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Ayurvedic management of Ducchen's Muscular Dystrophy - A Case report

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ABSTRACT

Ducchen's muscular dystrophy is most common X-linked recessive disorder affecting 30 in 100,000 live male births. The primary cause of this disease is mutations in Dystrophin gene which is essential for the structural and functional integrity of muscle. It is a progressive muscle wasting disease in which patients frequently develop contractures and lose the ability to walk between 6 and 12 years of age. With progressive disease most patients succumb to death from respiratory failure and cardiac dysfunction in their twenties. As this is a genetic disorder we can consider it as *Adibala Pravritta Vyadhi*. As *Mamsa Kshaya* is seen at some muscles and *Mamsa Vriddhi* at other this is an *Avarana Vata Vyadhi*. In both *Upsthambha* and *Nirupasthmbha Vatavyadhi, Basthi* is considered as prime choice of treatment. A Variety of *Ksheerabasti* in the form of *Kalabasti* is studied in this condition by taking subjective and objective parameters. As this has given better improvement with no adverse effects in the patient, it can be tried in large number of patients.

Key words: *Ducchene's muscular dystrophy, Ksheera Basti, Shastikashali Pinda Sweda.*

INTRODUCTION

Ducchene muscular dystrophy (DMD) is the most common form of all muscular dystrophies and it is an X-linked disorder. Patients experience difficulty in ambulation which steadily progresses to wheel chair confinement by the age of 12 and death between 25-30 years of age due to respiratory muscle weakness or cardiomyopathy. Lack of dystrophin, a structural sarcolemma protein that stabilizes the muscle fibre, causes muscle fibre degeneration, inflammation and fibrosis, clinically manifested as muscle weakness.[1] In Ayurveda this pathogenesis can be clearly understand by the concept of *Avarana Vata Vyadhi*. It is a *Adibala Parvritta Vyadhi*. Here pathogenesis occurs due to the *Bheeja Bhagaa Avayava Dusti* which leads to *Vata Parkopa* takes *Sthana Samshraya* in *Mamsa* and *Medo Dhatu* vitiates and depletes them. This *Agnimandya* caused at the level of the *Dhatus* leads to formation of *Ama*. While *Srotorodha* a subtype of *Srotodusti* produces the hypertrophy in the particular region, it also manifests as first *Prakopa* then depletion i.e. due to *Vata*. This complex variety of pathogenesis indeed is responsible for the progressive wasting and necrosis of muscle fibres. In both *Upsthambha* and *Nirupasthmbha Vata Vyadhi, Basthi* is considered as prime choice of treatment. *Ksheera Basti, Yapana Basti, Mamsarasa Basti, Madhutailika Basti* are indicated for *Gambhira Dhatu Gata Vata Vyadhi*. A Variety of *Ksheerabasti* in the form of *Kalabasti* is studied in this condition by taking subjective and objective parameters.

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CASE REPORT

A patient named Navadeep age 9 yrs. from Andrapradesh came to our JSS hospital with H/o of

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Karmaksahya of both upper and lower limbs since 3 years, *Gamaneashaktata* since 3 years associated with *Ruja* in all 4 limbs. According to the statement of his mother, he was born as a full term baby by LSCS (cord around the neck). The Anti natal, perinatal and post natal period was uneventful. All the developmental milestones were normalup to the age of 6years, according to the history given by mother.

Later the boy started complaining of pain and weakness of both lower limbs and gradually he started walking on toes, difficulty in walking with frequent falls. Difficulty in getting up from the floor and climbing stairs. Then they approached NIMS Hyderabad and AIIMS New Delhi, there he was diagnosed as DMD, but there was no improvement in the condition with treatment. After few months patient attenders noticed weakness in both upper limbs also.

- O/E - Vitals normal
- CNS examination: Higher mental functions - Normal
- Cranial nerve examination - Normal

Motor system

1. Tropical changes: darkening and roughness of skin fold on the knees and ankle joints.
2. Fasciculations and irritability: Absent
3. Muscle tone: Normal
4. Muscle bulk: Mild hypertrophy of calf muscles
5. Muscle power: Grade 3/5 in both upper limbs, Grade3/5 in both lower limbs
6. Contractures: Present in both knee joints and ankle joints
7. Involuntary movements: Absent
8. Co-ordination: Finger - nose test left and right hand possible
9. Knee - heel test not possible on both lower limbs
10. Clonus - Absent
11. Posture and gait - unable to walk, patient can stand with support, Genu Varum deformity.

Reflexes

1. Visceral reflexes - Bowel and bladder undercontrol
2. Superficial

    Glabellar tap - Negative

    Abdominal reflex - areflexia

    Babinsky sign - Plantar flexion

    Corneal reflex - Intact
3. Deep tendon reflexes –diminished

Sensory system - Normal

Investigation's

DNA study shows deletion of Exon 56 in the dystrophin gene

Serum C.P.K - 14,570 IU/L (normal-24-190IU/L)

Brief about DMD

DMD is also called pseudo hypertrophic muscular dystrophy,has an incidence of 30 per 100000 live born males.[2]

Clinical features

The boy falls frequently and have difficulty keeping up with friends when playing.By age 5 yrs muscle weakness is obvious by muscle testing, On getting up from the floor,the patient uses his hands to climb up himself (Gower'smanoeuvre). Contractures of the heel cords and iliotibial bands become apparent by age 6 years, when toe walking is associated with a lordotic posture. By age 16-18 years, patients are predisposed to serious, sometimes fatal pulmonary infections other causes of death include aspiration of food and acute gastric dilation.

Investigations

- Serum CPK invariably elevated to between 20-100 times normal.
- EMG demonstrates features typical of myopathy.
- Muscle biopsy shows muscle fibres of varying size as well as small groups of necrotic and regenerating fibres.

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- A definitive diagnosis of DMD can be established on the basis of dystrophin deficiency in a biopsy of muscle tissue or mutation analysis on peripheral blood. The most common gene mutation is adeletion.[3]

Treatment

Glucocorticoids, administered as prednisone in a dose of 0.75 mg/kg, significantly slow progression of DMD for up to 3 yrs.

Ayurvedic Approach

Two sittings of treatment was given in the interval of 2 months.

Ksheera Basti a form of *Niruhabasti* was alternate with *Anuvasana Basti* was given in the *Kalabasti* pattern for 16 days inthe morning time.

Niruha Basti

- *Makshika - 50ml*
- *Saindhava - 2gms*
- *Mahasneha - 50ml*
- *Satapuspha kalka - 10 gms*
- *Ashwaganda, Bala, Shatavari Ksheera Kashaya 200ml*

Anuvasana Basti

- *Mahasneha - 50ml*

Sarvanga Shastika Shali Pinda Sweda with Mahamasataila Abhyanga for 20 days in the evening time.

OBSERVATION

Patient was well tolerated to this *Niruha Basti* dosage there was no complications occurred during *Basti Karma*. The retention time for *Niruha Basti* is 10-15 minutes, and for *Anuvasanabasti* it is 3-4 hours.

RESULTS

Serum CPK is the indicator for assessing the reduced muscle destruction and prognosis in DMD the changes in serum CPK level before and after the treatment summarised as follows .

Objective improvements

Serum CPK	Before	After
1st Sitting	14,570IU/L	7490IU/L
2nd Sitting	7180IU/L	5620IU/L

Subjective improvements

1. Patient can straighten his legs freely
2. Patient able to get up from the bed without support
3. Patient can able to keep his foot firmly on the ground

DISCUSSION

When patient came to our hospital there was no *Avarana Lakshanas* only *Vata* predominant features are present in the patient, so we planned for *Ksheerabasti. Basti Karma* is a prime treatment modality in *Vatavyadhi. Basti* importance in *Sarvanga Vata* mentioned in *Susruta* as forceful wind is opposed by mountain, like wise *Vata* can be controlled with *Basti Chikitsa.*[4] In this study *Ksheerabasti* is prepared with *Ashwagandha, Bala, Shatavarichurna* in the dosage of 25 gram was boiled with 200ml of *Ksheera* and 800ml of water was reduced to 200ml of *Ksheerakashaya*. For *Vata Dosha Snigda Basti* is indicated.[5] Drugs used in this *Basti* are *Balya, Bruhmana* and have *Vatahara* property[6] which help in controlling *Vatadosha*, which in turn prevents muscle damage and improves functional ability. For *Anuvasana Mahasneha* from *Vaidyaratnam* pharamacy was used it is combination of *Chathursneha (Taila, Ghrita, Vasa, Majja) and Vatahara Dravyas* indicated in *Asthanga Hrudaya Vatavyadhi Adhyaya.*[7]

Shastikashali Pinda Sweda with *Maha Masa Taila Abhyanga* was advised. *Mahamasha Taila* specially indicated when there is *Stabdata and Sankocha.*[8] *Shastikashali* having the properties of *Sheeta Virya, Snigda, Laghu, Madhura, Tridoshashamaka.*[9] *Swedana* with *Shastikashali* helps in removing *Srotoavaroda* there by improves the normal functions

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of *Vata*. DMD is due to genetic defect there is no hope for restoring dystrophin production but this research work shows reduced serum CPK level 7080 IU in the first sitting, 1560 IU in the 2nd sitting of treatment this could be a sign of decreased muscle damage.

CONCLUSION

Ksheera Basti in the form of *Kala Basti* with *Shastikashali Pinda Sweda*, treatment has given significant improvements in subjective and objective parameter in the patient of Duchene's muscular dystrophy. Its effect can be further studied in large number of patients.

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