

DUCHENE MUSCULAR DYSTROPHY (DMD) -A SINGLE CASE STUDY WITH AYURVEDIC MANAGEMENT

¹*Dr. Rajarshichaudhuri and ²Dr. Tapas Bhaduri

¹Lecturer in the Dept. of Panchakarma, I.P.G.A.E & R at SVSP, Kolkata.

²Lecturer in the Department of Kayachikitsa, R .D. Memorial Smriti Ayurved Medical College and Hospital, Bhopal.

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***Corresponding Author**

Dr. Rajarshichaudhuri

Lecturer in the Dept. of
Panchakarma, I.P.G.A.E & R
at SVSP, Kolkata.

ABSTRACT

Duchene Muscular Dystrophy (DMD) is a recessive hereditary X linked chromosomal disorder in which skeletal muscles affected. In Duchene muscular dystrophy, dystrophin is almost totally absent; the less dystrophin that is produced, the worse the symptoms and etiology of the disease. Muscular dystrophy can occur at any age, but most Diagnosis occur in childhood. Young boys are more likely to have this disease than girls. There is no known cure for muscular dystrophy, but certain treatments may help. Individuals require a wheelchair before their teenage. The life expectancy for those with this disease is late

teens or 20s. In Ayurveda it can be classified under adibalapravritta vyadhi. Beeja Bhaga Avya Dusti leads to mamsamedadhatudusti. Mamsa meda gata vata can be correlated with DMD. Aggravation of vayu in muscle and fat tissue gives rise to the signs and symptoms like heaviness of the body, excessive pain in the body, excessive Fatigue.

INTRODUCTION

Duchene Muscular Dystrophy (DMD) is a recessive hereditary X linked chromosomal disorder in which skeletal muscles affected. DMD affects 1 in every 3600 to 6000 boys of life birth, commonly it occurs as a result of mutation in dystrophin gene. Progressive loss of Dystrophin Glyco Protein complex leads to the muscle wasting. Muscular dystrophy is caused by mutations on the X chromosome. Each version of muscular dystrophy is due to a different sets of mutations, but all prevent the body from producing dystrophin. Duchenne muscular dystrophy is caused by specific mutation in the gene that encodes the cytoskeletal protein dystrophin. Dystrophin makes up just 0.002 percent of the total proteins in striated

muscle, but it is an essential molecule for the general functioning of muscles. Dystrophin is part of an incredibly complex group of proteins that allow muscles to work correctly. If dystrophin is absent or deformed, this process does not work correctly, and disruption occurs in the outer membrane. This weakness of the and can also actively damage the muscle cells themselves. In Duchenne muscular dystrophy, dystrophin is almost totally absent; the less dystrophin that is produced, the worse the symptoms and etiology of the disease. In Ayurveda it can be classified under *adibalapravrittavyadhi*. *Beeja Bhaga Avya Dusti* leads to *mamsa meda dhatu dusti*. DMD is characterized by onset of muscle weakness usually before 4 years of age. Skeletal muscle involvement mainly pelvis and pectoral girdle hypertrophy of calf muscle, grossly elevated serum CPK levels, progressive weakness of muscle leading to inability to work within 10 years after onset of disease. There is no specific cure – management or medicine in any system of medicines and death usually occurred before the age of 20 years caused by cardio respiratory failure. Muscular dystrophy is a group of inherited diseases that damages and weakens your muscles over time. This damage and weakness is due to the lack of a protein called dystrophin, which is necessary for normal muscle function. In Ayurveda it can be correlated with *mamsa meda gata vata* in which there is excessive destruction of muscle and fat leads to heaviness of the body and excessive pain and fatigue. DMD comes under *vatavyadhi*. Prognosis of this disease is *yappya* (not curable but manageable). Treatment principle of *mamsa meda gata vata* is *virechan*. *Niruhavasti*, and *samsaman* therapy. On the basis of these principles we are planning for treatment.

Symptoms

- Trouble walking, a waddling gait
- Difficulty in standing up, frequent fall
- Pain and stiffness in the muscles, loss of reflexes
- Difficulty In running and jumping
- Difficulty sitting up or standing,
- Bone thinning
- Scoliosis which is an abnormal curvature of spine
- Mild intellectual impairment
- Breathing problems can become so severe that assisted breathing is necessary
- Difficulty of swallow, with a risk of aspiration pneumonia. A feeding tube is sometimes necessary.
- The muscles of the heart can be weakened, leading to cardiac problems.

- Learning disabilities, such as developing of speech later than usual
- **DIAGNOSIS**
- Blood TEST for the enzymes released by damaged muscles
- Blood test for the genetic markers of muscular dystrophy
- An electromyography test on muscle's electrical activity
- Muscle biopsy to test a sample of muscle for muscular dystrophy

There is currently no cure for muscular dystrophy, but treatment can help manage your symptoms and slow the progression of the disease. Treatments depend on your symptoms.

Treatment options include

- corticosteroid drugs, which help strengthen the muscles and slow muscle deterioration
- assisted ventilation if respiratory muscles are affected
- medication for heart problems
- surgery to help correct the shortening of your muscles
- surgery to repair cataracts
- surgery to treat scoliosis
- surgery to treat cardiac problems

Physical therapy

- **General exercises:** A range of motion and stretching exercises can help combat the inevitable inward movement of the limbs as muscles and tendons shorten. Limbs tend to become fixed in position, and these types of activities can help keep them mobile for longer. Standard low-impact aerobic exercises such as walking and swimming can also help slow the disease's progression.
- **Breathing assistance:** As the muscles used for breathing become weaker, it may be necessary to use devices to help improve oxygen delivery through the night. In the most severe cases, a patient may need to use a ventilator to breathe on their behalf.
- **Mobility aids:** Canes, wheelchairs, and walkers can help the person stay mobile.
- **Braces:** These keep muscles and tendons stretched and help slow their shortening. They also give added support to the user when moving.

Case history – Master M sk 12 years old muslim patient came in the OPD at IPGAE&R at SVSP hospital Kolkata, Reg no AYR/RG 1800018813 card no AYUR/ OR180018600 on

01.09.2018 at 10:57 am with complaining of unable to walk and stand without any back support for last 6 years. His body weight 20 kg, on general examination it was revealed that growth retardation, idiotic faces, no pallor, no icterus, no cyanosis, pelvic girdle muscle weakness +++, calf muscle stiffness ++, patient was dull looked, his intelligence was dull, calf muscle was hypertrophied, CPK level was 174994 unit/l. Medical history revealed that he is a known case of DMD, diagnosed at Bangur Institute of Neurosciences on 27/01/2016 Govt of West Bengal Kolkata. His problems were noticed by his family members at the age of 4 when he was not properly walking or standing without any back support. Previously his parents were told that his initial complaint was difficulty to stand from squatting position and recurrent fall. Local doctor treated symptomatically but complaint not cured. After that he went to different hospital (Private and govt hospital) for seeking treatment.

On 18/10/2011 Initial investigation were HB% 12.12 gm/dl, RBC 3.58 mil /cumm, WBC 9000/cumm, N 71 L25 M01 E 03, ESR 40 MM 1ST HR 72 MM 2ND HRS, T3 1.25 ng /ml, T4 6.51 ug/dl, TSH 7.03 uIU /ml (ultra sensitive TSH assay instrument used)

On 25/01/2012 T3 1.42 ng /ml, T4 9.50 ug/ml, TSH 1.27 uIU/ml

On 09/04/2012 T3 0.85 ng /ml, T4 6.61 ug/ml, TSH 5.46 uIU/ml,

On 13/06/2012 serum CPK 17499 unit /L (method NAC UV IFCC) free T4 2.03 ng /100ml, TSH 0.13 micro IU/ml (ultra sensitive TSH chemiluminescence immunoassay Architect, Abbott)

On 12/12 /2012 CPK total 12000 IU/L (method IFCC)

MRI study on 08/01/2014 of left knee joint and adjacent leg showed no obvious lesion.

MRI study on 29/12/2014 lumbar spine showed mild straightening of lumbarvertebrae and CT brain also showed within normal limits.

On 03/07/2015 EMG sampled muscles shows small duration polyphasic and low amplitude MUPS with early fall recurrent and complete interference pattern, generalized primary muscle disease.

Genetic counseling was done on 13/01/2016.

Before treatment CPK 8580 U/L, LDH 3066 U/L

During treatment CPK 1253.8 U/L (28/04/2018)

CPK 1078 U/L (18/06/18)

After treatment CPK 217 U/L, CPK MB 23 U/L (16/09/2018).

Treatment regimen was given

- Sandhavadi oil local massage for 1 month
- Trikatu churna 1 gm twice a day for 1 month with Dasamooladikwath 10 ml
- Aswagandha churna 1 gm + yastimadhu powder 250mg + pravalpisti 125mg + muktasuktipisti 125 mg + Punarnava Mandur 125 mg with honey

After 1 month saindhavadi oil was replaced by Balaaswagandha oil for massage.

Eranda oil 10 ml with milk every day at night.

Dasamooladi niruhavasti 250 ml followed by Matravasti with ksheerbala oil 30 ml as Kala vasti procedur.

During the first follow up of 1 month it was noticed that clinical symptoms of muscle stiffness specially calf muscle becomes soft and normal shaped. Veins are prominent in legs and general Condition gradually improved day by day. After 4 months Patient can not walking but patient is able to sit without support on his bed.

In nutshell patient symptomatic improve day by day. Though it was a single case study but need more patients to conclude on this topic.

BIBLIOGRAPHY

1. Yiu EM, Kornberg AJ. Duchenne muscular dystrophy *neurology* India, 2008; 56: 236-47. <http://dx.doi.org/10.4103/0028-3886.43441> PMID:18974549.
2. Bushby K, Finkel R, Birnkrant DJ, Case LE, Clemens PR, Cripe L, et al. Diagnosis and management of Duchenne muscular dystrophy, part 1: diagnosis and pharmacological and psychosocial management. *Lancet Neurol*, 2010; 9: 77-93. [http://dx.doi.org/10.1016/S1474-4422\(09\)70272-8](http://dx.doi.org/10.1016/S1474-4422(09)70272-8).
3. Nowak KJ, Davies KE. Duchenne muscular dystrophy and dystrophin: pathogenesis and opportunities for treatment. *EMBO Rep*, 2004; 5: 872-6. <http://dx.doi.org/10.1038/sj.embor.7400221> PMID:15470384 PMID: 1299132.
4. Koenig M, Hoffman EP, Bertelson CJ, Monaco EP, Feener C, Kunkel LM. Complete cloning of the Duchenne muscular dystrophy DMD cDNA and preliminary genomic organization of DMD gene in normal and affected individual. *Cell*, 1987; Jul 31; 509-17. [http://dx.doi.org/10.1016/0092-8674\(87\)90504-6](http://dx.doi.org/10.1016/0092-8674(87)90504-6).
5. Sushruta, Sushruta Samhita, with Dalhanacharya. In: Acharya YT ed. Nibandha Sangraha, Commentary. Reprint ed Vaanasi: Choukhambha orientalia, 2009; 113-114.

6. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: AcharyaYT, ed Ayurved Dipika, Commentary. Reprint ed. Varanasi: Chaukambha Surbharati Parkashan, 2008; 321-22.
7. Agnivesa, Charak Samhita, with Chakrapaanidatta. In: Acharya YT, ed Ayurved Dipika, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia, 2009; 617.
8. Sharma A. Madhavnidanam with Madhav vimarshini Commentary. 1st_edition, Varanasi: Chaukhambha Sankrit Pratishthan, 2007; 198-99.
9. Agnivesa, Charak Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika Commentary. edition, Varanasi: Chaukhambha Bharati Acadamy; 2011 reprint. p. 793.
10. Shastri K, Chaturvedi G. Charak Samhita with Vidyotini Commentary. Edition, Varanasi, Chaukhambha Bharati Acadamy; 2011 reprint. p. 793.
11. Agnivesa, Charak Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2009. p. 620.
12. Vagbhata, Astang Hridaya, with Arundatta. In: Kunte AM, ed. Sarvangasundaya, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 225.