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Management of Duchenne Muscular Dystrophy through Ayurveda: A Case Study

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Abstract

Duchenne muscular dystrophy (DMD) is the most common hereditary neuromuscular disease affecting all races and ethics groups. The incidence is 1 in 3500 male births. It is an x linked recessive disorder due to the absence or alteration of the dystrophin protein. Encoded by the DMD gene on chromosome xp21. Steroid therapy are the only medications that have shown to alter the course of DMD but are associated with there side effect such as weight gain, decreased appetite, increased tendency to develop cataract and osteoporosis. Another problem that remains unclear is when to cease steroid therapy. The present study is about management of 8 years old male child with difficulty in walking, frequent falls while walking, difficulty in sitting in squatting position, slipping of slippers for 6 years, progressive muscle weakness, hypertrophy of calves' muscles. Ayurveda it can be classified under *Adibalapravrittavyadhi* and the pathogenesis occur due to the *Beejbhaga avayavadushti* which leads to *vataprakopa* takes *sthanasamshraya* in *medomamsa dhatu* and deplete them. Based on ayurvedic principles, present case was managed with *samana* and *sodhana* for one sitting (45 days). After therapy, substantial improvement in CPK levels along with signs and symptoms was found. Though the genetic predisposition cannot be ruled out, the disease can be effectively managed by ayurvedic principles and lifestyle of the patient can be improved. The absence of specific treatment for muscular dystrophy makes it even more important to alternative approaches of treatment.

Keywords: Ayurveda, CPK, Duchenne muscular dystrophy, Dystrophin, Panchakarma,

Introduction:

The dystrophinopathies are X linked recessive disorders that are due to the absence or alteration of the dystrophin protein, encoded by the DMD gene on chromosome Xp21. DMD has a commonly cited, yet estimated, incidence of 1 in 3500 male births[1]. Deficiency of dystrophin leads to failure of muscle fibre integrity and to a degenerative process marked by myofiber necrosis, muscle fibrosis, and a failure of regenerative capacity. The result is the pathology of DMD. Fibrosis and fatty replacement, two of the classic components of the descriptions of dystrophic muscle, increase over time. DMD clinically manifests by the age of 5 years. It leads to wheelchair dependence by 10 to 12 years of age, and thereafter progresses relentlessly. At the time of presentation, proximal weakness is typically evident. Even at a young age, proximal muscle weakness is evident in difficulty in climbing stairs, hopping or arising from the floor. The latter is typically exemplified by Gowers' manoeuvre, which is always seen in DMD. Patients rise from the floor by climbing up the thighs with their hands. Other signs of DMD at presentation include muscle enlargement, first termed 'Pseudohypertrophy' by Duchenne who attributed the enlargement to increased fibrosis and fatty replacement rather than muscle fibre hypertrophy[2]. Contraction of Achilles tendons forces children to walk on their toes or on the balls of their feet. Mild lordosis is common. DMD is associated with elevations of serum creatinine kinase (CK), generally considered to be due to increased permeability of the muscle sarcolemmal membrane. In case of DMD, it is often 50-100 times the normal value at presentation. Even in the absence of an X-linked family history, diagnosis can be made on presentation alone with reasonable reliability. In our classical texts, the direct correlation of DMD with any single disease is not available. Since almost all major neuromuscular disorders are identified with Vata Dosha, this condition can be considered as

Adibala-Pravritta Mamsa Vata Kshaya due to *Srotorodha*. *Aatmakarmaja* and *Beejadosha* bring Khavaigunya at Mamsadhatu levels leading to vitiation of *Vata* which causes *Mamsadhatvagni* impairment. The depleted *Dhatvagni* forms *Aama* instead of proper *Mamsadhatu*. It is followed by vitiation of Kaphadosha. While *Srotorodha* produces hypertrophy in particular region, it also manifests as first *Prakopa* then depletion of *vata* ailment. This complex pathogenesis is responsible for progressive wasting and necrosis of the affected muscle fibres.

Case Report:

An 8 years old patient was brought to department of *Balrog* OPD with the complaints of that unable to stand from sitting position for 3 years, difficulty in walking, frequent falls while walking, difficulty in sitting in squatting position. As per his mother, he was her first child, FTND, having insignificant antenatal, history. The patient attained all developmental milestones as per chronological age. At the age of 5 years, he started developing weakness in limbs, affecting his gait. He was diagnosed with DMD 2 years back by allopathic paediatrician. The patient had medium appearance, toe walking and proximal weakness.

On Examination:

On general physical examination, the child had thin appearance, difficulty in getting up from sitting position, proximal weakness, calf muscle hypertrophy and positive *Gower's sign*. His Vitals was stable, CNS examination was in normal limit, Motor System- Fasciculation and irritability – Absent, Muscle tone – Normal, Muscle bulk - Wasting of muscle, B/L Pseudohypertrophy of calf muscle, Power grade >3/5 = both upper limb, Grade >3/5 - both lower limb, Gait - Short step gait, Gower's sign – present, Reflexes – Deep tendon reflex = normal, Plantar reflex = decreased *Dashvidha Pariksha:Prakriti*: *VataKapha Vikriti*: *Vatapradhanatridoshaja, Sara*: *Twak, Samhanana*: *Madhyama, Desha*: *Jangala, Satmya*: *Sarva Rasa, Satva*: *Madhyama, Ahara Shakti*: *Madhyama, Vyayama Shakti*: *Avara, Vayah*: *Kumara*

Investigation: Remarkable abnormality in CPK level: 8874 U/L

Treatment Plan:

Saman Chikitsa	Days
Panchkol Phant - 7 ml	4 Days
Chitrakadi Vati - 1 Tab	4 Days
Smriti Sagar Rasa – 1/2 Tab	For 45 Days
Aswagandharista -10 ml	For 45 Days
Vilwadi Gutika - 1 Tab	For 45 Days
Mansmitra Vatakam – 1/2 Tab	For 45 Days

Sodhan Chikitsa:	Days
Snehan & Nadi Swedan(Kshir Bala +Dasmoola Qwath Nadi Swedan)	Alternate Day 14 Days
Udawartan (Kolkulathadi Churn+ Triphala Churn)	Alternate Day 14 Days
Parishek (Dasmool Qwath)	15 Days
Parishek (Dasmool Kshir)	7 Days
SSPS	7 Days

Observation and Result:

CPK level was reduced noticeably, mentioned below. Along with improvement in CPK levels, improvement in symptoms was also appreciated by the patient. After 1st sitting, patient reported mild relief in generalized weakness and walking was mildly improved. Though the

patient was still unable to sit in squatting position. Patient appreciated relief in calf muscle pain and tightness. Mild improvement was also seen in sitting in squatting position. Walking was improved, and patient could walk or run without falling. There was no slipping of slippers by the end of treatment. Power in both the upper and lower limbs was improved to 4(+)/5 (Elevation against moderate resistance).

CPK LEVEL BEFORE AND AFTER TREATMENT U/L

S. No	Before Treatment	After Treatment
CPK	8874 u/l	1000 u/l

Discussion:

Muscular dystrophies are inherited, progressive muscle disorder results from defects in one or more genes needed for normal muscle function. Duchenne muscular dystrophy (DMD) is the most common and severe form of muscular dystrophy. DMD manifests as weakness affecting proximal muscles, typically that of lower limbs initially. Toe walking, waddling gait and lordosis are the later features. Most children are confined to wheelchair by the age of 14 years. No specific treatment for the same exists. Daily steroid does not cause significant longterm clinical improvement, but it possibly slows the course of the disease[4]. But these are associated with their side effects such as weight gain, decreased appetite, increased tendency to develop cataract and osteoporosis. Another issue that remains unclear is when to cease steroid therapy[2]. In Ayurveda no exact correlation can be found however there are certain references in classical texts which show similarity to DMD. Acharya *Kashyapa* in *Revatikalpadhyaya* has mentioned *Asadhya Jataharni*. One of them is Kulakshayakari, in which the female child survives easily, but the male children die[4]. Another reference with symptoms like that of DMD is mentioned by *Acharya Charaka* in *Vatavyadhi chikitsa adhyaya*. *Mamsakshaya* (depletion of muscular tissues), *Balakshaya* (deletion of strength) is a symptom of *Majjagatakupita Vata*. *Vyana Vayu* is responsible for the *Prasarana* (relaxation) and *Akunchana* (contraction) of the muscles[5]. In cases of DMD, due to weakness, muscle loses its ability to contract or relax, which can be attributed to the *Kupita Vyana Vayu*. Considering the genetic factor, *Beejabhagavyava dushti* can be thought of in Ayurveda. As the treatises say, nomenclature of a disease is not important[6]. Though it is impossible to cure a genetic deformity to restore dystrophin protein, yet efforts can be made to improve the lifestyle of patients suffering from DMD. *Acharya Charaka* emphasizes on fulfilment of the depleted *Dhatu* and depletion of the increased Dhatu[7]. *Mamsakshaya* and *Balakshaya* are the predominant manifestations in cases of DMD, which is a neuromuscular disorder. The Nervine functions are attributed to the *Vata*. Keeping the Ayurvedic principles in mind, the main aim in present case was to win over this *Kupita Vyana Vayu*, to provide nutrition to the muscles and body, improve the lifestyle of patient and in a way to stop further progression of the disease. *Abhyanga* with *kshir bala Taila* was planned, followed by *Shashtika Shali Pinda Swedana. Abhyanga* provides strength to body and tissues[8]. *Ashwagandha* is deputed to be *Balya*, *Brimhana*, is indicated in *Balashosha*, *Daurbalya* and *Vatavyadhi*[9]. *Shashtika Shali* imparts stability to the body *(Sthirata*)[10]. *Shashtika Shali Pinda sweda*, a form of *Snigdha Sankara Sweda*, is said to be *Shramahara* (removes fatigue), *Balakara* (offers strength) and *Pushtikara* (nourishes body)[11]. The schedule was followed for one sitting for 45 days. Along with these procedures, *Shamana Aushadha* was also advised, intended to magnify the body strengthening effects of panchakarma therapies being administered. As mentioned in observations, noticeable improvement was found in patient's gait; he could walk without falls. His mother was quite satisfied with the treatment.

Conclusion:

Duchenne muscular dystrophy is a neuromuscular disorder. There is no satisfactory treatment for the DMD in allopathic system of medicine. This article is an attempt to present a

case of Duchenne muscular dystrophy, effectively managed by Ayurvedic principles. All can be done to improve the quality of life in DMD patients. By observing the case study, it can be safely concluded that specific Panchakarma procedures along with internal medications can improve function, ambulation and thus increase life expectancy of the diseased child.

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