Case Report

www.ijrap.net

(ISSN Online: 2229–3566, ISSN Print: 2277–4343)

AYURVEDA TREATMENT APPROACH FOR LIMB-GIRDLE MUSCULAR DYSTROPHY: A CASE REPORT ON SIGNIFICANT SYMPTOMATIC AND BIOCHEMICAL IMPROVEMENTS

Vishwanath Wasedar [1], Bhagyesh Pangam [2]*, Aparna Siddhaye [3]

[1] Associate Professor, Department of Panchakarma, KAHERS's Shri B. M. Kankanwadi Ayurvedic Medical College and Hospital, Belgaum, Karnataka, India

[2] Assistant Professor, Department of Panchakarma, Shri Vasantadada Patil Ayurvedic Medical College and Hospital, Sangli, Maharashtra, India

[3] Medical Officer, Department of Kayachikitsa, All India Institute of Ayurveda, Dhargal, Pernem, Goa, India

Received on: 20/4/25 Accepted on: 02/6/25

***Corresponding author**
E-mail: pangam.bhagyesh@gmail.com

DOI: 10.7897/2277-4343.16395

ABSTRACT

Limb-Girdle Muscular Dystrophy (LGMD) is a genetically heterogeneous, progressive neuromuscular disorder under Muscular Dystrophies (MD), marked by the degeneration and weakening of limb-girdle muscles, causing significant functional disability. Despite advances in modern medicine, treatment remains palliative, with no definitive cure or disease-modifying therapy. Current pharmacological and rehabilitative approaches offer limited symptomatic relief, without halting disease progression or restoring muscle function. This case report presents a 49-year-old male diagnosed with LGMD, managed through a comprehensive Ayurvedic treatment plan including oral medications and Panchakarma therapies. Assessment focused on pain, walking ability, muscle strength, reflexes, and specialized neurological parameters. Investigations such as Serum Creatine Phosphatase (CPK), CPK-MB levels, and electromyography (EMG) were used to evaluate muscle and nerve function. According to Ayurveda, this condition falls under Vata vyadhi and is explained through adhi bala pravritta vyadhi (congenital disorders) due to beeja bhaga avayava dushti (hereditary defects), leading to Mamsagata Vata dushti (muscle and neuromuscular impairment), resulting in progressive muscle dysfunction. Over 1 year and 7 months of Ayurvedic management, the patient showed marked clinical improvement. Notably, CPK levels significantly reduced from 813 U/L to 23.8 U/L. At a routine follow-up on September 22, 2020 nearly a year later, CPK levels remained at 23.8 U/L, indicating sustained improvement. This case highlights the potential of Ayurvedic intervention in managing progressive neuromuscular disorders like LGMD, offering promising results in functional recovery and biochemical improvement.

Keywords: Ayurveda, Creatine Phosphatase, Limb-Girdle Muscular Dystrophy, Panchakarma, Vata vyadhi.

INTRODUCTION

Limb-girdle muscular Dystrophies (LGMDs) comprise a heterogeneous group of disorders characterized by progressive weakness and degeneration of the proximal limb-girdle muscles, particularly in the hip and shoulder region. LGMDs exhibit both interfamilial and intrafamilial variability, with phenotypes ranging from mild forms to severe, early-onset, and rapidly progressive manifestations.[1] Clinical features of the disease include tiptoe walking, waddling gait, difficulty running and climbing stairs, and scapular winging. Joint contractures, Achilles tendon shortening, and scoliosis are frequently observed, while facial and neck muscles typically remain unaffected.[2] Muscle weakness in Limb-Girdle Muscular Dystrophy (LGMD) typically manifests around 15 years of age, although onset can occur earlier (<12 years) or later (>30 years). Cardiac involvement, including arrhythmias, conduction abnormalities, and left ventricular dysfunction, is rarely reported.[3]

Current treatments for muscular dystrophies remain largely palliative, focusing on symptom management rather than a cure. Glucocorticoids like prednisone and deflazacort help slow muscle degeneration but have significant side effects. Physical and occupational therapy aids in mobility preservation, while assistive devices like braces and wheelchairs improve daily functioning without altering disease progression. Emerging treatments, such as gene therapy, demonstrate improved mobility in muscular dystrophy (MD) patients. However, these therapies are still under investigation and not widely available, highlighting the need for further research into effective disease-modifying treatments.[4-6]

Ayurveda and Panchakarma therapies offer a multi-modal approach to managing Limb-Girdle Muscular Dystrophy (LGMD) like conditions. They focus on muscle nourishment, metabolic regulation, and neuromuscular rejuvenation, which modern medicine currently lacks. Thus, they potentially enhance functional outcomes and quality of life beyond conventional symptomatic management. A 49-year-old male patient of LGMD underwent Niruha Basti (medicated enema) as a primary shodhana (bio-purification and rejuvenation) therapy within the Panchakarma framework. Additionally, he received sarvanga abhyanga (whole-body oleation), bashpa swedana (steam sudation therapy), and shashtika shali pinda swedana (therapeutic sudation using medicated rice boluses). Ayurvedic oral medications were administered based on their pharmacological properties and classical indications for a broad spectrum of Vata vyadhi (neuromuscular disorders). The efficacy of these herbo-mineral formulations in managing Vata vyadhi has been supported by several scientific studies, demonstrating their potential in addressing neuromuscular dysfunctions caused by diverse etiological factors. Based on Ayurveda principles, the condition was classified under Vata vyadhi and managed accordingly, integrating classical therapeutic guidelines with

clinical insights from similar cases. In Ayurveda, the pathophysiology of this disorder aligns with the concept of adhi bala pravritta vyadhi (congenital disorders)[7], wherein beeja bhagavayava dushti (genetic or hereditary defects)[8] leads to Mamsagata Vata dushti (muscular and neuromuscular dysfunction due to Vata dosha imbalance). This pathogenic process contributes to progressive muscle degeneration, weakness, and functional impairment, resembling the degenerative nature of neuromuscular disorders.

After 1 year and 7 months of Ayurvedic treatment, the patient exhibited notable clinical improvement along with a substantial reduction in CPK levels, decreasing from 813 to 23.8 U/L. This outcome underscores the therapeutic potential of Ayurveda in the management of Limb-Girdle Muscular Dystrophy (LGMD).

CASE REPORT

Patient Information

A 49-year-old male patient presented to the Panchakarma Outpatient Department (OPD) at KLE Ayurveda Hospital, Belagavi, Karnataka, India, on March 15, 2018, with complaints of progressive difficulty in ambulation, requiring support with a walking stick. He reported a sense of heaviness in both lower limbs and pain in the lower back and hip region, which worsened with walking. Additionally, the patient experienced difficulty in climbing stairs, necessitating assistance, along with occasional episodes of weakness in both upper limbs and difficulty in lifting heavy objects. The patient first experienced low back pain in January 2013 and sought medical consultation at a local hospital in Mudhol, District Bagalkot, Karnataka, India. His symptoms were initially managed with Nonsteroidal Anti-Inflammatory Drugs (NSAIDs), along with calcium and multivitamin supplementation. However, over time, the patient discontinued regular medical follow-up and resorted to frequent use of analgesics for symptomatic relief without further clinical evaluation or intervention.

As the low back pain persisted and the patient developed bilateral lower limb heaviness, he sought consultation at the Orthopedic Department of KLE Prabhakar Kore Hospital, Belagavi, Karnataka, India. An MRI of the lumbar spine was advised (06/3/2013), which revealed mild S-shaped scoliosis of the thoracolumbar spine without evidence of disc herniation, spinal canal stenosis, or other significant abnormalities. The patient was recommended physiotherapy and referred for further evaluation by the Neurology Department; however, he did not follow through with the advised investigations and instead opted for intermittent symptomatic management over the next four years.

In December 2017, as his condition worsened, he consulted a neurologist at KLE Prabhakar Kore Hospital, where a comprehensive clinical evaluation, including neurological examination, serum CPK levels, and nerve conduction study (NCS), led to a confirmed diagnosis of Limb-Girdle Muscular Dystrophy (LGMD). The patient's condition was initially managed with corticosteroids and rehabilitative therapies. Later, based on the recommendation of another patient treated at KLE Ayurveda Hospital, Belagavi, Karnataka, India, he approached the Panchakarma OPD. After a comprehensive medical evaluation, including clinical history and biochemical investigations, a structured Ayurvedic treatment protocol was implemented. The patient also reported that his sister exhibited a similar disease presentation and later succumbed to the condition. However, no medical documentation was available to confirm a diagnosis of muscular dystrophy in his sister.

Clinical Findings

Table 1: Vitals of the patient

Blood Pressure (BP)	134/84 mm of Hg
Pulse (P)	74 bpm
Respiratory Rate (RR)	19 cpm
Temperature (T)	97.6^{O} F

Systemic examination

CNS: The patient was well-oriented with the name, place, date
CVS: S1 and S2 head normally, no added abnormal sounds were heard.
RS: Normal bronchovesicular sounds were heard bilaterally, no added abnormal sounds were heard.

Locomotor examination

Gait: Waddling gait.
Upper and Lower Limbs: The patient exhibited bilateral shoulder muscle atrophy, with marked wasting of the deltoid muscles. Additionally, there was thinning of the thigh muscles and pseudo-hypertrophy of both calf muscles.
Spine: No visible scar marks or swelling; however, there was evidence of scoliotic changes with right-sided convexity.
Lower back pain was grade 6 on the Visual Analog Scale (VAS).[9]
Walking distance on flat surfaces assessed by 6 min walk test (6MWT) was 30-40 m with assistance.[10]
The muscle strength was graded as 4 out of 5 in both the bilateral knee joints and all upper and lower limbs.

Special tests

Gower's Test: Positive.[11]
Achilles Tendon Reflex: Grade 1+ (Diminished/Hyporeflexia)[12]
Barthel Index Score: 55 (indicating 'severe' dependency)[13]
No sensory abnormality was observed.
Serum CPK: 776 U/L (On 11/12/2017 At KLE Prabhakar Kore Hospital, Belagavi)
Nerve Conduction Study (NCS): The study was abnormal and revealed evidence of severe demyelinating motor sensory neuropathy. (on 13/12/2017)
Serum CPK: 813 U/L, and CPK MB: 26 U/L (On 16/03/2018 After consultation At KLE Ayurveda Hospital, Belagavi)
Antinuclear antibodies (ANA) profile: Negative
Other blood parameters like complete blood count, Thyroid profile, Random Blood Sugar, Renal Function Test, Liver Function Test, and Erythrocyte Sedimentation Rate were within normal limits.
The electrocardiogram (E.C.G.) was within normal limits.

Diagnostic Assessment

Based on the progressive proximal muscle weakness, waddling gait, positive Gower's sign, significant muscle atrophy, and elevated serum creatine phosphokinase (CPK) levels, along with abnormal nerve conduction study (NCS) findings indicative of severe demyelinating motor-sensory neuropathy, the patient was clinically diagnosed with Limb-Girdle Muscular Dystrophy (LGMD). The differential diagnosis for Limb-Girdle Muscular Dystrophy (LGMD) includes Duchenne and Becker Muscular Dystrophies (DMD/BMD), Spinal Muscular Atrophy (SMA), inflammatory myopathies (Polymyositis/Dermatomyositis), Myasthenia Gravis (MG), Charcot-Marie-Tooth Disease (CMT), Inclusion Body Myositis (IBM), metabolic myopathies, and Amyotrophic Lateral Sclerosis (ALS). Electromyography (EMG) confirmed a myopathic pattern, ruling out neurogenic disorders, while nerve conduction studies (NCS) excluded peripheral neuropathies. Autoimmune markers were negative, eliminating inflammatory myopathies. Persistently elevated CPK levels and

clinical findings (waddling gait, proximal muscle weakness, positive Gower's sign) strongly supported LGMD.[14]

This condition does not correspond exactly to any specific disease classification in Ayurveda. It is categorized under the broader classification of Vata vyadhis (diseases caused by Vata dosha) within the context of beeja bhagavayava dushti (genetic or hereditary defects)[8] due to its association with adhi bala pravritta vyadhi (congenital disorders)[7]. Additionally, Limb-Girdle Muscular Dystrophy (LGMD) is recognized for its genetic defect association, which aligns with this Ayurvedic understanding. The patient shared that his sister suffered from a similar condition and later died, though no medical evidence was available to confirm whether it was muscular dystrophy. The patient reported a history of parental consanguinity, a common factor associated with the autosomal recessive inheritance pattern of Limb-Girdle Muscular Dystrophy type 2 (LGMD2). In these cases, both parents act as asymptomatic carriers of the mutated gene, with each offspring having a 25% likelihood of inheriting the disorder if both parents pass on the defective allele. This genetic anomaly is conceptually explained in Ayurveda as beeja bhagavayava dushti.[15]

The patient exhibited symptoms of Mamsagata Vata, including a sensation of heaviness throughout the body and severe body pain resembling the impact of dandamushthihata (as if struck with a cudgel or fist).[16] In such muscular disorders, the involvement of Mamsavaha srotas (micro-channels responsible for nourishing muscular tissues) leads to Mamsa dhatwagni mandya (impaired local muscular tissue metabolism). This disruption affects the sequential nourishment of successive dhatus (utharothara dhatu poshana), resulting in Mamsa shosha, characterized by muscle degeneration, fibrotic changes, and functional impairment due to inadequate nourishment and progressive tissue depletion.[17] Difficulty in climbing stairs, and walking further suggests Mamsavaha srotodusti, indicating a disruption in the function of Mamsa dhatu (nutrients related to muscular tissues) that are transported through these channels. Consequently, the Ayurveda diagnosis was established as Mamsagata Vata. Given the chronic nature of the disease, lasting over a year, it was categorized as yapya vyadhi (manageable but incurable disease).

Ethical Consideration: The case study was conducted according to the ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants.

Informed Consent: Informed consent had been obtained from the patient before the intervention and for the publication of clinical data in the case report. However, the patient had not consented to the inclusion of video evidence; only clinical data and reports were provided.

Therapeutic Intervention

Table 2: Schedule of Panchakarma and other Snehana (oleation) and swedana (sudation) therapy.

<table>
<tr><th>Sr</th><th>Duration</th><th>Panchakarma treatment</th></tr>
<tr><td>1.</td><td>17/03/2018
To
23/03/2018</td><td>Whole body Twak (Cinnamomum verum) and Nirgundi (Vitex negundo) leaves Kashaya Parisheka (decoction streaming sudation)

Anuvasana Basti (AB) was given with Dhanwantara taila 50 ml (given after food)

Vaitarana Basti (VB) - (given on an empty stomach)
Ingredients: Guda (paste of jaggery) - 80 ml,
Saindhava Lavana (rock salt) - 5 gm,
Sneha (Medicated oil) - Dhanwantara taila 60 ml.
Kalka (medicated fine powder) - Yashtimadhu (Glycyrrhiza glabra) 10 gm, Punarnava (Boerhavia diffusa) 10 gm, Guduchi (Tinospora cordifolia) 10 gm
Kashaya (decoction) - Erandamoola (Ricinus communis) 300 ml,
Gomutra (cow's urine) - 50 ml, Paste of chincha (tamarind) - 50 ml.

<table><tr><td>17/03</td><td>18/03</td><td>19/03</td><td>20/03</td><td>21/03</td><td>22/03</td><td>23/03</td></tr><tr><td></td><td>VB</td><td>VB</td><td>VB</td><td>VB</td><td>VB</td><td>VB</td></tr><tr><td>AB</td><td>AB</td><td>AB</td><td>AB</td><td>AB</td><td>AB</td><td>AB</td></tr></table></td></tr>
<tr><td>2.</td><td>21/04/2018
To
27/04/2018</td><td>Anuvasana Basti (AB) was given with Dhanwantara taila 60 ml (given after food)

Mustadi Yapana Basti (MYB) - (given on an empty stomach)
Ingredients: Madhu (Honey) - 60 ml,
Saindhava Lavana - 5 gm,
Sneha - Dhanwantara taila 60 ml + Vidaryadi Ghrita 60 ml
Kalka - Shatapushpa (Anethum sowa Kurz) 10 gm + Ashwagandha (Withania somnifera Dunal) 10 gm + Guduchi (Tinospora cordifolia) 10 gm
Ksheerapaka - Milk processed with Mustadi Yapana Basti Kwatha drugs 120 ml.
Mamsa Rasa (Goat meat soup) – 120 ml.

<table><tr><td>21/04</td><td>22/04</td><td>23/04</td><td>24/04</td><td>25/04</td><td>26/04</td><td>27/04</td></tr><tr><td></td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td></tr><tr><td>AB</td><td></td><td></td><td>AB</td><td></td><td></td><td>AB</td></tr></table>
Sarvanga abhyanga with Dhanwantara taila followed by mrudu bashpa swedana (mild whole-body sudation)</td></tr>
<tr><td>3.</td><td>02/06/2018
To
08/06/2018</td><td>Anuvasana Basti (AB) - Same as above.
Mustadi Yapana Basti (MYB) - Same as above.

<table><tr><td>02/06</td><td>03/06</td><td>04/06</td><td>05/06</td><td>06/06</td><td>07/06</td><td>08/06</td></tr><tr><td></td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td></tr><tr><td>AB</td><td></td><td></td><td>AB</td><td></td><td></td><td>AB</td></tr></table></td></tr>
</table>

<table>
<tr><td></td><td></td><td>Whole body Shalishashtika pinda sweda
300 gm shashtika shali (rice harvested after 60 days of growth/njavara rice) is cooked in 1.5 L of decoction of Bala moola (roots of Sida cordifolia) and milk. The cooked mixture is made into pottali (a bolus form in cotton cloth). Bala moola kashaya and milk mixture are used for reheating the pottali during the procedure.
Procedure: A whole-body massage using Dhanwantara taila was performed for 15 minutes, followed by a 30-minute massage with a pottali filled with shashtika shali. Subsequently, another 10-minute whole-body massage with Dhanwantara taila was done.</td></tr>
<tr><td>4.</td><td>15/08/2018
To
21/08/2018</td><td>All treatment protocols same as above
<table><tr><td>15/08</td><td>16/08</td><td>17/08</td><td>18/08</td><td>19/08</td><td>20/08</td><td>21/08</td></tr><tr><td></td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td></tr><tr><td>AB</td><td></td><td></td><td>AB</td><td></td><td></td><td>AB</td></tr></table></td></tr>
<tr><td>5.</td><td>10/12/2018
To
16/12/2018</td><td>All treatment protocols are the same as above
<table><tr><td>10/12</td><td>11/12</td><td>12/12</td><td>13/12</td><td>14/12</td><td>15/12</td><td>16/12</td></tr><tr><td></td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td><td>MYB</td></tr><tr><td>AB</td><td></td><td></td><td>AB</td><td></td><td></td><td>AB</td></tr></table></td></tr>
<tr><td>6.</td><td>05/02/2019
To
11/02/2019</td><td>Sarvanga abhyanga with Dhanwantara taila followed by mrudu bashpa swedana.</td></tr>
<tr><td>7.</td><td>16/04/2019
To
22/04/2019</td><td>Same as above.</td></tr>
</table>

Table 3: Treatment schedule of Ayurveda oral medications

Sr	Duration	Oral Medications
1.	17/03/2018 To 15/04/2018	Dhanadhanayadi kashaya 10 ml thrice daily with 50 ml water after food Tab Suvarna Malini Vasant Rasa (125 mg) 1 tab twice daily with water after food. Tab Vata Kulantaka Rasa (125 mg) 1 tab thrice daily with water after food Vaishwanara churna 5 gm with 50 ml hot water once a day after food
2.	21/04/2018 To 20/05/2018	Same as above
3.	25/05/2018 To 23/06/2018	Dhanwantararishta 10 ml thrice daily with 50 ml water after food Vata Kulantaka Rasa (125 mg) 1 tab thrice daily with water after food Suvarna Malini Vasant Rasa (125 mg) 1 twice once daily with water after food.
4.	29/06/2018 To 28/07/2018	Dhanwantararishta 10 ml thrice daily with 50 ml water after food Suvarna Malini Vasant Rasa (125 mg) 1 tab once daily with water after food. Kushmanda Rasayana 15 gm with 50 ml hot milk once daily before food.
5.	06/08/2018 To 03/09/2018	Same as above
6.	10/09/2018 To 10/10/2018	Ashtavarga Kashaya 10 ml thrice daily with 50 ml water after food. Suvarna Malini Vasant Rasa (125 mg) 1 tab once daily with water after food. Kushmanda Rasayana 15 gm with 50 ml hot milk once daily before food.
7.	16/10/2018 To 14/11/2018	Cap. Lashuna Rasayana 500 mg 2 Capsules once a day with 50 ml of milk before food Tab Brihat vata chintamani rasa (125 mg): 1 tab twice daily with water after food. Kushmanda Rasayana 15 gm with 50 ml hot milk once daily before food. Tab Laghu Malini Vasant (125mg) 1 tab thrice daily with water after food.
8.	20/11/2018 To 30//04/2019	Cap. Lashuna Rasayana 500 mg 1 Capsule once on alternate days with 50 ml milk before food. Tab Laghu Malini Vasant (125mg) 1 tab thrice daily with water after food.

RESULTS AND DISCUSSION

Table 4: Timeline of Follow-ups and Results

SrNo	Date	Complaints	Clinical/Biochemical examinations
Chronological Case Summary: 1. Jan 2013: Patient experienced low back pain; consulted a local hospital in Mudhol; managed with NSAIDs, calcium, and multivitamins. 2. Mar 6, 2013: MRI (lumbar spine) showed mild S-shaped scoliosis; no significant abnormalities. Physiotherapy and neurology evaluation were advised but not followed. 3. 2013–2017: Symptoms gradually worsened; patient relied on intermittent analgesics without regular medical follow-up. 4. Dec 2017: Neurology consultation at KLE Prabhakar Kore Hospital; diagnosed with Limb-Girdle Muscular Dystrophy (LGMD) based on clinical evaluation, serum CPK, and NCS. Managed with corticosteroids and rehabilitative therapies. 5. Mar 15, 2018: Consulted Panchakarma O.P.D. at KLE Ayurveda Hospital for Ayurvedic treatment. 6. Family History: Sister had similar symptoms and later succumbed to the condition (no medical records available).			
1.	17/03/2018	Heaviness in both legs, aggravated by walking. Lower back and hip pain, aggravating with walking Difficulty in climbing stairs, requiring assistance. Occasional weakness in upper limbs and difficulty lifting heavy objects.	Gait: Waddling gait. 6MWT: 30-40 m with assistance. Barthel Index Score: 55 (Severe dependency). Bilateral shoulder muscle atrophy, marked deltoid wasting. Thigh muscle thinning with pseudo-hypertrophy of calf muscles. Power: 4/5 in bilateral knee joints and all limbs. Spine: Scoliotic changes with right-sided convexity; no scars or swelling. VAS: Grade 6 (Lower back and hip region). Gower's Test: Positive. Achilles Tendon Reflex: Grade 1+ (Diminished). Sensory Function: No abnormalities. Serum CPK - 813 U/L, CPK MB: 26 U/L
2.	24/03/2018	Heaviness in both legs, aggravated by walking. Mild reduction in low back and hip pain but still aggravating with walking. Difficulty in climbing stairs, requiring assistance. Occasional weakness in upper limbs and difficulty lifting heavy objects	Gait: Waddling gait. 6MWT: 70 m with assistance. Barthel Index Score: 55 (Severe dependency). Bilateral shoulder muscle atrophy, marked deltoid wasting. Thigh muscle thinning with pseudo-hypertrophy of calf muscles. Power: 4/5 in bilateral knee joints and lower limbs. 5/5 in upper limbs. VAS: Grade 5 (Lower back and hip region). Gower's Test: Positive. Achilles Tendon Reflex: Grade 1+ (Diminished). Serum CPK - 800 U/L
3.	26/05/2018	Mild reduction in both lower limb's heaviness. Remarkable reduction in low back and hip pain. Aggravation of pain and heaviness during walking is reduced than before. The patient can climb stairs better than before but still requires assistance. Occasional weakness in the upper limbs is reduced than before.	Gait: Waddling gait. 6MWT: 150 m with assistance. Barthel Index Score: 60 (Severe dependency). Gross reduction in bilateral shoulder muscle atrophy. Deltoid wasting improved than before. Gross reduction in thigh muscle thinning with pseudo-hypertrophy of calf muscles. Power: 4/5 in bilateral knee joints and all limbs. VAS: Grade 3 (Lower back and hip region). Achilles Tendon Reflex: Grade 1+ (Diminished). Gower's Test: Positive. Serum CPK - 737 U/L
4.	15/10/2018	Gross reduction in both lower limb's heaviness. Mild Low back and hip pain. Aggravation of pain and heaviness during walking was reduced completely. The patient can climb stairs better than before but still requires assistance. Occasional weakness in the upper limbs was absent.	Gait: Waddling gait improved than before 6MWT: 300 m with assistance. Barthel Index Score: 75 (Moderate dependency). Mild muscle atrophy in the bilateral shoulder. Deltoid wasting improved than before. Gross reduction in thigh muscle thinning with pseudo-hypertrophy of calf muscles. Power: 4/5 in bilateral knee joints and all limbs. VAS: Grade 1 (Lower back and hip region). Achilles Tendon Reflex: Grade 2+ (Normal). Gower's Test: Positive. Serum CPK - 459 U/L
	16/12/2018	Mild heaviness in both lower limbs. No pain in the lower back and hip region. The patient can climb stairs slowly without assistance.	Gait: Normal 6MWT: 500 m without assistance. Barthel Index Score: 85 (Moderate dependency). Mild muscle atrophy in the bilateral shoulder persists. Significant gain in thigh and calf muscle mass. Mild pseudo-hypertrophy of calf muscles persists. Power: 5/5 in bilateral knee joints and all limbs. VAS – grade 0 (Lower back and hip region). Gower's Test: Negative. Serum CPK - 275 U/L

	11/02/2019	Mild heaviness in both lower limbs. The patient can climb stairs better than before without assistance.	6MWT: 700 m without assistance. Barthel Index Score: 94 (Slight dependency). Serum CPK - 180 U/L
	14/10/2019	Mild heaviness in both lower limbs. The patient can climb stairs much faster than before.	6MWT: 800 m without assistance. Barthel Index Score: 96 (Slight dependency). Serum CPK - 70 U/L
The patient had a normal value of CPK on the last follow-up. Even though he is advised to take Cap. Lashuna Rasayana 500 mg and Tab Laghu Malini Vasant (125mg) continuously as mentioned in the last oral medication follow-up.			
The patient returned for a routine follow-up with no fresh complaints on September 22, 2020, nearly a year later. During this visit, CPK levels were re-evaluated and found to be 23.8 U/L.			

Muscular Dystrophy (MD) is a genetic disorder characterized by progressive muscle degeneration and weakness due to genetic mutations responsible for muscle structure and function. From an Ayurvedic perspective, this can be understood through the concept of beeja bhaga avayava dushti, where the defective genetic material (beeja bhaga avayava) leads to improper development and function of Mamsa dhatu (muscle tissues). Kapha dosha plays a fundamental role in maintaining the structure, stability, and nourishment of body tissues, including muscles. However, when Kapha undergoes dushti (vitiation) at the level of reproductive elements (beeja), it can lead to impaired tissue formation and progressive degenerative changes.

Kapha dosha has guru (heaviness), manda (slowness), sthira (stability), and abhishyandi (obstructive) properties,[18] which are crucial in muscle growth and maintenance. However, in the case of muscular dystrophy, these properties become dysfunctional due to genetic mutations affecting dystrophin production, a key protein responsible for muscle integrity.[19,20] The manda guna (sluggishness) of vitiated Kapha at the cellular level leads to dhatwagni mandya (impaired muscle metabolism), causing progressive muscle atrophy and weakness. The sthira (stable) property, when altered, results in rigidity and contractures, reducing muscle elasticity and function over time. Additionally, abhishyandi (obstructive) nature of Kapha correlates with the accumulation of abnormal protein structures in dystrophic muscle fibers, as seen in histopathological studies of MD patients. Vitiated Kapha creates avarana (obstruction) to the normal functioning of Vata, resulting in Vata prakopa and manifesting as Kapha-avrita Vata vyadhi (a disorder where Vata is enveloped by Kapha). Scientific studies on muscular dystrophy reveal that defective dystrophin disrupts cellular homeostasis, leading to chronic inflammation, fibrosis, and oxidative stress, all of which align with the Kapha-dominant pathological cascade.[21] The loss of proper muscle repair mechanisms due to Kapha dushti further exacerbates tissue degeneration. Moreover, the presence of pseudo-hypertrophy in certain MD types (such as LGMD) can be linked to Kapha-mediated abnormal tissue proliferation, where non-functional fibrotic and fatty tissues replace functional muscle fibers.

The initial treatment approach focused on removing dushita Kapha avarana to restore Vata's normal function. Whole-body parisheka swedana was administered using a kashaya of Twak (*Cinnamomum verum*) and Nirgundi (*Vitex negundo*) leaves. Parisheka swedana, a specialized sudation therapy, facilitates Kapha pacification and alleviates the obstruction to Vata gati (movement). The selected herbs possess ushna virya (hot potency) and Kapha-Vata Shamana (pacifying) properties [22,23], aiding in enhancing circulation and improving neuromuscular function. Acharya Charaka emphasizes that Basti is the most effective therapeutic intervention for managing all types of Vata vyadhis.[24] Basti primarily acts on Apana Vayu, subsequently influencing other subtypes of Vata, while also correcting imbalances in dushita Kapha and Pitta. This process enhances the absorption and efficacy of active compounds in Niruha Basti, reinforcing its role as a key therapeutic intervention (ardha chikitsa) for various Vata disorders [25]

In this case, the initial formulation of Niruha Basti was based on the principles of Vaitarana Basti, as described by Acharya Chakradatta and Acharya Vangasena. They specifically highlight Vaitarana Basti for its effectiveness in managing shoola (pain), Kapha-Vata disorders, sankocha (stiffness), and Vishama Jwara (intermittent fevers). It contains Chincha (Tamarind) and gomutra (cow's urine), which possess teekshna (penetrating) and Kapha Vatahara properties, facilitating the removal of Kapha avarana (obstruction), thereby restoring Vata's normal function.[26-28] Dhanadhanayadi kashaya contains teekshna (sharp), ushna (hot), and laghu (light) dravyas as well as deepana (digestive stimulant) and pachana (metabolic enhancer) properties that improve dhatwagni (tissue metabolism) by removing Kapha avarana, preventing further degeneration of Mamsa dhatu (muscle tissue).[29]

Vaishwanara churna, composed of Sunthi (*Zingiber officinale*), Ajamoda (*Apium leptophyllum*), and Haritaki (*Terminalia chebula*), possesses shothahara (anti-inflammatory), deepana (digestive stimulant), and pachana (carminative) properties.[30-32] It helps reduce stiffness, inflammation, and pain, making it beneficial in conditions like Muscular Dystrophy, Rheumatoid Arthritis (Amavata), Sciatica (Gridhrasi), and Neuromuscular disorders. Additionally, it supports gut health, preventing aama (toxic undigested material) accumulation, a key factor in Kapha-dominated pathologies. In muscular dystrophy, adipose (fat) tissue progressively replaces healthy muscle tissue.[33]

This aligns with the pathological process where fatty replacement of muscle tissue occurs due to dystrophin-associated disruptions, correlating with Kapha avarana to Vata, leading to progressive muscle weakness and stiffness. Therefore, the primary treatment approach focused on eliminating vitiated Kapha dosha first, followed by addressing nirupasthambita Vata (unobstructed by other doshas) to restore balance and improve muscle function.

The progression of muscular dystrophies is often associated with increased Serum CPK and CK-MB levels. CPK is an enzyme predominantly found in skeletal muscle cells, and its presence in the bloodstream signifies muscle membrane disruption. In MD, genetic mutations lead to structural protein deficiencies, such as dystrophin, resulting in increased sarcolemmal permeability. This heightened permeability allows CPK to leak from muscle fibers into the bloodstream, causing elevated serum CPK levels.[34] Muscle breakdown triggers an inflammatory cascade, increasing pro-inflammatory cytokines (TNF-α, IL-6) and leading to oxidative stress, endothelial dysfunction, and microvascular inflammation.[35]

Suvarna Malini Vasanta Rasa is a classical Rasayana and Dhatugata Jwarahara (reduce tissue level fever/inflammation) formulation containing Suvarna (Gold), Mukta (Pearl), Shuddha Hingula (c), Maricha (Black Pepper), and Shuddha Kharpara (purified calamine).[36] This formulation has anti-inflammatory,

antioxidant, and endothelial-protective properties, making it beneficial in muscular dystrophy-associated inflammation. Suvarna (gold) helps to downregulate pro-inflammatory cytokines (TNF-α, IL-6), reducing chronic inflammation and oxidative stress.[37,38] Shuddha Hingula and Shuddha Kharpara aid in detoxification and microvascular protection, while Maricha (*Piper nigrum*) enhances bioavailability. By modulating immune response, preserving mitochondrial function, and improving muscle regeneration, this formulation effectively counteracts muscle degeneration and inflammatory damage, supporting overall muscle health.[39,40]

Vata Kulantaka Rasa is aptly named for its role in managing hereditary (kula-related) Vata disorders by minimizing their impact and enhancing the quality of life. With consistent use, it effectively alleviates and eradicates all Vata-related imbalances as described in the classics. Most of its ingredients possess tikta (bitter) and katu (pungent) rasa, along with laghu (light), ruksha (dry) guna, and ushna (hot) veerya, which contribute to the pacification of vitiated Kapha, then targeting Vata dosha.[41] Vata Kulantaka Rasa primarily contains Kasturi (musk), which is composed of polypeptides, proteins, muscone, musclide, steroids, muscopyridine, and other active compounds. These constituents contribute to its anti-inflammatory, immune-regulating, and neuroprotective properties, supporting its therapeutic efficacy in various health conditions.[42]

Subsequently, the treatment approach was adjusted to specifically address particular Vata dosha. Yapana Bastis holds significant importance as it is indicated for both healthy individuals (swastha) and patients (atura) and can be administered at any time (sarva kala). Possessing key therapeutic properties such as mamsa balajanana (muscle-strengthening), shulahara (pain-relieving), janu-uru-jangha graham (improving stability and function of knee, thigh, and calf muscles), sadyobalajanana (instant strength enhancement), and rasayana (rejuvenation), Yapana Basti plays a crucial role in muscle reinforcement and exhibits neuroprotective effects.[43] Mustadi Yapana Basti (MYB) is traditionally utilized for its Vata-pacifying, balya (strength-promoting), and rasayana (rejuvenation) properties. MYB enhances strength rapidly by mitigating Vata as Yapana Bastis are sadyobalajanana.[44-46] In MD, elevated inflammatory mediators contribute to dystrophic pathology and muscle wasting. Guduchi (*Tinospora cordifolia*), which is present in both Vaitarana Basti and MYB, has been found to regulate NF-κB signaling, thereby controlling inflammatory cascades and preserving muscle integrity. Additionally, its role in improving mitochondrial function and reducing fibrosis can help counteract the progressive degeneration observed in MD. These mechanisms suggest that *T. cordifolia* may serve as an adjunct therapy in MD by alleviating inflammatory damage and supporting muscle regeneration.[47] The Ashwagandha (*Withania somnifera*) in MYB has a crucial role in muscle protein synthesis and regeneration, which is hampered in the case of MD.[48]

Research has demonstrated that Punarnava (*Boerhavia diffusa*) used in Vaitarana Basti exhibits significant anti-inflammatory and antioxidant properties, which contribute to its ability to scavenge free radicals and prevent oxidative stress-induced muscle degeneration.[49] Disrupted calcium homeostasis is a significant pathological feature of MD. Studies have shown that abnormalities in calcium handling contribute to muscle degeneration.[50] Niruha Basti (medicated enema) regulates calcium homeostasis in dystrophic muscles by influencing systemic absorption, gut microbiota, and metabolic pathways through active and passive diffusion mechanisms. The colon's mucosal lining allows lipophilic and small hydrophilic molecules from Basti dravyas to diffuse passively into the systemic circulation. Lipid-soluble components from Dhanwantara Taila used in VB, MYB, and AB as well as Vidaryadi Ghrita used in MYB, enhance the absorption of fat-soluble nutrients, which can modulate calcium signaling at the cellular level.[51] Lipophilic drugs can passively diffuse across cell membranes, while small hydrophilic drugs may require specific transport mechanisms or formulation strategies to enhance their absorption.[52] This formulation strategy is already integrated into Basti preparations, where components like madhu, saindhava lavana, and gomutra interact with the colonic mucosa to influence active transport mechanisms through ion channels and ATP-dependent pumps, helping to reduce calcium overload in muscle tissues. When madhu, saindhava lavana, taila, and/or ghrita are combined in the Basti emulsion, they enhance the penetration of other medicinal ingredients due to their yogavahi (catalytic) effect. The concept of yogavahi, which improves the bioavailability of substances, has been a cornerstone of Ayurvedic practice for centuries [53-55]

Impaired angiogenesis in muscular dystrophy refers to the reduced ability of the body to form new blood vessels, which is a crucial process for muscle regeneration and repair. Drugs like Guduchi in Basti have been shown to upregulate Vascular Endothelial Growth Factor (VEGF), a key protein that promotes the formation of new blood vessels. By reducing inflammation, Guduchi helps protect endothelial cells from damage, supporting healthy vascular formation [56]

Dhanwanthara Taila is widely used in Basti preparations and for external oleation through sarvanga abhyanga as part of this treatment protocol. This taila is considered as 'Sarva Vatavikarajit' (conqueror of all Vata disorders) and is also known for its rasayana (rejuvenation) properties.[57,58] Vidaryadi Ghrita, used in MYB, contains vidaryadi gana herbs known for their brimhana (nourishing) and Vata shamana (Vata-pacifying) properties. These attributes contribute to its potential as an immunomodulator and its ability to rejuvenate degenerative conditions [59,60]

Shashtika shali (njavara rice) based therapies, including shashtika pinda sweda (sanskrit) or njavarakizhi (Malayalam) and shashtika anna lepa (Sanskrit) or njavara theppu (Malayalam), are integral components of Keraliya Panchakarma, a specialized branch of Panchakarma treatments developed historically in Kerala by the Ashtavaidyas. These therapies continue to be highly regarded for their efficacy in managing a wide range of Vata-impaired conditions.[61,62]

Shashtika Shali Pinda Sweda utilizes snigdha (unctuous) substances such as ksheera (milk) and shashtika shali dhanya. It exhibits snigdha, guru (heavy), sthira (stable), and tridoshaghna (balances all three doshas) properties. While primarily a swedana karma (sudation therapy), it also possesses brimhana guna (nourishing properties), contributing to its therapeutic benefits. It has also shown improvement in motor deficits in various Vata vyadhis.[63] Shashtika shali contains significant levels of tricin and two unique flavonolignans: tricin 4'-O-(erythro-β-guaiacylglyceryl) ether and tricin 4'-O-(threo-β-guaiacylglyceryl) ether. These compounds have demonstrated notable antioxidant activities, which are crucial in mitigating oxidative stress, a contributing factor in neuromuscular degeneration.[64] Bala (*Sida cordifolia*) root decoction used in shashtika shali pinda sweda is a prominent herb classified under brimhana dashaimani (ten nourishing herbs), balya dashaimani (ten strength-promoting herbs), madhura skandha (group of sweet-tasting herbs), and Vatashamaka varga (group of herbs that pacify Vata dosha) [65-67]

The ingredients of Ashtavargam Kashaya exhibit Vatashamaka (Vata-pacifying), vedana sthapana (pain-relieving), tarpana (nourishing), balya (strength-enhancing), rasayana

(rejuvenating), and sroto vishodhana (channel-cleansing) properties. These therapeutic actions aid in managing Vata-related disorders, enhance dhanwagni (tissue metabolism) and facilitate tissue regeneration.[68] This pathological condition is associated with Mamsa shosha, characterized by muscle degeneration, fibrotic changes, and functional impairment resulting from insufficient nourishment and progressive tissue depletion. Kushmanda Rasayana is administered to mitigate these effects, as it is traditionally indicated in the management of karshya (emaciation and malnourishment), promoting tissue regeneration and nutritional support.[69]

Brihat Vata Chintamani Rasa is widely used in many degenerative Vata vyadhis, which contain various types of bhasmas like Suvarna Bhasma (Calx of Gold), Rajat Bhasma (Calx of Silver), Abhrak Bhasma (Calx of Mica), Loha Bhasma (Calx of Iron), etc.[70] Bhasma is traditionally regarded as a biologically synthesized form of nanoparticles and has been an integral component of Ayurvedic therapeutics, often prescribed in combination with various herbal formulations. The utilization of metals in finely reduced particle sizes dates back to the Charaka Samhita (circa 1500 BCE). A well-prepared herbo-mineral/metalloid formulation exhibits key attributes such as rasayana (immunomodulatory and rejuvenating properties) and yogavahi (enhanced bioavailability and targeted drug delivery). Additionally, when properly processed, these formulations are non-toxic, easily absorbable, biocompatible, and metabolically adaptable within the human body.[71-73] Rajata Bhasma, in this preparation, has a free radical scavenging effect.[74] Abhraka Bhasma is a potent restorative agent with powerful cell-regenerating properties and is recognized as an effective rejuvenator.[75-77] Loha Bhasma contains elemental iron, which is vital for numerous physiological processes at the tissue level. It plays a crucial role in oxygen transport, energy production, and DNA synthesis. Iron is a key component of hemoglobin in red blood cells, facilitating oxygen delivery to tissues, and is also integral to myoglobin in muscle cells, supporting oxygen storage and utilization. Additionally, iron is a cofactor for various enzymes involved in cellular respiration and DNA replication.[78] Brihat Vata Chintamani Rasa, as a whole preparation, has also shown neuroprotective activity [79]

Lashuna Rasayana and Laghu Malini Vasant Rasa were recommended during the last follow-up and are advised to be continued to prevent the recurrence of neuromuscular tissue degeneration. Lashuna (*Allium sativum*) is the prime ingredient of Lashuna Rasayana.[80] Lashuna possesses high antioxidant properties.[81] Acharya Vagbhata regarded Lashuna as the most effective among Vatahara dravyas. He emphasized its significance as a Rasayana in managing upasthamita Vata (Vata disorders with obstruction) and nirupasthambita Vata (without obstruction). Additionally, classical texts also recommend its use in conditions such as Jeerna Jwara (chronic fever) and shosha (wasting disorders).[82,83] The abundantly present garlic-derived ImPs, QR-1, and QR-2, identified as lectins or agglutinins ASA II and ASA I, have demonstrated potent mitogenic activity, indicating their potential utility in therapeutic immunomodulation[84]

Laghu Malini Vasanta Rasa is indicated in cases of jeerna dhatugata Jwara (chronic tissue-level inflammation/fever), which can be correlated with the persistent inflammatory cascade affecting Mamsa dhatu (muscle tissue) in muscular dystrophy-like conditions. This formulation is also responsible for providing nourishment.[85] This formulation contains Zinc Sulfide as its primary ingredient, recognized for its tissue-regenerating properties and its role in promoting tissue repair and healing.[86]

Following treatment for 1 year and 7 months, the VAS score for the low back and hip region showed a significant reduction from 6 to 0, indicating complete pain relief. The waddling gait improved to a normal walking pattern, and functional independence was notably enhanced, as reflected in the Barthel Index Score, which improved from severe dependency (55) to minimal dependency (96). Gower's test, which was previously positive, turned negative, demonstrating improved muscle strength. The Achilles Tendon Reflex, initially diminished (Grade 1+), and returned to normal (Grade 2+), signifying neuromuscular recovery. The 6MWT distance, which was previously limited to 30–40 meters with assistance, increased to 800 meters without assistance, indicating a significant improvement in endurance and mobility. Gross muscle atrophy of the bilateral shoulders was visibly reduced, with only minimal residual atrophy at the end of treatment. Deltoid wasting improved significantly, and there was a remarkable reduction in thigh muscle thinning, with substantial muscle mass gain in the thighs and calves. The pseudo-hypertrophy of the calf muscles, which was prominent earlier, was minimally present post-treatment.

Additionally, muscle power across bilateral knees and all limbs improved from Grade 4 to Grade 5, indicating a return to near-normal strength. A significant biochemical improvement was also observed, as CPK levels decreased from 813 U/L to 70 U/L, reflecting reduced muscle degeneration and enhanced overall muscular health in this case of LGMD. On September 22, 2020, nearly a year after the previous visit, the patient came for a routine follow-up, and CPK levels were evaluated again, showing a value of 23.8 U/L.

Since this condition is classified as adibalapravritta vyadhi (a hereditary disorder), it is yapya in nature. Management strategies focus on genetic repair, where Panchakarma procedures and Rasayana therapies may influence epigenetic mechanisms, potentially beneficially modulating gene expression. Evidence suggests that essential nutrients and trace metals can induce epigenetic modifications, thereby contributing to disease management.[87-89] The findings of this case are significant as they highlight an effective therapeutic approach, offering promising outcomes for a condition that typically requires lifelong supportive care. After this treatment protocol, the patient had a significant reduction in clinical symptoms as well as in other assessment parameters and biochemical analyses. The comprehensive pathophysiology of muscular dystrophy, derived from multiple research studies [90,91], along with the potential comparison of mechanisms of action of Ayurveda pathophysiology and interventions in this case report, is presented in Figure 1.

CONCLUSION

The results demonstrate significant clinical and functional improvements in a patient with Limb-Girdle Muscular Dystrophy (LGMD) following Ayurveda and Panchakarma-based interventions. Notable enhancements were observed in pain reduction (VAS: 6 to 0), mobility (6MWT: 30–40m to 800m), muscle strength (power: 4 to 5), and functional independence (Barthel Index: 55 to 96). Additionally, neuromuscular reflexes normalized (Achilles Reflex: 1+ to 2+), muscle atrophy and pseudo-hypertrophy showed remarkable recovery, and serum CPK levels significantly decreased from 813 U/L to 70 U/L, indicating reduced muscle degeneration. Even after a year, CPK levels remained within normal limits (23.8 U/L), with no recurrence of symptoms.

These promising outcomes suggest that Ayurveda, particularly Panchakarma therapies, herbo-mineral preparations, and

Rasayana formulations, may offer a viable complementary approach to managing progressive neuromuscular disorders like LGMD. However, future research with larger clinical trials, molecular studies on epigenetic modifications, and mechanistic insights into Ayurveda-based interventions is essential to establish its role in muscle regeneration, gene expression modulation, and long-term disease management in muscular dystrophy-like conditions.

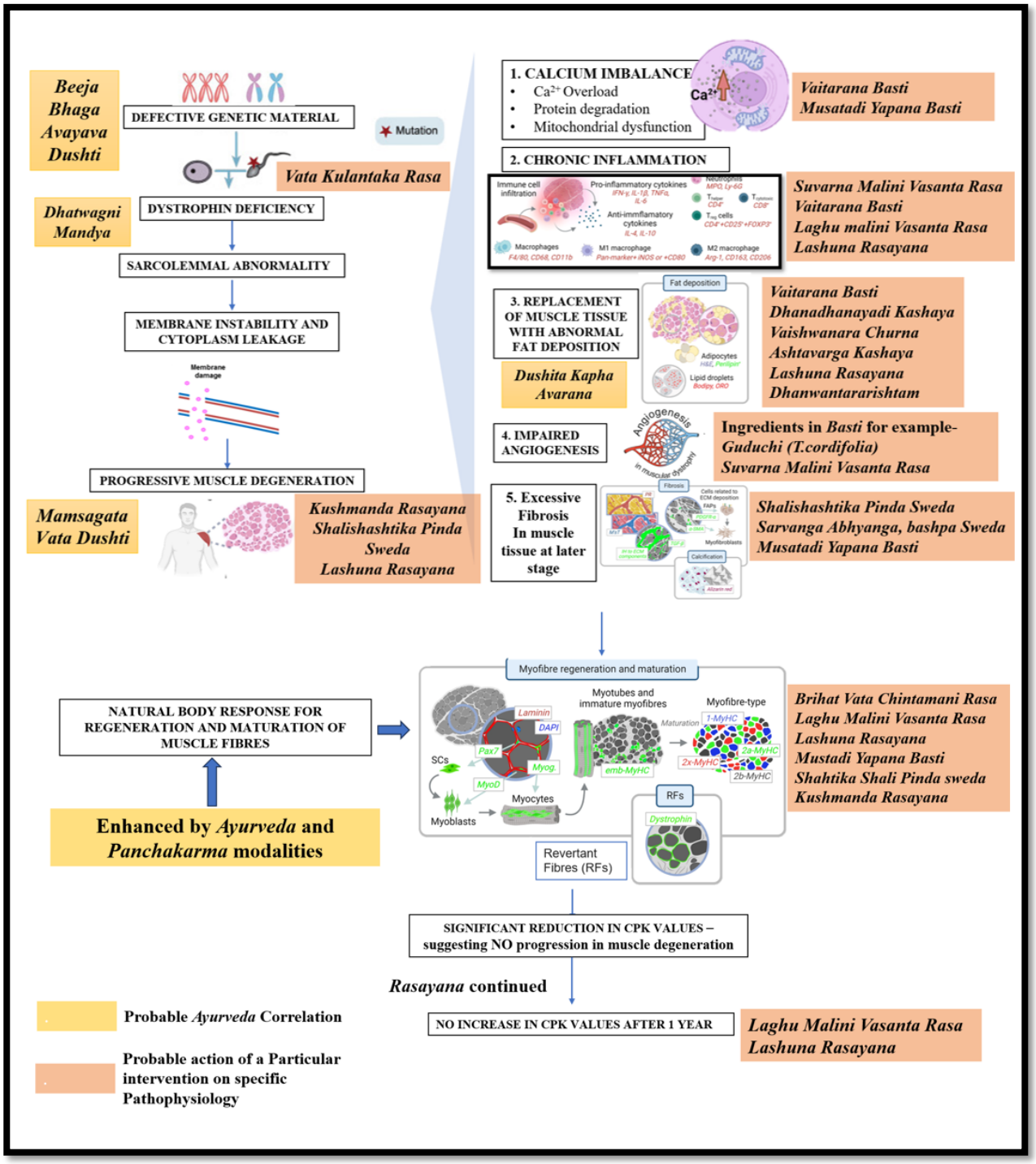

Figure 1: The comprehensive modern pathophysiology of muscular dystrophy, along with the potential comparison of mechanisms of action of Ayurveda pathophysiology and interventions.

REFERENCES

1. Nigro V, Savarese M. Genetic basis of limb-girdle muscular dystrophies: the 2014 update. Acta Myol. 2014; 33:1–12.
2. Angelini C, Fanin M. Calpainopathy. In: Adam MP, Ardinger HH, Pagon RA editors. GeneReviews®. Seattle, WA: University of Washington 2017. 1–30.
3. Mori-Yoshimura M, Segawa K, Minami N, Oya Y, Komaki H, Nonaka I, *et al.* Cardiopulmonary dysfunction in patients with limb-girdle muscular dystrophy 2A. Muscle Nerve. 2017; 55:465–9. DOI: 10.1002/mus.25369.
4. Rawls A, Diviak BK, Smith CI, Severson GW, Acosta SA, Wilson-Rawls J. Pharmacotherapeutic Approaches to

Treatment of Muscular Dystrophies. Biomolecules 2023; 13:1536. DOI: https://doi. org/10.3390/biom13101536
5. Siciliano G, Simoncini C, Giannotti S, Zampa V, Angelini C, Ricci G. Muscle exercise in limb girdle muscular dystrophies: pitfall and advantages. Acta Myol. 2015 May;34(1):3-8. PMID: 26155063; PMCID: PMC4478773.
6. Ramos J, Chamberlain JS. Gene Therapy for Duchenne muscular dystrophy. Expert Opin Orphan Drugs. 2015;3(11):1255-1266. DOI: 10.1517/21678707. 2015.1088780. Epub 2015 Oct 6. PMID: 26594599; PMCID: PMC4651452.
7. Sushruta Samhita: Ayurveda Tatva Sandeepika Hindi Commentary by Kaviraj Ambica Dutt Sastri, Chaukhamba Sanskrit Series Office, Varanasi, edition 2009. su.su.24/4, p 113.
8. Charak Samhita: Text with English translation and critical exposition based on Chakrapani Dutta"s ayurveda Dipika by Dr. R.K. Sharma and Vaidya Bhagavan Dash, Chaukhamba Sanskrit series office, Varanasi, edition 2009, ch.sa 3/17, p 384.
9. Mark P. Jensen, Connie Chen, Andrew M. Brugger, Interpretation of visual analog scale ratings and change scores: A reanalysis of two clinical trials of postoperative pain, The Journal of Pain. 2003; 4(7):407-414. DOI: https://doi.org/10.1016/S1526-5900(03)00716-8.
10. McDonald CM, Henricson EK, Han JJ, Abresch RT, Nicorici A, Elfring GL, Atkinson L, Reha A, Hirawat S, Miller LL. The 6-minute walk test as a new outcome measure in Duchenne muscular dystrophy. Muscle Nerve. 2010 Apr;41(4):500-10. DOI: 10.1002/mus.21544. PMID: 19941 337.
11. Chang RF, Mubarak SJ. Pathomechanics of Gowers' sign: A video analysis of a spectrum of Gowers' maneuvers. Clin Orthop Relat Res. 2012 Jul;470(7):1987-91. DOI: 10.1007/s11999-011-2210-6. Epub 2011 Dec 28. PMID: 22203329; PMCID: PMC3369091.
12. Zimmerman B, Hubbard JB. Deep Tendon Reflexes. [Updated 2023 Jul 24]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025
13. Mahoney FI, Barthel D. Functional evaluation: the Barthel Index. Maryland State Med J. 1965; 14:56-61
14. Kate Bushby, Fiona Norwood, Volker Straub, The limb-girdle muscular dystrophies—Diagnostic strategies, Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease. 2007;1772(2):238-242. DOI: https://doi.org/ 10.1016/j.bbadis.2006.09.009.
15. Mitsuhashi S, Kang PB. Update on the genetics of limb girdle muscular dystrophy. Semin Pediatr Neurol. 2012 Dec;19(4): 211-8. DOI: 10.1016/j.spen.2012.09.008.
16. Pandey G, editor. Commentary Vidhyotini of Charaka Samhita of Agnivesa 2nd volume, chikitsa sthan chapter 28, verse 32. Varanasi: Chaukhamba Sanskrit Sansthan; 2012. p. 782.
17. Shastri A, editor. Commentary Ayurveda tattva sandipika of Sushruta Samhita of Maharshi Sushruta, 1st volume, sharira sthana chapter 9, verse 12. Varanasi: Chaukhamba Sanskrit Sansthan; 2012. p. 71.
18. Charak Samhita, English Translation by Prof. P. V. Sharma volume 1, Chaukhamba Orientalia, ninth edition, sutrasthan 1/60, 2005; p 8.
19. Forcina L, Pelosi L, Miano C, Musarò A. Insights into the Pathogenic Secondary Symptoms Caused by the Primary Loss of Dystrophin. Journal of Functional Morphology and Kinesiology. 2017; 2(4):44. DOI: https://doi.org/10.3390/ jfmk2040044.
20. Kharraz Y, Guerra J, Pessina P, Serrano AL, Muñoz-Cánoves P. Understanding the process of fibrosis in Duchenne muscular dystrophy. Biomed Res Int. 2014;2014:965631. DOI: 10.1155/2014/965631. Epub 2014 May 4. PMID: 24877152; PMCID: PMC4024417.
21. González-Jamett A, Vásquez W, Cifuentes-Riveros G, Martínez-Pando R, Sáez JC, Cárdenas AM. Oxidative Stress, Inflammation and Connexin Hemichannels in Muscular Dystrophies. Biomedicines. 2022; 10(2):507. DOI: https:// doi.org/10.3390/biomedicines10020507
22. Acharya Bhavmishra. Bhavaprakasa Nighantu. Pandey Dr GS, editor. Varanasi: Chaukhamba Bharti Academy; 2004: p 224-228.
23. Bhavamishra. Bhavaprakasa Nighantu - Hindi Commentary by KC Chunekar. 1st ed. Varanasi: Published by Chaukhamba Bharathi Academy; 2002, p 984
24. Trikamji Y. Editor. Charak Samhita Ayurveda Dipika, Sutra Sthana, Yajjapurushiya Adhaya, 25/40. First edition. Varanasi, Chaukhamba Subhani Pratishtha, 2006 p. 131.
25. Sushruta Acharya, Dalhana, Gayadas, Chikitsa sthana chapter Netrabastipramana Pravibhaga Chikitsa Adhyaya, verse 6, In Acharya YT, Narayana Sushrutha Samhita with Nibandha Samgraha, Nyayachandrika commentary. Reprint Edition: 2014. Varanasi Chaukhamba Sanskrit Sansthan. p. 525
26. Dutta Shastri Ambika, editor. Sushruta Samhita. Sutra sthana. fifth ed. 160 volume Chapter 46, verse 159- 160. Varanasi: Chaukhamba Sanskrit Pratisthan; 1978. p. 256
27. Sreekanta Moorthy KR. Bhavaprakasa Nighantu of Bhavamisra. second ed. volume 1. Varanasi: Chaukhamba Krishna Das Academy; 2001. verse 203- 204. p. 331
28. Bhavamishra BN, Chunekar PK. Varanasi: Chaukhamba Bharati Academy. 2010. p. 195–197
29. Rao G.P. Chaukhamba Sanskrit Series; Varanasi: 2016. Sahasra Yogam, chapter 3, verse 192; p. 91
30. Patil Usha. Pharmacognostic and Phytochemical Evaluation of Vaishvanara Churna. International Journal of Ayurveda and Pharma Research. 2016;4(7):1-4.3.
31. Priyabrata Pattanayak, Danendra Kumar Hardel, Prithwiraj Mohapatra. Standardization of Vaisvanara Churna: A Poly-herbal Formulation. Pharmacognosy Journal. January 2010;2(5):50-59.
32. Santhi Krishnan, I.S. Aswathy, Jasmine Peter, Vidya Sabu, A. Helen. Anti–Inflammatory Potential of Vaisvanara Churnam – An Ayurvedic Polyherbal Formulation In Cholesterol Fed Rats. Indian Journal of Scientific Research. 2018;18(2):29-37.
33. Xie Z, Xiao J, Zheng Y, Wang Z, Yuan Y. Magnetic Resonance Imaging Findings in the Muscle Tissue of Patients with Limb Girdle Muscular Dystrophy Type 2I Harboring the Founder Mutation c.545A>G in the FKRP Gene. Biomed Res Int. 2018 May 29; 2018:3710814. DOI: 10.1155/2018/ 3710814. PMID: 30003095; PMCID: PMC5996470.
34. Rocha CT, Hoffman EP. Limb-girdle and congenital muscular dystrophies: Current diagnostics, management, and emerging technologies. Curr Neurol Neurosci Rep 2010;10: 267-76.
35. Kim EY, Lee JW, Suh MR, Choi WA, Kang SW, Oh HJ. Correlation of Serum Creatine Kinase Level with Pulmonary Function in Duchenne Muscular Dystrophy. Ann Rehabil Med. 2017 Apr;41(2):306-312. DOI: 10.5535/arm. 2017.41.2.306. Epub 2017 Apr 27. PMID: 28503465; PMCID: PMC5426263.
36. Mishra SN, Bhaishajya Ratnavali Govind Das Sen edited with 'Siddhiprada' Hindi Commentary, Chaukhamba Surbharati Prakashan Varanasi, Jwara Rogadhikar Shloka 1183-1184, p 192.
37. Di Bella, D., Ferreira, J.P.S., Silva, R.d.O. *et al.* Gold nanoparticles reduce inflammation in cerebral microvessels of mice with sepsis. J Nanobiotechnol. 2021; 19:52. DOI: https://doi.org/10.1186/s12951-021-00796-6
38. Ibrahim KE, Bakhiet AO, Awadalla ME & Khan HA. A Priming Dose Protects Against Gold nanoparticles-induced

Proinflammatory Cytokines mRNA Expression in Mice. Nanomedicine. 2017;13(3): 313–323. DOI: https://doi.org/10.2217/nnm-2017-0332
39. Sujatha Pushpakanthi Hewageegana HG, Hewageegana AU, Ashanthi Menuka Arawwawala LD. Purification, Detoxification, and Incineration Methods of Minerals and Metals in Traditional Medicine Formulations of Sri Lanka. Evid Based Complement Alternat Med. 2021 Jan 15; 2021:6634553. DOI: 10.1155/2021/6634553. PMID: 33510804; PMCID: PMC7822661.
40. Singh S, Tripathi JS, Rai NP. An appraisal of the bioavailability enhancers in Ayurveda in the light of recent pharmacological advances. Ayu. 2016 Jan-Mar;37(1):3-10. DOI: 10.4103/ayu.AYU_11_15. PMID: 28827948; PMCID: PMC5541464.
41. Mishra SN, editor. Bhaishajya Ratnavali. Apasmara Rogadhikara. 40-43. Varanasi: Chaukhamba Surbharati Prakashan; p. 752.
42. Shuquan Lv, Zhixin Lei, Ge Yan, Sayed Afzal Shah, Saeed Ahmed, Taolei Sun, Chemical compositions and pharmacological activities of natural musk (Moschus) and artificial musk: A review, Journal of Ethnopharmacology. 2022; 284:114799. DOI: https://doi.org/10.1016/j.jep.2021.114799.
43. Agnivesha, Charaka, Chakrapanidatta, Siddisthana, Chapter Uttarabasti Siddi Adhyaya, verse 16, In: Y T Acharya (Edi.). Reprint Edition 2015, Varanasi Chaukhamba Orientalia, p. 731
44. Sushruta. Sushruta Samhita, Chikitsa Sthana, Niruha Krama Chikitsitham, 38/96101, translated by Srikantha Murthy KR, Shastri KA, Vol. 2, Reprint. ed. Chaukhamba Orientalia, Varanasi; 2012; p 379.
45. Acharya YT; ed; Agnivesa. Caraka Samhita with Ayurveda Dipika commentary of Chakrapanidatta. 2013 ed. Varanasi: Chaukhamba Prakashana: Siddhisthana: Chapter 12, sloka 15, P 731, P 738
46. Govindadas. Bhaishajya Ratnavali, Balarogachikitsa Prakaran, 71/63, translated by Sastry AD. Volume 3, Reprint. ed. Varanasi: Chaukhamba Sanskrit Sansthan, 2009; p 678.
47. Sharma B, Dutt V, Kaur N, Mittal A, Dabur R. Tinospora cordifolia protects from skeletal muscle atrophy by alleviating oxidative stress and inflammation induced by sciatic denervation. J Ethnopharmacol. 2020 May 23; 254:112720. DOI: 10.1016/j.jep.2020.112720. Epub 2020 Feb 27. PMID: 32114167.
48. Wang J, Zhang H, Kaul A, Li K, Priyandoko D, Kaul SC, Wadhwa R. Effect of Ashwagandha Withanolides on Muscle Cell Differentiation. Biomolecules. 2021 Oct 4;11(10):1454. DOI: 10.3390/biom11101454. PMID: 34680087; PMCID: PMC8533065.
49. Gharate, M., Kasture, V. Evaluation of anti-inflammatory, analgesic, antipyretic and antiulcer activity of Punarnavasava: An Ayurvedic formulation of *Boerhavia diffusa*. Orient Pharm Exp Med. 2013; 13: 121–126. DOI: https://doi.org/10.1007/s13596-012-0081-3
50. Zabłocka B, Górecki DC, Zabłocki K. Disrupted Calcium Homeostasis in Duchenne Muscular Dystrophy: A Common Mechanism behind Diverse Consequences. Int J Mol Sci. 2021 Oct 13;22(20):11040. DOI: 10.3390/ijms222011040. PMID: 34681707; PMCID: PMC8537421.
51. Azman M, Sabri AH, Anjani QK, Mustaffa MF, Hamid KA. Intestinal Absorption Study: Challenges and Absorption Enhancement Strategies in Improving Oral Drug Delivery. Pharmaceuticals (Basel). 2022 Aug 8;15(8):975. DOI: 10.3390/ph15080975. PMID: 36015123; PMCID: PMC9412385.
52. Guo, S., Liang, Y., Liu, L. *et al.* Research on the fate of polymeric nanoparticles in the process of the intestinal absorption based on model nanoparticles with various characteristics: size, surface charge and pro-hydrophobics. J Nanobiotechnol. 2021; 19: 32. DOI: https://doi.org/10.1186/s12951-021-00770-2
53. Singh J, Dubey RK, Atal CK. Piperine-mediated inhibition of glucuronidation activity in isolated epithelial cells of the guinea pig small intestine: Evidence that piperine lowers the endogeneous UDP-glucuronic acid content. J Pharmacol Exp Ther 2002; 302:645-50.
54. Tripathi B, editor. Ashtanga Hridaya of Vagbhata, Sutra Sthana, Ch. 19, Ver. 38-40. Reprint ed. Delhi: Chaukhamba Sanskrit Pratisthan; 2003. p. 235.
55. Shashti RD, editor. Charaka Samhita of Agnivesha, Chikitsa Sthana, Ch. 2/4, Ver. 10. Reprint ed. Varanasi: Chaukhamba Bharti Academy; 2005. p. 85.
56. Leyon PV, Kuttan G. Effect of *Tinospora cordifolia* on the cytokine profile of angiogenesis-induced animals. Int Immunopharm 2004 Dec 15;4(13):1569–75. DOI: https://doi.org/10.1016/j.intimp.2004.06.015
57. Shastri HP, editor. Ashtanga Hridayam of Vagbhata, Shareera Sthana; Garbha Vyapat. Chapter 2, Verses 47-52. Varanasi: Chaukhamba Surbharati Prakashan; Reprint 2018. p. 373.
58. Sharma R, Sharma S. Sahasrayogam, Dhanvantara taila, 2007 Delhi Chaukhamba Sanskrit Pratishthana: 74 reprint 2007. Chapter 5, P. 322
59. Brahmanand T. editor. Kasachikitsa Adhyaya: Chapter 3, verse 10. In Chikitsasthana, editor. Ashtanga Hridayam. Delhi: Chaukhamba Sanskrit Pratisthan; 2017. p. 587.
60. Brahmanand T. editor. Shodhanadi Gana Samgraha Adhyaya: Chapter 15, verses 9-10. In: Sutraasthana, editor. Ashtanga Hridayam. Delhi: Chaukhamba Sanskrit Pratisthan; 2017. p. 199.
61. Singh RH. (ed.). Panca Karma Therapy: Ancient Classical Concepts, Traditional Practices, and Recent Advances. Varanasi: Chaukhamba Sanskrit Series Office. 1992
62. Sreejayan U, Suresh Kumar V, Varghese G, Jacob TM, Thomas G. Stratification and population structure of the genetic resources of ancient medicinal rice (*Oryza sativa* L.) landrace Njavara. Genet. Resour. Crop Evol. 2011; 58: 697–711. DOI: 10.1007/s10722-010-9613-1
63. Vyas Apexa G, Kumar Kori Virendra, Rajagopala S, Patel Kalpana S. Etiopathological study on cerebral palsy and its management by shashtika shali pinda sweda and Samvardhana ghrita. Ayu. 2013 Jan–Mar;34(1):56–62. DOI: 10.4103/0974-8520.115450.
64. Mohanlal S, Parvathy R, Shalini V, Helen A, Jayalekshmy A. Isolation, characterization and quantification of tricin and flavonolignans in the medicinal rice Njavara (*Oryza sativa* L.), as compared to staple varieties. Plant Foods Hum Nutr. 2011 Mar;66(1):91-6. DOI: 10.1007/s11130-011-0217-5. PMID: 21373805.
65. Acharya Charaka, Charaka Samhita by Agnivesha revised by Charaka and Dridhabala of Chakrapanidatta, Sutrastana 4/2 chapter, edited by Acharya Yadavji and Trikamji, first edition, Varanasi, Chaukhamba Sanskrit Sansthan, 2001, P. 32.
66. Acharya Charaka, Charaka Samhita by Agnivesha revised by Charaka and Dridhabala of Chakrapanidatta, Sutrastana 25/40 chapter, first edition, edited by Acharya Yadavaji and Trikamji, Varanasi, Chaukhamba Sanskrit Sansthan, 2001, P. 132.
67. Acharya Vagbhata, Ashtanga Samgraha commentary of Shashilekha by Indu, Sutrastana 15, edited by Dhivaprasad Sharma, 7th edition Varanasi, Chaukhamba Sanskrit Series Office, 2006, P. 130.
68. The Ayurvedic Formulary of India. 1st edition. New Delhi, India: Department of Indian Systems of Medicine and Homeopathy, Ministry of Health and Family Welfare, Government of India; 2003. P. 265

69. Shastri A, Shastri R, Bhaisharjya Ratnavali (Hindi commentary), Raktapitta Rogadhikara 45-51, Chaukhamba Prakashan, 2013; P 406.
70. Raviraj Govind Dasa Virachita. In: Siddhi Nandan Mishra, editor. Bhaishajya Rathnavali. Vatavyadhi Rogadhikara verse 141-144. Varanasi: Chaukhamba Surbharati Prakashan; 2019. p. 530.
71. Pal D, Sahu CK, Haldar A. Bhasma: The ancient Indian nanomedicine J Adv Pharm Technol Res. 2014;5:4–12.
72. Vayalil PK, Kuttan G, Kuttan R. Rasayanas: Evidence for the concept of prevention of diseases Am J Chin Med. 2002;30:155–71.
73. Mishra LC, Adra T, Batchu SV, Bhatt HA Scientific Basis for Ayurvedic Therapies. 2004 Boca Raton, Florida CRC Press, LLC:84–99.
74. Bagul MS, Kanaki NS, Rajani M. Evaluation of free radical scavenging properties of two classical polyherbal formulations Indian J Exp Biol. 2005;43:732–6
75. Raisuddin S. Ayurvedic bhasma. In: Mishra LC (Eds), scientific basis for Ayurvedic Therapies. CRC Press LLC, New York, 2004. P 83-100.
76. Pal D, Sahu CK and Haldar A. Bhasma. The ancient Indian nanomedicine, Journal of Advanced Pharmaceutical, Technology and Research. 2014; 5(1):4-12.
77. Pal SK. The Ayurvedic bhasma: The ancient science of nanomedicine. Recent patents on Nanomedicine. 2015;5:12-18
78. Abbaspour N, Hurrell R, Kelishadi R. Review on iron and its importance for human health. J Res Med Sci. 2014 Feb;19(2):164-74. PMID: 24778671; PMCID: PMC3999603.
79. Vikram Goshan, Mundugaru Ravi, Narayana Prakash Sudhakar Bhat, Ravishankar Basavaiah. Evaluation of neuro-protective activity of Brihatvata Chinthamani rasa. J Phytopharmacol. 2015; 4(4):207–211.
80. Vagbhata. Ashtanga Hridaya with commentaries Sarvangasundara of Arunadatta and Ayurvedarasayana of Hemadri; Annotated by: Dr. Anna Moreshwar Kunte and Krishna Ramachandra Shastri Navre. In: Pt. Hari Sadasiva Sastri Paradakara Bhishagacharya, editor. Chikitsa sthana, Rasayanadhyaya. Chapter 39, verses 113-127. Varanasi: Chaukhamba Surbharati Prakashan; 2010. p. 931
81. Locatelli DA, Nazareno MA, Fusari CM, Camargo AB. Cooked garlic and antioxidant activity: Correlation with organosulfur compound composition. Food Chem. 2017; 220:219–24. DOI: 10.1016/j.foodchem.2016.10.001.
82. Kashyap Samhita, or Vriddhajivakiya Tantra, text with English translation and commentary, By Prof. P.V. Tiwari, Chaukhamba Visvabharati, Varanasi, Kalpasthana, Lasunakalp adhyaya, p 337.
83. Prof. P.V. Tewari, Kashyapa Samhita (2002), Edited by Prof. P.V. Tewari, translation and commentary by P.V. Tewari, Chaukhamba Visvabharati oriental publishers Varanasi, Kalpasthana chapter 2, P 327.
84. Clement F, Siddanakoppalu NP, Yeldur PV *et al*. Identity of the immunomodulatory proteins from garlic (*Allium sativum*) with the major garlic lectins or agglutinins. International Immunopharmacology 2010; 10: 316-324.
85. Shastri, Bhisagratna Brahmasankar, Yogartnakara with Vidhyotini Hindi Comm., Reprint Edition 2015 Chaukhamba Prakashan Varanasi, Uttrardha, Jwaradhikara Verse 211. P 186.
86. Bo Han, William H. Fang, Shuqing Zhao, Zhi Yang, Ba X Hoang, Zinc sulfide nanoparticles improve skin regeneration, Nanomedicine: Nanotechnology, Biology and Medicine. 2020;29:102263. DOI: https://doi.org/10.1016/j.nano.2020.102263.
87. Vishwanatha U, Guruprasad KP, Gopinath PM, Acharya RV, Prasanna BV, Nayak J, *et al.* Effect of Amalakirasayana on DNA damage and repair in randomized aged human individuals. J Ethnopharmacol 2016; 191:387e97. DOI: https://doi.org/10.1016/j.jep.2016.06.062. PMID: 27364038.
88. Sharma H. Ayurveda: science of life, genetics, and epigenetics. Ayu 2016 Apr-Jun;37(2):87e91. DOI: https://doi.org/10.4103/ayu.AYU_ 220_16. PMID: 29200745; PMCID: PMC5688840.
89. Weinhold B. Epigenetics: the science of change. Environ Health Perspect 2006;114(3): A160e7. DOI: https://doi.org/10.1289/ehp.114-a160. PMID: 16507447; PMCID: PMC1392256.
90. Dubuisson N, Versele R, Planchon C, Selvais CM, Noel L, Abou-Samra M, Davis-López de Carrizosa MA. Histological Methods to Assess Skeletal Muscle Degeneration and Regeneration in Duchenne Muscular Dystrophy. Int. J. Mol. Sci. 2022;23: 16080. DOI: https://doi.org/10.3390/ijms232416080
91. Kaziród K, Myszka M, Dulak J, Łoboda A. Hydrogen sulfide as a therapeutic option for the treatment of Duchenne muscular dystrophy and other muscle-related diseases. Cell Mol Life Sci. 2022 Nov 28;79(12):608. DOI: 10.1007/s00018-022-04636-0. PMID: 36441348; PMCID: PMC9705465.

Cite this article as:

Vishwanath Wasedar, Bhagyesh Pangam and Aparna Siddhaye. Ayurveda treatment approach for Limb-Girdle Muscular Dystrophy: A case report on significant symptomatic and biochemical improvements. Int. J. Res. Ayurveda Pharm. 2025;16(3):52-63
DOI: http://dx.doi.org/10.7897/2277-4343.16395

Source of support: Nil, Conflict of interest: None Declared