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A REVIEW ON CONCEPTUAL STUDY OF DUCHENNE MUSCULAR DYSTROPHIES: AN AYURVEDIC PERSPECTIVE

[1]*Dr. Komal Dhiman, [2]Dr. Reena Dixit and [3]Dr. Sujata Sharma

[1]M.D Second Year, Department of Kaumarbhritya Rishikul Campus, Haridwar Uttarakhand Ayurved University.
[2]Associate Professor, Department of Kaumarbhritya Rishikul Campus, Haridwar Uttarakhand Ayurved University.
[3]Assistant Professor, Department of Kaumarbhritya Rishikul Campus, Haridwar Uttarakhand Ayurved University.

***Corresponding Author: Dr. Komal Dhiman**

M.D. Second Year, Department of Kaumarbhritya Rishikul Campus, Haridwar Uttarakhand Ayurved University.

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ABSTRACT

Muscular dystrophies are a category of hereditary illnesses defined by degeneration of the skeletal muscles that control mobility, culminating in muscle atrophy, weakness, confinement to a wheelchair, and eventually death. The most common muscular dystrophy is Duchenne muscular dystrophy (DMD), caused by a lack of the dystrophin gene. In Ayurvedic approach it can be classified under *Adi-balapravritta vyadhi*. Pathogenesis occurs due to *Beejbhaga Avayavadushti* due to which *vata dosha prakopa* takes place at *medomamsa dhatu* and deplete them. There is no curative treatment to DMD in modern science but according to *Ayurveda*, *Panchkarma* therapies like *Snehana, Swedana* and *Basti* alongwith *Rasayana* (*Brihamana chikitsa*), are the important therapy in this disease. *Ayurveda* do not proclaim as curative but can be used to improve the quality of life of DMD patient. The current review article discusses the notion of muscular dystrophy from an *Ayurvedic* perspective.

KEYWORDS: *Adi-balapravritta vyadhi, Beejbhaga Avayavadushti, vata dosha prakopa, medomamsa dhatu, Panchkarma, Rasayana.*

INTRODUCTION

The Greek term **dystrophy (***dys* meaning aberrant and trophy meaning nourishment) implies aberrant growth or nutrition of muscle fibers. Muscular dystrophies are a heterogenous group of unrelated inherited disorders having different genetic traits and clinical course and expressions. Muscular dystrophies have four obligatory criteria that distinguish them from other neuromuscular disorders, namely

1. They are primary myopathies.
2. They have genetic basis.
3. They have progressive downhill course.

They have degeneration and death of muscle fibers at some stage.[1]Duchenne muscular dystrophy (DMD) is the commonest (1:3600 male livebirth) hereditary neuromuscular disease in Indian children affecting all races.[2]

Being the most common form of progressive muscular dystrophies, DMD is transmitted in an X-linked recessive manner (affecting only males and carried by females), manifests before the 5th year of life and generally proves fatal in the second decade.

In *Ayurveda,* the direct link of DMD with any particular disease is not present in our traditional books. Because *Vata dosha* is linked to practically all major neuromuscular ailments, this condition can be classified as *Adibala-pravritta vyadhi or Mamsagata Vata kshaya* due to *Shrotorodha* (obstructive pathology occurring in channels). *Aatmakarmaja* and *Beejadosha* increase *Khavaigunya* in the *Mamsa dhatu*, causing *Vata* vitiation and *Mamsadhatvagni* impairment. Instead of the proper *Mamsa dhatu*, the depleted *Dhatvagni* forms Ama It is followed by *Kapha dosha* vitiation. While *Shrotorodha* causes hypertrophy in a specific area, it also presents as *Prakopa* followed by *Vata* depletion. This complex pathogenesis is responsible for progressive wasting and necrosis of the affected muscle fibers.

ETIOPATHOGENESIS

Four hypothesis have been suggested about DMD pathogenesis-

- Muscle lesions are secondary to microinfarcts as a result of disordered circulation.
- Muscle lesion are due to neuronal dysfunction.
- Genetic defect in muscle surface membranes.
- Defective DNA repair mechanism as a result of insult from some DNA damaging agent, e.g. ionizing radiation or similar injury.

Pathologic changes include various stages of necrosis of muscle fibers, phagocytosis of degenerating fibers, abnormally small fibers and increase in fat and

connective tissue. Signs of regeneration eventually disappear.

Occasionally, DMD in its mild form can be encountered in girls. In girls with Turner's Syndrome (45 Xo), full blown DMD may occur when the X-chromosome has the Xp21 gene deletion.[3]

CLINICAL FEATURES

- Duchenne muscular dystrophy affects children before they reach the age of five, and they may have a history of delayed walking. At the age of 3-4 years, gait abnormalities become apparent. At this stage, **waddling gait**, **Gower sign**, and **calf muscle pseudohypertrophy** are classical findings.
- Infant boys are rarely symptomatic at birth or early infancy, although some are mildly hypotonic.
- The onset of neck flexor muscle weakening is quite early. Between the ages of 3 and 6, the progression of weakness may reach a halt. As a result, walking difficulties increases, contractures form, and lumbar lordosis increases.
- Natural history studies have found that in untreated Duchenne muscular dystrophy, the age at which independent ambulation is lost is between 8.8 and 10.5 years. There is worsening kyphoscoliosis, increased upper limb weakness, and bulbar dysfunction after the loss of ambulation.
- Variable degrees of intellectual incapacity and reduced gastric motility are further clinical characteristics of Duchenne muscular dystrophy.

Muscle Features- Hypertrophy of calf muscle is a striking feature; calf, glutei, deltoid, brachioradialis and tongue muscles may appear large, sternal head of pectoralis major, and supraspinatus are atrophied.

DIAGNOSIS- Even though DMD is first confirmed by the absence of dystrophin protein expression on muscle biopsy, a blood sample is usually required. The following tests should be performed to verify a DMD diagnosis:

1. **Multiplex PCR** is a standard genetic test for detecting dystrophin mutations.
2. Multiplex ligation-dependent probe amplification (**MLPA**).
3. Multiplex amplifiable probe hybridization.
4. **Creatine kinase** levels rise in conditions of active muscle fiber necrosis and injury, making it a good screening test for muscle disease in clinical practice.
5. **Electromyography (EMG)** shows reduced amplitude and duration of motor unit potentials.

DISCUSSION

According to *Ayurveda*, *Shukra* and *Artava* seeds have chromosomes with genes that symbolize the organs produced in the future. In Ayurveda, these are known as *Beeja* and *Beeja bhaga*. The *Beeja* (a division of sperm or ovum responsible for the production of a specific organ; the closest term in contemporary genetics is the Chromosome) or *Beejabhaga* (a component of *Beeja;* the most relative term in modern genetics is the Gene) are responsible for the development of the corresponding organs. When Doshas vitiate these, the corresponding derived Avayavas (organs) get deformed. Because *Mamsa dhatu* is a type of *Matruja bhava*, any V*ikrut*i to *Stree beeja* will increase the chances of Muscular Dystrophy. If the part of the Beeja (seed) responsible for the production of *Mamsa* dhatu becomes vitiated, *Mamsa dhatu* will become vitiated as well. If it is not vitiated, there would be no vitiation of the *Mamsa dhatu* either.[5]

Muscular Dystrophy is a Kind of ***Adibala pravrutta vyadhi. Adibala pravruttha vyadhi*** is a type of disease produced by the abnormalities of *Shukra* (semen vis-à-vis sperm) and *Shonita* (menstrual blood a vis-à-vis ovum) which leads to vitiation of *Beeja bhaga* and *Beeja bhaga avayava dusti*, resulting in Muscular Dystrophy manifestation.[6]

General Principles of Treatment and Prevention of (Muscular Dystrophy)

Muscular dystrophy is classified as *Meda-mamsadhatu dusthi* in Ayurvedic scriptures and vitiated *Vata dosha* plays a significant part in the disease. Line of treatment is mainly at three levels, *Shrotoshodhana*, which includes *Lekhana aushadhi* and *Dhatwagni deepan* and, *Dhatukshaya janya vatavyadhi chikitsa* (to enhance tissue metabolism) followed by *Brihana chikitsa*.

According to modern science there is no cure for any form of muscular dystrophy in today's medical science. Treatment only aids in the prevention and reduction of deformities. Maintaining strength and joint range of motion through exercise, physiotherapy, and avoiding extended immobility are the mainstays of management. Corticosteroids are the only therapies known to increase strength and prolong ambulation in children with DMD.

According to *Ayurveda*, there is no medical cure or method to slow down the progression. Maintain ambulation and prevent contractures is the only way to increase the lifespan of individual. But according to Ayurvedic literature, to breakdown the pathogenesis of the diseases, removing the *Shrotorodha* and pacifying the *Vatadosha* are important.

BAHIR PARIMARJAN CHIKITSA	***ANTAHA PARIMARJANA CHIKITSA***	***SHAMANA AUSHADI***	***RASAYANA***
1.Pachana (aama) ***2.Udvartana*** ***3.Abhyanga-*** *Vatahara, Brumhana Snehas like Mahamasha taila, Balashwagandha taila, Ksheerbala taila, lakshadi taila, chandanbalalaxadi taila.* ***4.Swedana-*** *shastika shali pinda sweda, masha pinda sweda etc*	***1.Matra basti/Sneha basti/Anuvasana basti*** *with bruhmana dravyas like ashwagandha ghrita, ksheerbala taila.* ***2.Yapana basti:*** *as it is Balya, Brimhana*	*Ashwagandha churna, Shatavari churna. Ashwagandharishta, Balarishta, Dashamoolarishta, Draksharishta, Pippalyasava, Arvindasava. Ekangveera rasa, Vasanta kusumakara rasa, Kushmanda-ghrita, ashwagandha ghrita.*	*Ashwagandha rasayana, Kushmanda rasayana, Chyavanprasha rasayana, Agastya rasayana.* *Vidharyadigana dravya, Kakolyadigana dravya,laghupanchmoola dravya siddha kalpas are helpful.*

CONCLUSION

In Duchenne Muscular Dystrophy, where progression of disease is very fast and fatal and no cure is available in modern medicine, the *Ayurvedic* treatment proved to slow down the progression and bring out a mild improvement.

In the long-term management of DMD, *Purva-panchakarma* therapies *(Snehana*, *Shashti shali pinda Swedana*) combined with *Anuvasana basti* are beneficial. *Basti* should be administered to correct *Agn*i, balance Doshas, remove metabolic toxins from *Dhatu*, and provide nourishment to the various *Dhatus*. Single Ayurvedic drugs with properties such as *Medhya* (memory-boosting), *Balya* (strengthening), *Agnivardhana* (digestive & carminative), *Vatadoshahara* (*Vata* pacifying) and *Rasayana* (rejuvenating) are administered both internally and externally as a primary guideline for nourishment, followed by strengthening and rejuvenation of *Mamsadhatu.*

Thus, *ayurvedic* formulations in conjunction with *Panchakarma* therapies, play a significant role in preventing DMD complications.

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