

Ayurvedic management of limb girdle muscular dystrophy in a twelve-year-old patient with *Panchakarma* and *Shamana Chikitsa*: a case study.

Mangesh Bhosale*¹, Raghavendra Pandilwar²

¹PG Scholar, ²Associate Professor,

Department of Kaumarbhritya, YMT Ayurvedic Medical College and Hospital, Kharghar, Maharashtra, India

*Corresponding Author: Email Address – mangeshbhosale700@gmail.com

ABSTRACT:

Background: Limb girdle muscular dystrophy is a genetically heterogeneous group of progressive muscle disorders for which no curative treatment exists, and care is largely supportive. In Ayurveda the presentation can be understood as *Mamsa dhatu kshaya* with *Vata* vitiation.

Aim and objectives: To document the Ayurvedic management of one child with limb girdle muscular dystrophy and to describe the clinical and biochemical course during hospital care.

Observations: A twelve-year-old girl presented with difficulty in rising from sitting, inability to climb stairs, frequent falls while running, a waddling gait and a positive Gowers sign. Serum creatine phosphokinase was 3528.1 at baseline, with raised liver transaminases and a low vitamin D level. Neurology and rheumatology opinions and echocardiography were obtained, and blood was sent for genome sequencing. She was

admitted for 53 days and received *Abhyanga* with *Mahanarayana taila* and *Mahamasha taila*, *Shashtikashali pinda sweda*, and *Basti* with *Bala taila*, *Mustadi yapana basti* and *Sanvardhana ghrita*, together with oral *Shamana* formulations and nutritional supplements.

Main findings: Creatine phosphokinase measured 1429, 1123 and 1308 on repeat testing, a fall of about 63 percent from baseline at the last measurement. She was haemodynamically stable at discharge and no adverse event was recorded. Functional outcome was not quantified and the genetic diagnosis was awaited.

Conclusion: The biochemical trend is encouraging, but it cannot be attributed to the intervention in a single uncontrolled case. Structured functional scales, genetic confirmation and longer follow-up are needed in future reports.

Keywords: Limb girdle muscular dystrophy, *Mamsa dhatu kshaya*,

INTRODUCTION

Muscular dystrophies are inherited disorders in which progressive degeneration of skeletal muscle leads to weakness and loss of function [1]. Limb girdle muscular dystrophy is a genetically heterogeneous group within this family. It is characterised by predominant weakness of the shoulder and pelvic girdle muscles, a raised serum creatine kinase level and dystrophic changes on muscle biopsy [1,2]. The nomenclature was revised in 2018 so that each subtype is named by its inheritance pattern (recessive or dominant), the order of gene discovery and the affected protein [2]. Pooled population data place the prevalence of the limb girdle forms in the order of a few cases per 100,000 people, so each individual presentation is uncommon in clinical practice [3].

About thirty subtypes have been described, and they differ in age at onset, rate of progression and involvement of the heart and respiratory muscles [4,5]. Recessive forms generally present in childhood or adolescence and tend to progress faster than dominant forms, which usually present in adulthood [2,6]. Some subtypes carry a risk of cardiomyopathy or respiratory failure, so a molecular diagnosis matters for prognosis, for decisions about surveillance and for genetic counselling [4,7]. The relative frequency of subtypes also differs between populations [7].

In childhood the disease typically announces itself through difficulty in rising from the floor or from a chair, a waddling gait caused by pelvic girdle weakness, frequent falls, and difficulty in climbing stairs and running. Management remains

supportive and is built around physiotherapy, surveillance of cardiac and respiratory function, attention to nutrition and bone health, and genetic counselling. Disease-modifying treatment is not available for most subtypes [8]. Families therefore frequently explore complementary systems of medicine, and documentation of such care is useful, provided it is reported honestly and with its limitations.

Ayurveda does not describe limb girdle muscular dystrophy under a single name. The clinical picture can, however, be studied through the concepts of *Mamsa dhatu* (muscle tissue), *Dhatu kshaya* (tissue depletion) and *Vata vyadhi* (disorders of *Vata*). Classical texts state that depletion of the *dhatu* aggravates *Vata*, and that wasting of *Mamsa dhatu* is accompanied by dryness, weakness and loss of bulk [9-11]. Disorders that are present from birth or arise from the hereditary seed are grouped as *Sahaja vyadhi* [12]. The principles of treatment for such states are *Snehana* (oleation), *Swedana* (sudation), *Brimhana* (nourishing therapy) and *Basti* (medicated enema), followed by *Shamana* and *Rasayana* measures [13,14].

This article describes the Ayurvedic in-patient management of a twelve-year-old girl with limb girdle muscular dystrophy and presents the biochemical course observed during 53 days of hospital care.

MATERIALS AND METHODS

Study design and ethics

This is a descriptive single-patient case study compiled from the in-patient discharge summary of the paediatric (*Balroga*) in-patient department of Y.M.T. Ayurvedic Medical College and Charitable Hospital, Kharghar, Navi Mumbai.

Reporting follows the CARE guidelines for case reports [15]. All identifying details, including name, address and hospital registration numbers, have been removed. Written informed consent for publication was obtained from the parent of the patient.

Patient information

A twelve-year-old girl was admitted on 28 May 2026 and discharged on 20 July 2026, a stay of 53 days. Her complaints were difficulty in standing from the sitting

position, ability to stand only with support, falling down while running, inability to climb stairs and difficulty in running. There was no history of hypertension, diabetes mellitus, ischaemic heart disease, tuberculosis, rheumatoid arthritis or thyroid disorder. The discharge record does not state the duration of symptoms, age at onset, birth and developmental history, family history, consanguinity, anthropometry or previous treatment. The sequence of care is summarised in Table 1.

Table 1. Sequence of care as recorded in the discharge summary

Date	Event
27 May 2026	Baseline blood investigations
28 May 2026	Admission to the paediatric in-patient department
During admission	Neurologist and rheumatologist opinions, two-dimensional echocardiography, and blood sent for genome sequencing; <i>Panchakarma</i> with <i>Shamana Chikitsa</i> started after investigations
1 June 2026	Electrocardiogram – Normal
16 June 2026	Repeat creatine phosphokinase
30 June 2026	Repeat blood investigations
14 July 2026	Repeat blood investigations
20 July 2026	Discharge; haemodynamically stable
23 July 2026	Appointment advised with a consultant, with genome sequencing reports
28 July 2026	Follow-up advised with the treating consultant

Clinical findings

Examination showed a waddling gait and a positive Gowers sign. The abdomen was soft and non-tender. Muscle power grades, deep tendon reflexes, calf muscle bulk, spinal and joint findings and the *Ashtavidha*

pariksha findings were not recorded in the discharge summary.

Diagnostic assessment

The consultant diagnosis was limb girdle muscular dystrophy, unspecified (International Classification of Diseases

code G71.039). Complete blood count, serum creatinine, electrolytes, serum calcium, liver enzymes, serum vitamin D3, creatine phosphokinase and lactate dehydrogenase were measured, and an electrocardiogram and two-dimensional echocardiogram were performed. Opinions of a neurologist and a rheumatologist were obtained, and blood was sent for genome sequencing, the result of which was not available at discharge. Muscle biopsy, electromyography and muscle imaging are not mentioned in the record.

Ayurvedic assessment

On the basis of the presentation, the authors interpret the condition as *Mamsa dhatu kshaya* with *Vata prakopa* (*Mamsagata vata*), with a probable *Sahaja* (hereditary)

component pending genetic confirmation. This interpretation is made by the authors and was not recorded in the source document.

Therapeutic intervention

After investigations, *Panchakarma* with *Shamana Chikitsa* was started. The *Panchakarma* procedures were *Abhyanga* with *Mahanarayana taila* or *Mahamasha taila*, *Swedana* in the form of *Shashtikashali pinda sweda*, and *Basti* with *Bala taila*, *Mustadi yapana basti* and *Sanvardhana ghruta* (Table 2). The record does not state the number of sittings, the quantity of medicine, the order of procedures or the date on which each was started.

Table 2. Panchakarma procedures as recorded, with their intended Ayurvedic action

Procedure	Material used	Intended action in this context
<i>Abhyanga</i>	<i>Mahanarayana taila</i> or <i>Mahamasha taila</i>	Oleation of the body with <i>Vata</i> -pacifying oils to reduce dryness and stiffness and to support <i>Mamsa dhatu</i>
<i>Swedana</i>	<i>Shashtikashali pinda sweda</i>	Nourishing sudation (<i>Brimhana sweda</i>) using boluses of cooked <i>Shashtika</i> rice, intended to strengthen muscle and improve circulation
<i>Basti</i>	<i>Bala taila</i> , <i>Mustadi yapana basti</i> , <i>Sanvardhana ghruta</i>	Medicated enemas regarded as the principal therapy for <i>Vata</i> ; <i>Yapana basti</i> is used to sustain strength and tissue without marked depletion

Oral *Shamana* and supportive medicines given during the stay and advised at discharge are listed in Table 3. A duration was recorded only for Q-Carni 10 syrup

(three months); for the other medicines the duration field was left as days without a number.

Table 3. Oral medicines given in hospital and advised at discharge, as recorded

Medicine	Dose and frequency	Remarks
Calcimax syrup	5 ml twice daily	Calcium supplement; given in hospital and advised at discharge
Q-Carni 10 syrup	5 ml twice daily for 3 months	Supplement; composition not stated in the record
Vitneurin syrup	5 ml morning and night	Vitamin supplement
<i>Vidaryadi kashayam</i>	10 ml morning and night	Advised at discharge
<i>Kamdudha vati</i>	2 tablets twice daily	Advised at discharge
<i>Sanjivani vati</i>	2 tablets twice daily	Advised at discharge
<i>Yogaraj guggulu</i>	1 tablet morning and night	Advised at discharge
<i>Brahmi, Shatavari, Ashwagandha and Shankhapushpi</i> combination	Half a unit morning and night	Advised at discharge; an earlier in-patient version contained <i>Guduchi</i> in place of <i>Shatavari</i>
Vitamin D3 sachet 60 K	Frequency not recorded	In-patient only
Cetirizine syrup and Chericoff Junior syrup	Frequency not recorded	In-patient only; symptomatic

Outcome measures

Outcomes were the course of serum creatine phosphokinase and other routine parameters, and the clinical status at discharge. No standardised functional scale was recorded.

RESULTS

Baseline investigations were obtained one day before admission (Table 4). Haemoglobin, leucocyte count, platelet count, electrolytes and serum calcium appeared to be within usual limits for age.

Serum creatine phosphokinase was markedly raised at 3528.1. Serum glutamic oxaloacetic transaminase (76.1) and serum glutamic pyruvic transaminase (106.1) were raised, and serum vitamin D3 was 18.54. Serum creatinine was 0.19. Lactate dehydrogenase was recorded as 399.88. Units and reference ranges were not printed on the discharge summary, so values are reproduced as recorded. The enzyme labelled CPK MB in the hospital record is reported here as creatine phosphokinase,

because values of this magnitude are consistent with the total enzyme.

Table 4. Laboratory investigations as recorded

Parameter	27 May 2026	16 June 2026	30 June 2026	14 July 2026
Haemoglobin	12.7	-	13.8	12.5
White blood cells	9800	-	9560	7910
Red blood cells	4.48	-	4.85	4.5
Platelets	329000	-	283000	2.82 (as recorded)
Serum creatinine	0.19	-	0.38	0.37
Serum calcium	9.7	-	-	-
Sodium	139	-	-	-
Potassium	4.27	-	-	-
Chloride	105.12	-	-	-
Serum glutamic oxaloacetic transaminase	76.1	-	-	-
Serum glutamic pyruvic transaminase	106.1	-	-	-
Serum vitamin D3	18.54	-	-	-
Creatine phosphokinase	3528.1	1429	1123	1308

Repeat creatine phosphokinase was 1429 on 16 June, 1123 on 30 June and 1308 on 14 July (Figure 1). Relative to baseline this is a fall of 59.5, 68.2 and 62.9 percent respectively. The decline was not steady, since the value rose by 185 between the last two measurements. According to the

record, transaminases and vitamin D were not repeated. Haemoglobin and cell counts remained stable, and serum creatinine was 0.38 and 0.37 on repeat testing. The platelet count on 14 July was recorded as 2.82, a figure that appears to be expressed in lakh per cubic millimetre.

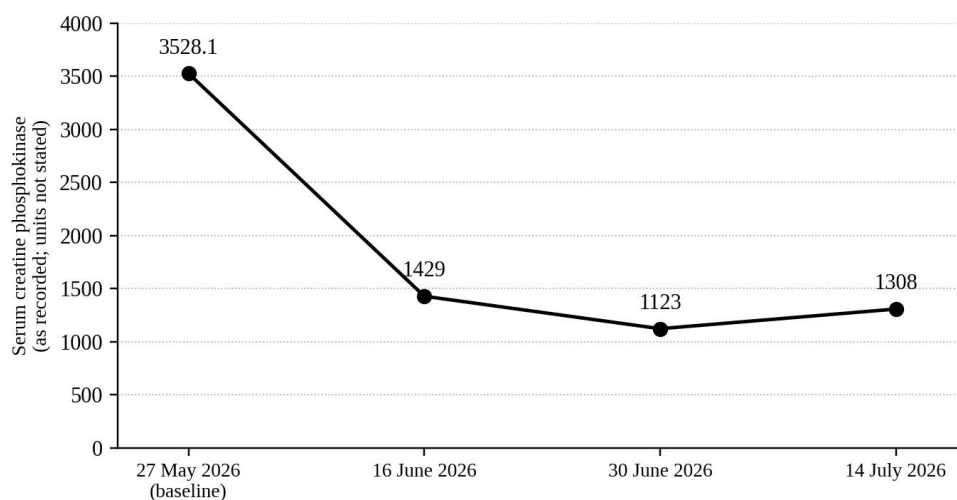


Figure 1. Course of serum creatine phosphokinase at four time points during hospital care. Values are as recorded; units were not stated in the source.

At discharge the child was haemodynamically stable. The discharge summary does not report any change in gait, Gowers sign, stair climbing or running, and no adverse event related to the procedures or medicines is recorded. Follow-up with the treating consultant on 28 July 2026 and an appointment on 23 July 2026 with genome sequencing reports were advised.

DISCUSSION

Ayurvedic interpretation

The presenting features of proximal weakness, falls and difficulty in rising correspond to loss of strength of *Mamsa dhatu*. Textual descriptions of *Mamsa kshaya* include wasting of the buttocks, thighs and trunk, dryness and weakness of the limbs [11]. The same texts explain that depletion of tissue leaves space for aggravated *Vata* to settle in the weakened tissue [9,10]. The relentless course of a hereditary disorder places it among the *Sahaja* conditions, which are said to arise from a defect in the *Beeja* (seed) and are

traditionally regarded as difficult to cure [12]. The therapeutic aim in such a case is therefore realistic: to nourish the tissue, to pacify *Vata*, to support *Agni* and to slow functional decline, rather than to promise reversal.

Rationale of Panchakarma

Snehana and *Swedana* are the standard preparatory measures for *Vata* disorders, and *Basti* is described as the foremost therapy for *Vata*, and is said to be half of all treatment or even the whole of it [13,14,16]. *Abhyanga* with *Mahanarayana taila*, a sesame oil preparation made with the *Dashamula* group and drugs such as *Bala* (*Sida cordifolia*) and *Ashwagandha* (*Withania somnifera*), and with *Mahamasha taila*, in which *Masha* (*Vigna mungo*) is a principal ingredient, offers local nourishment of muscle and relief of stiffness. *Shashtikashali pinda sweda* is a nourishing sudation in which cooked *Shashtika* rice (*Oryza sativa*) is prepared with milk and a decoction of *Bala* and applied in warm boluses; it is classically

used where muscle wasting and weakness predominate, because it combines heat with *Brimhana* substances. *Basti* with *Bala taila* and *Sanvardhana ghrita* provides an oleating, strength-giving enema, while *Mustadi yapana basti* belongs to the *Yapana* group, which is intended to maintain life, strength and tissue in chronic and debilitating states without the depletion that follows strong purification. Formulations such as *Mahanarayana taila* and *Yogaraj guggulu* are described in the *Vatavyadhi* chapters of the *Bhaishajya Ratnavali* [17], and *Vidaryadi kashayam* in the *Kashaya* section of the *Sahasrayogam* [18]. These choices are coherent with the stated principles of treatment, although the present record does not allow the contribution of each procedure to be separated.

Rationale of Shamana and supportive therapy

Vidaryadi kashayam is built around *Vidari* (*Pueraria tuberosa*) and is used for its *Brimhana* and *Balya* action. *Yogaraj guggulu* is a standard *Vatahara* formulation used in disorders of muscle and joint. *Sanjivani vati* acts as *Deepana* and *Pachana* and was presumably used to correct *Agni*, so that nutrition could be assimilated into *Dhatu*. *Kamdudha vati* is a *Pitta*-pacifying preparation that is also a source of calcium. The combination of *Brahmi* (*Bacopa monnieri*), *Shatavari* (*Asparagus racemosus*), *Ashwagandha* and *Shankhapushpi* (*Convolvulus pluricaulis*) is *Rasayana* in intent. *Ashwagandha* in particular is reported to have antioxidant and anti-inflammatory activity in experimental work [19]. Alongside these, the child received calcium, vitamin D3, a carnitine-containing supplement and vitamins, which are reasonable supportive

measures in a child with reduced mobility and a low vitamin D level.

Biochemical findings

A markedly raised creatine kinase is expected in dystrophic muscle, where fragile membranes leak enzyme into the circulation. The level is usually highest early in the course and tends to decline with age as muscle mass is lost, and it varies with physical activity [1,8]. Persistent elevation of creatine kinase is itself a recognised trigger for neuromuscular evaluation, and the level should be interpreted against activity, age and sex [20]. The fall seen here, from 3528.1 to 1308 over seven weeks, therefore has several possible explanations: natural variation, reduced exertion during a long admission, concurrent nutritional treatment and the Ayurvedic intervention. A 185-point rise between the last two readings also shows that the trend is not linear. The raised transaminases deserve comment because in muscle disease they originate from muscle rather than liver; this is a well-recognised source of unnecessary hepatic investigation, and a normal gamma-glutamyl transferase or a parallel rise in creatine kinase points to muscle as the source [21]. Repeat transaminases were not recorded, so the effect of treatment on them is unknown. A serum creatinine of 0.19 is low for age and is consistent with reduced muscle mass, which also means that creatinine-based estimates of kidney function can overestimate true function in children with low muscle mass [22]. The vitamin D level of 18.54, if expressed in nanograms per millilitre, falls in the deficiency range (below 20) defined by the Endocrine Society guideline [23]. Bone health is a recognised concern in neuromuscular disease with reduced mobility [24], so

correction of the deficiency was appropriate.

Comparison with contemporary supportive care

Current guidance for limb girdle muscular dystrophy stresses multidisciplinary supportive care: physiotherapy and stretching to limit contracture, periodic cardiac and pulmonary assessment, nutritional support, attention to bone health and genetic counselling. Corticosteroids are not established as beneficial outside Duchenne-type disease [6,8]. The comprehensive care framework for Duchenne muscular dystrophy is a different disease, but it is widely used as a template for neuromuscular care, including the timing of cardiac, respiratory and bone assessments [24-26]. Against this background the present admission covered several elements: neurological opinion, an electrocardiogram and echocardiogram, calcium and vitamin D supplementation, and a genetic investigation. Elements that are not documented are pulmonary function testing, a structured physiotherapy programme, assessment of contractures and scoliosis, and a plan for periodic cardiac review. *Abhyanga* and *Swedana* may be regarded as complementary to physiotherapy and not as a replacement for it, and the child will need the standard surveillance regardless of the Ayurvedic treatment she receives.

Implications for practice and research

Ayurvedic treatment is typically multi-component, combining external procedures, internal medicines, diet and lifestyle, and any fair evaluation must accommodate this feature [27]. For a progressive genetic disorder, the most useful next step is likely to be well-

documented case series with a common minimum data set, rather than isolated reports. Such a data set would include the genetic diagnosis, a validated functional measure repeated at fixed intervals, muscle enzyme levels, cardiac and respiratory assessments, and the exact schedule of each *Panchakarma* course. Adherence to reporting guidelines makes such reports comparable and strengthens their value for later synthesis [15]. Co-management with neurology and rehabilitation services, as happened in this admission, is also the safest arrangement for the family, because it keeps standard surveillance in place while the Ayurvedic measures are given.

Strengths and limitations

The strengths of this report are the clear baseline and repeated measurement of a muscle enzyme, the transparent listing of every intervention, and an honest account of what the record does not contain. The limitations are substantial. This is a single uncontrolled case with several simultaneous interventions, so no causal inference is possible. Genetic confirmation was pending, which leaves the subtype and its expected natural history unknown. Functional status was not measured with a validated tool, and no muscle imaging, biopsy or cardiac results are reported. The number of procedure sittings, the doses and the post-discharge course are not available, and some laboratory entries lacked units or appear to have been mis-entered. Future reports should record age at onset, family history, muscle power grades, timed function tests and a disease-appropriate rating scale at baseline and at fixed intervals, together with repeated cardiac and respiratory assessment.

Conclusion

This case records the Ayurvedic in-patient care of a child with limb girdle muscular dystrophy in whom serum creatine phosphokinase fell by about 63 percent from baseline over seven weeks while she received *Panchakarma*, *Shamana* medicines and nutritional supplementation, and who was haemodynamically stable at discharge. Because the case is uncontrolled, functional outcome was not measured and the genetic subtype is unknown, no claim of efficacy can be made. The report is offered as a documented clinical experience and as a template for better-designed observations of *Panchakarma* in childhood muscular dystrophies.

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