosus* was evaluated by using acetic acid induced writhing and Eddy's hot plate. Results revealed that methanol extracts of *Costus speciosus* has significant anti-inflammatory and analgesic activities⁴⁶. A different extract of *C. speciosus* has been tested on the ileum of a guinea pig by Banerji et al. (1982). The results obtained proved that the plant has moderate levels of spasmolytic activity⁴⁷. Petroleum ether extract of *P. longum* produced respiratory stimulation in smaller dose^{48,49}. Pentazocine and ibuprofen were used as pharmacological controls when *P. longum* root was used for opioid type analgesia using the rat tail-flick method and for NSAID type analgesia using the acetic-acid writhing method. Mice and rats were given an aqueous suspension of *P. longum* root powder. The study demonstrated that *P. longum* root has high NSAID type analgesic action but modest

opioid analgesic activity⁵⁰. For anti-convulsant effectiveness against strychnine-induced as well as pentylenetetrazol (PTZ)-induced convulsive seizures in several animal models, the chloroform, ethanol, and aqueous extracts of CP have been tested. Strychnine and PTZ-induced clonic convulsions were prevented by *Convolvulus Prostratus* extracts at a concentration of 500 mg/Kg with statistically significant ($p < 0.001$) results (Ratha and Mishra, 2012; Siddiqui et al., 2014)⁵¹. The fundamental mechanism behind such evident anti-convulsing activity of CP might be the presence of coumarins and triterpenoids⁵². When tested in hot plate method and tail-flick assays in rats, the ethanolic extract of CP at dose 750 mg/Kg exhibited statistically significant analgesic effect as compared to the standard analgesics like morphine sulphate⁵³. By preventing the synthesis of the cyclooxygenase enzyme and prostaglandins, flavonoids, volatile oils, alkaloids, polyphenols, and organic acids collectively contribute to this apparent analgesic function⁵⁴. The CP ethanolic extract has exhibited spasmolytic activity in isolated rabbit ileum, isolated rat uterus, intact intestine and tracheal muscles of dog⁵⁵. Convolvine, a particular alkaloid found in this CP plant, is primarily responsible for this anti-spasmodic activity by inhibiting the generation of acetylcholine⁵⁶.

The oil applied to the skin penetrates the epidermis through the stratum corneum. It enters the systemic circulation through the cutaneous circulation and lymphatics. It improves blood circulation in the area where Abhyanga is applied. Furthermore, it stimulates venous return to the main bloodstream by specific strokes directed at the heart. It contributes to increased cardiac output. Abhyanga promotes venous return by exerting a direct mechanical and reflexive action on blood vessels. Oil bath softens the body, reduces Kapha and Vata aggravation⁵⁷.

The heating impact of *Swedana* promotes circulation in the area. There are arteries that supply the skin from dense networks of arterioles and capillaries that branch to all dermal tissues and nourish the stratum germinativum of the epidermis by dilatation of the arterioles, which does not receive direct blood flow. This raises the skin's temperature and is also responsible for the amount of heat lost through radiation and sweating. It provides more oxygen, nutrients, polymorphs, and endorphins to the damaged area, which aids in the healing of the local pathology as well as overall healing. Effect on sensory nerve endings helps in reducing pain. Pain may be subsided due to removal of waste products from the tissue by the vasodilatation provided by heat.

Swedana thus plays an important role in *Panchkarma*. *Swedana* by its attributes *Ushna* (hot), *Tikshna* (sharp), *Drava* (liquid), *Snigdha* (unctuous), *Ruksha* (rough) *Sukshma* (subtle), *Sara* (fluid) *Sthira* (stable) and *Guru* (heavy) liquefies the morbid materials in the minutes channels in the body which has undergone properly Oleation therapy and then by its circulatory effects help them to come into the concerned *Koshtha*⁵⁸.

Shashtik Shali Pinda Sweda performs the function of *Brimhana* and provides *Dhatu Poshana* (nourishment). *Ushna Guna* stimulates the sympathetic nervous system and causes vasodilation. *Sara* as well as *Sukshama Guna* liquifies the *lina Dosha* and then these *Doshas* are expelled out through the micropores. It plays an important role in the management of muscular dystrophy^{59,60}. Heating the skin has been demonstrated to produce a decrease in gamma activity. With a decrease in gamma activity, the stretch on the muscle spindle would be less, thus reducing afferent firing from the spindle. This indirect method ultimately results in decreased alpha motor neuron firing and thus less muscle spasm. Elevating muscle temperature can also alter strength and endurance. Heating

can result in decreased joint stiffness and increased tissue extensibility, thus facilitating ease of motion and gains in range of movements⁶¹.

The active principle from *Basti Dravyas* inserted into the sigmoid colon via anus is absorbed through rectal veins and via portal vein it spreads to whole body and produces its effect. Moreover, it certainly affects the enteric nervous system and through neurotransmitters present in the enteric system it acts on the brain via spinal tract and produced its effect on whole body⁶².

Ghee, milk, and other natural oils commonly used in *Bastis* include short and medium chain fatty acids and they do not require bile salt, pancreatic lipase, or micelle formation for absorption⁶³. These two fatty acids are absorbed via the colon wall and furthermore, the other ingredients in *Basti* may be metabolised by intestinal bacteria, creating short chain fatty acids such as butyric, propionic, ethanoic, and valeric acids⁶⁴. SCFA is quickly absorbed and increases intestinal sodium and fluid absorption⁶⁵. The *Niruha* therapy, in addition to its therapeutic benefits, has a cleansing impact on the colon. Cleaning the colon may reduce the quantity of toxins in the cecum, making them easier to remove. The therapeutic result is increased muscular tone, which promotes peristaltic movement and increases nutrient absorption from the cecum and ascending colon while reducing toxic waste absorption. The *Niruha* by its cleaning action minimize the toxin load in the large intestine resulting in reduces burden on the liver, allowing eliminative organs to function optimally⁶⁶.

CONCLUSION

Duchenne muscle dystrophy is a neuromuscular condition. Only *Ayurveda* offers effective treatment for DMD. After comparison of different authors, it is concluded that DMD is *Adibala Pravritta Vyadhi* and there is certainly involvement of

Beejabhagaavyava (Genes) leading to vitiation of *Vata* which causes *Bhutagni* and *Dhatvagni* impairment which further lead to *Aama* instead of proper *Mamsadhatu* and *Shrotorodha* also produces hypertrophy in a particular region *Ayurvedic* oral medication, combined with *Panchkarma* treatment, improves quality of life, extends lifespan, and delays advancement of cardiac disease. But these certain ailments required long-term treatment.

Declaration by Authors

Ethical Approval: Not Applicable

Acknowledgement: None

Source of Funding: None

Conflict of Interest: The authors declare no conflict of interest.

REFERENCES

1. Harsh Mohan. (2015). Textbook of Pathology (7th ed.). New Delhi: Jaypee the Health Science Publishers. pp. 848-850.
2. Anthony A Amato, Robert H. Brown(2016) Harrison's Principles of Internal Medicine, 19th edition, New Delhi, McGraw Hill Education (India)Private Limited, vol 3, 462e 1-8
3. Anthony, D. C., Frosch, M. P., & Girolami, U. D. (2011). In V. Kumar, A. K. Abbas, N. Fausto, J. C. Aster (Eds.), Robbins and Cotran Pathologic Basis of Disease (8th ed., pp. 1268). South Asia.
4. Flanigan, K. M. (2014). In D. Hilton-Jones & M. R. Turber (Eds.), Oxford Textbook of Neuromuscular Disorders (pp. 2076). Oxford University Press.
5. Jamison, S. W. (Translator & Contributor), Brereton, J. P. (Translator & Contributor). (2014). The Earliest Religious Poetry of India: Mandala 1, Kaksivant dairghatamasa/19 (Vol. 1). United States of America: Oxford University Press, South East Research.
6. Prasad, V. V. (Ed.). (2002). Sushruta Samhita, Sutra Sthana, Vyadhi Samuddeshiya Adhyaya, Chapter No. 24, Verse No. 4-5 (1st ed.). New Delhi: Rashtriya Ayurveda Vidhya Peeth Publication. p. 242.
7. Vaidya Yadavji Trikamji Acharya (Ed.). (2004). Charak Samhita of Maharshi Agnivesh, Sharira Sthana, Mahati, Chapter 4/30(1st edition reprint). Varanasi: Chaukhambha Sanskrit Sansthan. p. 877.
8. Trivedi, B. B., Mer, R., & Viramgami, J. (2017). Conceptual Aspect of Duchenne Muscular Dystrophy. International Journal of Ayurveda and Pharma Research, 5(4), 42-48.
9. Chaturvedi, A., et al. (2013). Role of Panchakarma in Duchenne Muscular Dystrophy. International Journal of Research in Ayurveda and Pharmacy, 4(2), 272-275.
10. Meena, M. K. (2017). A Review Article on Muscular Dystrophy and Its Management Through Ayurveda. World Journal of Pharmaceutical Research, 6(11), 378-388.
11. Srivastava, N., et al. (2018). Recent Advancement in Treatment of Duchenne Muscular Dystrophy (DMD) in Ayurveda. International Journal of Research and Analytical Reviews, 5(3), July–Sept, 934-937.
12. Robert M Kliegman, Joseph St (2020) Nelson textbook of pediatrics, 21st edition, Geme Philadelphia, Elsevier 3281
13. Shukla, V., & Tripathi, R. D. (2017). Charaka Samhita, Volume 2, Chikitsasthan - 28/75-84. Delhi: Chaukhambha Sanskrit Pratishthan. pp. 701-702.
14. Shukla, V., & Tripathi, R. D. (2017). Charaka Samhita, Volume 2, Chikitsasthan - 28/93. Delhi: Chaukhambha Sanskrit Pratishthan. p. 704.
15. Shukla, V., & Tripathi, R. D. (2017). Charaka Samhita, Volume 2, Chikitsasthan - 28/241. Delhi: Chaukhambha Sanskrit Pratishthan. p. 723.
16. Sharma, P. V. (2001). Charaka Samhita, 7th Edition, Volume 1, Sutra Sthan Snehadhyaya. Varanasi: Chaukhambha Orientalia. p. 112.
17. Kar, P. K. (2013). Mechanism of Panchakarma and Its Module of Investigation (1st ed.). Chaukhamba Sanskrit Pratishthan. pp. 30-33.
18. Vidyanath, R., Nishteswar, K. (2006). Sahasra Yoga, Taila Yoga Prakarana-3/2. Varanasi: Chaukhambha Sanskrit Pratishthan. p. 110.
19. Kar, P. K. (2013). Mechanism of Panchakarma and Its Module of Investigation (1st ed.). Chaukhamba Sanskrit Pratishthan. pp. 34-41.
20. Agnivesha, Charaka Samhita (14th Su.). Edited by Prof. Priyavrata Sharma. (2017). Volume 1, Sutrasthan 14. Delhi: Chaukhambha Sanskrit Pratishthan. p. 220.

21. Agnivesha, Charaka Samhita. Edited by Vaidya Yadavji Trikamji Acharya. (Sutra Sthana; 14:44). Varanasi: Chaukhambha Krishnadas Academy. pp. 90.
22. 23.Kushwaha, V. H. C. S. (Ed.). (2005) Charak Samhita. Sutra Sthana, Chapter 14/8. Varanasi, India: Chaukhambha Orientalia.
23. Kushwaha, V. H. C. S. (Ed.). (2005) Charak Samhita. Sutra Sthana, Chapter 14/39-41. Varanasi, India: Chaukhambha Orientalia.
24. Mahor, B., et al. (2023). Shashtika Shali Pinda Sweda in the Management of Cerebral Palsy: A Critical Review. *Nat Ayurvedic Med*, 7(1), 000387.
25. Kasture, H. S. (Year). *Ayurvediye Panchakarma Vigyana*. (p. 88). Shri Baidyanath Ayurved Bhawan Ltd.
26. Kushwaha V.H.C.S(Ed) (2005) Charaka Samhita. Sutrasthana, Chapter 3/18 Varanasi,India: Chaukhambha Orientalia.
27. Vagbhata. (2011). *Astanga Hridaya with Arundatta* (In: Kunte A., Ed. *Sarvangasundari, Commentary. Reprint Ed.*). Varanasi: Chaukhambha Orientalia. pp. 223-225.
28. Lavekar, G. S. (2009). *A Practical Handbook of Panchakarma Procedures*. New Delhi: CCRAS. p. 60.
29. Sushruta. (1979). *Sushruta Samhita* (Shastri A., Ed.). (5th ed., Ch. 35, Ver 18). Varanasi: Chaukhambha Orientalia. p. 154.
30. Kasinath Shastri. (2012). *Charaka Samhita: Part-II, Siddhisasthana, Kalpanasiddhi-1/29-30*. Varanasi: Chaukhambha Bharati Academy. p. 969.
31. Kasinath Shastri. (2012). *Charaka Samhita: Part-II, Siddhisasthana, Uttarbastisiddhiadhy-12/16-1*. Varanasi: Chaukhambha Bharati Academy. p. 1091.
32. Kasinath Shastri. (2012). *Charaka Samhita: Part-II, Siddhisasthana, Uttarbastisiddhiadhy-12/8*. Varanasi: Chaukhambha Bharati Academy.
33. Wankhede, S., Langade, D., Joshi, K., Sinha, S. R., & Bhattacharyya, S. (2015). Examining the effect of *Withania somnifera* supplementation on muscle strength and recovery: a randomized controlled trial. *Journal of the International Society of Sports Nutrition*, 12, 43. doi: 10.1186/s12970-015-0104-9. PubMed PMID: 26609282; PubMed Central PMCID: PMC4658772.
34. Mishra, L. C., Singh, B. B., & Dagenais, S. (2000). Scientific basis for the therapeutic use of *Withania somnifera* (ashwagandha): a review. *Alternative Medicine Review*, 5, 334–346.
35. Vyas, V. K., Bhandari, P., & Patidar, R. (2011). A Comprehensive Review on *Withania somnifera* Dunal. *Journal of Natural Remedies*, 11, 1–13.
36. Singh, G., Sharma, P. K., Dudhe, R., & Singh, S. (2010). Biological activities of *Withania somnifera*. *Annals of Biological Research*, 1, 56–63.
37. Mazen, E. S., Pavelescu, M., & Grigorescu, E. (1990). Testing of Analgesic Activity of Dichloromethanic and Methanolic Extract from *Withania-Somnifera* Roots. *Revista Medico-Chirurgicala Societatii de Medici si Naturalisti din Iasi*, 94(3–4), 603–605. Contributions to the Pharmacodynamic Study of roots of *Withania somnifera* Dun Species Of Pakistani origin.
38. Zhu, X., Zhang, W., Zhao, J., Wang, J., & Qu, W. (2010). Hypolipidaemic and hepatoprotective effects of ethanolic and aqueous extracts from *Asparagus officinalis* L. by-products in mice fed a high-fat diet. *Journal of the Science of Food and Agriculture*, 90(7), 1129–1135.
39. Muruganadan, S., Garg, H., Lal, J., Chandra, S., & Kumar, D. (2000). Studies on the immunostimulant and antihepatotoxic activities of *Asparagus racemosus* root extract. *Journal of Medicinal and Aromatic Plant Sciences*, 22, 49–52.
40. Karmakar, U. K., Sadhu, S. K., Biswas, S. K., Chowdhury, A., Shill, M. C., & Das, J. (2012). Cytotoxicity, analgesic, and antidiarrhoeal activities of *Asparagus racemosus*. *Journal of Applied Sciences*, 12, 581–586.
41. Singh, E., Sharma, S., Pareek, A., Dwivedi, J., Yadav, S., & Sharma, S. (2011). Phytochemistry, traditional uses and cancer chemopreventive activity of *Amla* (*Phyllanthus emblica*): The Sustainer. *Journal of Applied Pharmaceutical Science*, 02(01), 176-183.
42. Heidari, M. R., Mehrabani, M., Pardakhty, A., Khazaeli, P., Zahedi, M. J., Yakhchali, M., et al. (2007). The analgesic effect of *Tribulus terrestris* extract and comparison of gastric

- ulcerogenicity of the extract with indomethacin in animal experiments. *Annals of the New York Academy of Sciences*, 1095, 418-427.
43. Phillips, O. A., Mathew, K. T., & Oriowo, M. A. (2006). Antihypertensive and vasodilator effects of methanolic and aqueous extracts of *Tribulus terrestris* in rats. *Journal of Ethnopharmacology*, 104, 351–355.
 44. Karkal, Y. R., & Bairy, L. K. (2007). Safety of aqueous of *Tinospora cordifolia* (Tc) in healthy volunteers: A double-blind randomized placebo-controlled study. *Iranian Journal of Pharmacology and Therapeutics*, 6, 59–61.
 45. Nagarkatti, D. S., Rege, N. N., Desai, N. K., & Dahanukar, S. A. (1994). Modulation of Kupffer cell activity by *Tinospora cordifolia* in liver damage. *Journal of Postgraduate Medicine*, 40, 65–67.
 46. Pawar1, P. R., & Pawar2, P. R. (2014). *Costus speciosus*: An Important Medicinal Plant. *International Journal of Science and Research (IJSR)**, *3*(7), Paper ID: 02014885. ISSN (Online): 2319-7064.
 47. Bhattacharya, S. K., Parikh, A. K., Debnath, P. K., Pandey, V. B., & Neogy, N. C. (1973). Title of the article. *Journal of Research in Indian Medicine*, 8(1), 10-19.
 48. Kulshresta, V. K., Singh, N., Shrivastava, R. K., & Kohli, R. P. (1969). A study of the central stimulant effect of *Piper longum*. *Indian Journal of Pharmacology*, 1(2), 8-10.
 49. Kulshresta, V. K., Singh, N., Shrivastava, R. K., Kohli, R. P., & Rastogi, S. K. (1971). A study of central stimulant activity of *Piper longum*. *Journal of Research in Indian Medicine*, 6(1), 17-19.
 50. Vedhanayaki, G., Shastri, G. V., & Kuruvilla, A. (2003). Analgesic activity of *Piper longum* Linn, Root. *Indian Journal of Experimental Biology*, 41(6), 649-651.
 51. Siddiqui, N. A., Ahmad, N., Musthaq, N., Chattopadhyaya, I., Kumria, R., & Gupta, S. (2014). Neuropharmacological profile of extracts of aerial parts of *Convolvulus pluricaulis Choisy* in mice model. *Open Neurology Journal*, 8, 11–14. doi:10.2174/1874205X01408010011
 52. Quintans Júnior, L. J., Almeida, J. R., Lima, J. T., Nunes, X. P., Siqueira, J. S., Oliveira, L. E. G. D., et al. (2008). Plants with anticonvulsant properties: a review. *Revista Brasileira de Farmacognosia*, 18, 798–819. doi:10.1590/S0102-695X2008000500026
 53. Agarwal, P., Sharma, B., & Alok, S. (2014). Screening of anti-inflammatory and anti-analgesic activity of *Convolvulus pluricaulis Choisy*. *International Journal of Pharmaceutical Sciences and Research*, 5, 2458–2463. doi:10.13040/IJPSR.0975-8232.5(6).2458-63
 54. Aleebrahim-Dehkordy, E., Tamadon, M. R., Nasri, H., Baradaran, A., Nasri, P., & Beigrezaei, S. (2017). Review of possible mechanisms of the analgesic effect of herbs and herbal active ingredients. *Journal of Young Pharmacists*, 9, 303–306. doi:10.5530/jyp.2017.9.60
 55. Barar, F. S., & Sharma, V. N. (1965). Preliminary pharmacological studies on *Convolvulus pluricaulis Choisy* – an Indian indigenous herb. *Indian Journal of Physiology and Pharmacology*, 9, 99–102.
 56. Amin, H., Sharma, R., Vyas, M., Prajapati, P. K., & Dhiman, K. (2014). *Shankhapushpi (Convolvulus pluricaulis Choisy)*: Validation of the Ayurvedic therapeutic claims through contemporary studies. *International Journal of Green Pharmacy*, 8, 193–200. doi:10.4103/0973-8258.142666
 57. Murthy, K. R. S. (2005). *Susruta Samhita* (2nd ed.). Varanasi: Chaukhamba Orientalia. Su.Ci 24/30.
 58. Kar, P. K. (2013). Mechanism of Panchakarma and Its Module of Investigation. (1st ed.). Chaukhamba Sanskrit Pratishtan. pp. 34-41.
 59. Meena, M. K. (2017). A review article on muscular dystrophy and its management through Ayurveda. *World Journal of Pharmaceutical Research*, 6(11), 378–388.
 60. Mudadla, S., & Injamuri, R. (2015). Ayurvedic management of Duchenne Muscular Dystrophy. *Ayushdhara*, 2(3), 179–183.
 61. Mudadla, S., & Injamuri, R. (2015). Ayurvedic Management of Duchenne Muscular Dystrophy. *AYUSHDHARA*, 2(3), 188-193.
 62. Chaurasia, B. D. (2010). *Human Anatomy, Volume 2, Chapter 33: Rectum and Anal Canal*. New Delhi, India: CBS Publishers and Distributors.
 63. Vasudevan, D. M., & Sreekumari, S. (2005). *Textbook of Biochemistry (For Medical Students)* (4th ed.). p. 153.

64. Wong, J. M., de Souza, R., Kendall, C. W., Emam, A., & Jenkins, D. J. (2006). Colonic health: fermentation and short-chain fatty acids. *Journal of Clinical Gastroenterology*, 40(3), 235-243. doi:10.1097/00004836-200603000-00015.
65. Kasper, D. L., & Braunwald, E. (2005). *Harrison's Principles of Internal Medicine* (16th ed.). McGraw-Hill Professional Publishing. p. 1767.
66. Kar, P. K. (2013). Mechanism of Panchakarma and Its Module of Investigation (1st ed.). Chaukhamba Sanskrit Pratishtan. pp. 88-89.

How to cite this article: Minaj Dosani, Harish Kumar Singhal. An ayurvedic approach in muscular dystrophy in children. *Int J Health Sci Res.* 2024; 14(3):105-116. DOI: <https://doi.org/10.52403/ijhsr.20240318>
