Review Article

www.ijrap.net

ROLE OF PANCHAKARMA IN DUCHENNE MUSCULAR DYSTROPHY

Chaturvedi Ashutosh^{1*}, Rao Prasanna N², U Shailaja³, M Ashvini Kumar⁴

¹PG Scholar, ²Principal, ⁴Associate Professor and Head, Department of PG studies in Panchakarma, ³Professor and Head, Department of PG studies in Kaumarbhritya, SDM College of Ayurveda & Hospital, Hassan, Karnataka, India

Received on: 02/12/12 Revised on: 20/01/13 Accepted on: 10/02/13

*Corresponding author

E-mail: drashutoshchaturvedi@hotmail.com

DOI: 10.7897/2277-4343.04238

Published by Moksha Publishing House. Website www.mokshaph.com

All rights reserved.

ABSTRACT

Duchenne muscular dystrophy (DMD) is an X-linked recessive disorder that affects muscles and causes progressive weakness. Dystrophin, the product of the DMD gene, is one of several membrane proteins that form the dystrophin–glycoprotein complex, which helps to maintain the integrity of muscle cells; loss of these proteins leads to the wasting of muscle. In Ayurveda it has been classified under Adibala Parvitta Vyadhi. Here pathogenesis occurs due to the Bheejabagahaavyava Dusti which lead to Medomamsa dusti further vitiates the Vata. Panchakarma shows great improvement among which Virechana and Basti explained in the principle of Vata chikitsa. This pioneer approaches gives patient quality of the life and longer survival upon muscular dystrophy.

Keywords: DMD, Medomamsa Dusti, Panchakarma, Basti, Virechana, Snehana, Swedana.

INTRODUCTION

Duchenne muscular dystrophy (DMD) is a recessive hereditary disease, linked to the X chromosome, which affect skeletal muscles, heart and brain, with progressive evolution until death around the second decade, generally caused by cardio respiratory events.¹ DMD affects one in every 3,600 to 6,000 boys of live birth and it occurs as a result of mutations in the dystrophin gene (locus Xp21.2).² Around 1/3 of all new cases diagnosed arise from “new” mutations.^{3,4} Dystrophin is a large structural protein (427 kDa) whose function is to connect the internal cytoskeleton of the skeletal fiber with the extracellular matrix proteins, stabilizing muscular contraction.⁵ In DMD, the protein is absent or dysfunctional, thus resulting in imbalance in the integrity of the lipid bilayer of the membrane, with influx of calcium and cellular necrosis.^{6,7}

When to suspect DMD

Suspicion of the diagnosis of DMD should be considered irrespective of family history and is usually triggered in one of three ways: (1) most commonly, the observation of abnormal muscle function in a male child; (2) the detection of an increase in serum creatine kinase tested for unrelated indications; or (3) after the discovery of increased transaminases (aspartate aminotransferase and alanine aminotransferase, which are produced by muscle as well as liver cells). The diagnosis of DMD should thus be considered before liver biopsy in any male child with increased transaminases. Initial symptoms might include delayed walking, frequent falls, or difficulty with running and climbing stairs. Although DMD is typically diagnosed at around 5 years of age, the diagnosis might be suspected much earlier because of delays in attainment of developmental milestones, such as independent walking or language; such delays have been documented prospectively by following patients with DMD identified by newborn screening.⁸ The presence of Gower's sign in

a male child should trigger the diagnostic investigation of DMD, especially if the child also has a waddling gait. Toe walking might be present but is not additionally helpful in deciding whether to suspect DMD. In the presence of a positive family history of DMD, there should be a low threshold for testing creatine kinase, although this will be influenced by the age of the child. In a child less than 5 years of age, suspicion of DMD probably cannot be excluded completely by a normal muscle examination. However, with increasing age, a normal muscle examination renders the chance of a child having DMD progressively less likely. A boy older than 10 years of age with normal muscle function is thus highly unlikely to have DMD.

Confirmation of the diagnosis

The route to confirming the diagnosis depends on local availability of rapid and reliable testing, which must be interpreted alongside the clinical presentation owing to the range of severity possible with dystrophin mutations. Testing for a DMD mutation in a blood sample is always necessary even if DMD is first confirmed by the absence of dystrophin protein expression on muscle biopsy. The results of genetic testing provide the clinical information required for genetic counseling, prenatal diagnosis, and consideration for future mutation-specific therapies. Different types of mutations in DMD can be the genetic basis for DMD.⁹ The genetic tests commonly used to identify dystrophin mutations are multiplex PCR,¹⁰ multiplex ligation-dependent probe amplification,¹¹ single-condition amplification/internal primer,^{12,13} and multiplex amplifiable probe hybridisation.¹³ Multiplex PCR is widely available and the least expensive, but only detects deletions and does not cover the whole gene, so that a deletion might not always be fully characterized. Multiplex ligation-dependent probe amplification and amplifiable probe hybridisation will detect deletions and duplications and cover all exons, and single-condition

amplification/internal primer will detect deletions and provide sequence data. None of these techniques is universally available. If analysis by one or more of these techniques leads to the identification and full characterization of a dystrophin mutation, then no further testing is required. If deletion/duplication testing is negative, then dystrophin gene sequencing should be done to look for point mutations or small deletions/insertions.^{12, 13} Full characterization of the mutation (deletion endpoints or exact position of any point mutation) is required to allow correlation of the predicted effect of the mutation on the reading frame of the gene, which is the major determinant of the phenotypic variability seen in dystrophinopathy,¹⁴⁻¹⁶ as well as to determine eligibility for the mutation-specific treatments currently in trials.^{17, 18} A muscle biopsy could be done, depending on the clinical situation, availability of genetic testing, and the facilities in the centre where the patient is seen.¹⁹ A needle biopsy might be appropriate if testing is only for DMD or if the clinician is skilled in taking multiple cores of tissue from pediatric patients.^{20,21} In those centers where it is done, the conchotome technique has the advantage of providing a larger sample than a single-core needle biopsy, and does not require an open surgical procedure.^{22,23} The key tests done on the muscle biopsy for DMD are immunocytochemistry and immunoblotting for dystrophin, and should be interpreted by an experienced neuromuscular pathologist. A muscle biopsy can provide information on the amount and molecular size of dystrophin as long as the protein is present.¹⁹ Differentiating total and partial absence of dystrophin can help to distinguish DMD from a milder dystrophinopathy phenotype. Electron microscopy is not required to confirm DMD. Genetic testing after a positive biopsy diagnosis of DMD is mandatory. A muscle biopsy is not necessary if a genetic diagnosis is secured first, particularly as some families might view the procedure as traumatic. However, if genetic testing has been done and no mutation identified, but creatine kinase concentrations are increased and signs or symptoms consistent with DMD are present, then the next necessary diagnostic step is to do a muscle biopsy. This is also the case if there is a family history of DMD and a suspicion of the diagnosis, but no family mutation is known. Whereas electromyography and nerve-conduction studies have been a traditional part of the assessment of a child with a suspected neuromuscular disorder, these tests are not believed by the expert panels to be now indicated or necessary for the specific assessment of DMD.

Ayurveda and DMD

In Ayurveda this pathogenesis can be clearly understood by the concept of Adibala Parvritta Vyadhi viz. Sushruta's vyadhi vargikarana²⁴. Here pathogenesis occurs due to the Bheejabagahaavyava Dusti which leads to Vata Parkopa takes sthana samsraya in Mamsa and medo Dhatu vitiates and depletes them (x-linked progressive degenerative disorder of muscle tissue)²⁵. Acharya Charaka has clearly mentioned about the close relation of both Mamsa and Medo Dhatu Viz. to Dhatukshayaz vata pathogenesis which in turn degrades and causes the Dusti²⁶ (a defect in the sarcolemmal

membrane). This Ansha-ansha kalpana of the Dhatus clearly signifies the involvement of the Dhatuvagni Mandhya causes Kshaya. This agnimandya caused at the level of the Dhatus leads to formation of Ama. Madhavkara explained Srotodusti as type of Ama itself²⁷. While Srotorodha a subtype of srotodusti produces the hypertrophy in the particular region, it also manifests as first parkopa then depletion i.e. due to vata. This complex variety of pathogenesis indeed is responsible for the progressive wasting and necrosis of muscle fibers. Therefore it was well understood thousands of years back with its severity and termed as Ashadya²⁸.

Panchakarma in DMD

In India, with this incidence and no cure in contemporary system of medicine, patients of DMD approaches Ayurveda with lots of hope. In Ayurveda for the management of this disorder concept of the paraspar dhatu paka is of prime importance whereas Acharyas have mentioned specific chikitsa sootra for the condition by considering its severity and importance which can easily be understood by the physicians²⁹.

Acharyas while explaining the dhatupaka avastha clearly signifies the importance of Agni which is whole and sole responsible for the formation of the next dhatus. Thus correction of agni should be done by administration of deepana and pachana dravyas in order to strengthen the process, doshas must be balanced and metabolic toxins must be eliminated from dhatus through panchakarma³⁰. The pre-operative process quoted by Acharyas has the concept of "Brhmanyastu mrudu langyet" that signifies the usage of Rukshana for better brihmana treatment modalities³¹ for example udvartana which helps in the removal of srotorodha and does Sthiri karana of angas. Pachana medicines are also explained as a mode of Rukshana chikitsa and it is also must in the treatment of DMD initially with deepana, like parishekha with Dhanyamla³²⁻³³.

Panchakarma the penta bio purifactory methods of Ayurveda i.e. Vamana, Virechana, Niruha, Anuvansan and Nasya are of prime importance.³⁴

Vamana of mrudu kind i.e. using the drugs like madana phala which has anapaitava as guna, has least complications, if the person is present with kapha sthana gata pitta or utkilstha kapha lakhans as it pacifies the vitiated kapha but also corrects the depleted medas³⁵⁻³⁶.

Another set of data shows usage of vacha as dravya for the vamana which signifies major improvement in pediatrics age for the neuromuscular disorder³⁷.

Virechana Karma of mrudu in nature explained under Vatsya upkarma has anulomana property and tridosahara property³⁸. Thus its repeated course is beneficial. Amritprasha ghrutha and Tikta ghrutas are used as shodhana snehapana. Research has shown that Virechana does the detoxification which lead to better absorption of Rasyana Drugs, other Brihmana Dravyas and correction of Agni.³⁹

Basti is another variety of the Karma especially Brihmana variety of basti which clearly shows its efficacy in this condition for example usage of Mamsa rasa Basti and yapana basti (contains madhanaphala) with kala and karma format, considering the condition as gambhir dhatu

gata vikara⁴⁰.

Tiktika Ghruthas, Ashwanganadha ghrutha and Chagalayadi ghrutha can be administered as Anuvanasa basti⁴¹⁻⁴². It also rejuvenates the body and further helps in improving from the dhatukshaya caused due by the vata dosha that is why both virechana and basti are explained in the principle of Medomamsa dusti⁴³.

Nasya has less importance when we talk about genetic disorder however it is assumed that it can be used for the treatment of various associated symptoms like depression due to its mana prasdana action⁴⁴.

After the purification Rasyana therapy can be adopted. Not only these invasive therapies like virechana, Basti etc. but upkarma i.e Para panchakarma procedures are very much essential for the same. It is very well understood in the treatment principle of Vataroga by Charaka and Yogaratnakar that upakarmas like Abhyanga, Svedana are having prime treatment modalities⁴⁵. Snehana both bahya and abhyanga helps to pacifies the vata dosha⁴⁶. In contrast Abhyanga a variety of bhaya sneha with oil like Balaashwagandhalakshadi taila, Mahanaryana Taila and Mahamamsadi taila helps in subsiding the vata dosha, improves the tonicity of the muscle and compacts the body⁴⁷⁻⁴⁸. Whereas swedana like Shastikashaali pinda swdana also improves the tone of the body⁴⁹.

Swedana karma increases the metabolic activity which in turn increases the oxygen demand and blood flow. This vasodilatation stimulates the superficial nerve ending causing a reflex dilatation of the arterioles. Due to the effect of heat on the sensory nerve ending there will be a reflex stimulation of sweat glands in the areas exposed to heat. This rise in temperature induces muscle relaxation and increases the efficacy of muscle action as the increased blood supply ensures the optimum condition for the muscle contraction⁵⁰. Swedana also acts by the mechanism of thermoregulation regulated by skin and coordinated with the functions of the other excretory organs. It is supplied with many groups of nerves, which conduct various stimuli. The secretion of sweat is under nervous system control, especially autonomous. The hair of the skin is tactile sense organs and their secretion produces some nervous changes. Thus, swedana can bring about changes indirectly on the autonomic nervous system and the heat can bring about changes in conduction of nerve stimuli, by changing sodium-ion-concentration⁵¹.

Thus these modalities are of prime importance as no treatment acts on prime pathogenesis and present approach is taken to improve quality of life over muscular dystrophy.

CONCLUSION

The absence of specific treatment for muscular dystrophy in modern medicine demands the role of contemporary and alternative approaches of treatment. The Ayurvedic treatment with special reference to Panchakarma procedures followed by administration of rasayana Rasayana group of herbo-mineral or gold based medicine, yogic support have shown definite protective influence. Ayurveda never claims the cure of DMD with reference to asadhya whereas its unique or pioneer approach gives patients of DMD, quality of life and longer survival upon

muscular dystrophy²⁸.

REFERENCES

1. Yiu EM, Kornberg AJ. Duchenne muscular dystrophy Neurol 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 PMCID: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. Barbujani G, Russo A, Danieli GA, Spiegler AW, Borkowska J, Petruszewicz IH. Segregation analysis of 1885 DMD families: significant departure from the expected proportion of sporadic cases. Hum Genet. 1990;84:522-6. <http://dx.doi.org/10.1007/BF00210802> PMID:2338336
6. Hoffman EP, Dressman D. Molecular pathophysiology and targeted therapeutics for muscular dystrophy. Trends Pharmacol Sci. 2001 Sep;22(9):465-70. [http://dx.doi.org/10.1016/S0165-6147\(00\)01770-3](http://dx.doi.org/10.1016/S0165-6147(00)01770-3)
7. Wrogemann K, Pena SD. Mitochondrial calcium overload: a general mechanism for cell necrosis in muscle diseases. Lancet. 1976;1:672-4. [http://dx.doi.org/10.1016/S0140-6736\(76\)92781-1](http://dx.doi.org/10.1016/S0140-6736(76)92781-1)
8. Nicholson LV, Johnson MA, Bushby KM, Gardner-Medwin D, Curtis A, Ginjaar IB. Integrated study of 100 patients with Xp21 linked muscular dystrophy using clinical, genetic, immunochemical, and histopathological data. Part 2. Correlations within individual patients. J Med Genet. 1993;30:737-44. <http://dx.doi.org/10.1136/jmg.30.9.745>
9. Muntoni F, Torelli S, Ferlini A. Dystrophin and mutations: one gene, several proteins, multiple phenotypes. Lancet Neurol 2003;2: 731-40. [http://dx.doi.org/10.1016/S1474-4422\(03\)00585-4](http://dx.doi.org/10.1016/S1474-4422(03)00585-4)
10. Prior TW, Bridgeman SJ. Experience and strategy for the molecular testing of Duchenne muscular dystrophy. J Mol Diagn 2005; 7: 317-26. [http://dx.doi.org/10.1016/S1525-1578\(10\)60560-0](http://dx.doi.org/10.1016/S1525-1578(10)60560-0)
11. Lalic T, Vossen RH, Coff a J, et al. Deletion and duplication screening in the DMD gene using MLPA. Eur J Hum Genet 2005; 13:1231-34. <http://dx.doi.org/10.1038/sj.ejhg.5201465> PMID:16030524
12. Flanigan KM, von Niederhausern A, Dunn DM, Alder J, Mendell JR, Weiss RB. Rapid direct sequence analysis of the dystrophin gene. Am J Hum Genet 2003; 72: 931-39. <http://dx.doi.org/10.1086/374176> PMID:12632325 PMCID:1180355
13. Dent KM, Dunn DM, von Niederhausern AC, et al. Improved molecular diagnosis of dystrophinopathies in an unselected clinical cohort. Am J Med Genet A 2005; 134: 295-98. <http://dx.doi.org/10.1002/ajmg.a.30617> PMID:15723292
14. Manzur AY, Kuntzer T, Pike M, Swan A. Glucocorticoid corticosteroids for Duchenne muscular dystrophy. Cochrane Database Syst Rev 2008; 1: CD003725 PMID:18254031
15. Jeppesen J, Green A, Steffensen BF, Rahbek J. The Duchenne muscular dystrophy population in Denmark, 1977-2001: prevalence, incidence and survival in relation to the introduction of ventilator use. Neuromuscul Disord 2003; 13: 804-12. [http://dx.doi.org/10.1016/S0960-8966\(03\)00162-7](http://dx.doi.org/10.1016/S0960-8966(03)00162-7)
16. Yasuma F, Konagaya M, Sakai M, Kuru S, Kawamura T. A new lease on life for patients with Duchenne muscular dystrophy in Japan. Am J Med 2004; 117: 363.23 Eagle M, Bourke J, Bullock.
17. Lim LE, Rando TA. Technology insight: therapy for Duchenne muscular dystrophy—an opportunity for personalized medicine Nat Clin Pract Neurol 2008; 4: 149-58. <http://dx.doi.org/10.1038/ncpneu0737> PMID:18268530
18. Van Ommen GJ, van Deutekom J, Aartsma-Rus A. The therapeutic potential of antisense-mediated exon skipping. Curr Opin Mol Ther 2008; 10: 140-49. mPMid:18386226
19. Muntoni F. Is a muscle biopsy in Duchenne dystrophy really necessary? Neurology 2001; 57: 574-75. <http://dx.doi.org/10.1212/WNL.57.4.574> PMID:11524463

20. Lacomis D. The use of percutaneous needle muscle biopsy in the diagnosis of myopathy. *Curr Rheumatol Rep* 2000; 2: 225–29. <http://dx.doi.org/10.1007/s11926-000-0083-x> PMID:11123063
21. Lacomis D. The utility of muscle biopsy. *Curr Neurol Neurosci Rep* 2004; 4: 81–86. <http://dx.doi.org/10.1007/s11910-004-0017-5> PMID:14683634
22. Henriksson KG. Semi-open muscle biopsy technique. A simple outpatient procedure. *Acta Neurol Scand* 1979; 59: 317–23. <http://dx.doi.org/10.1111/j.1600-0404.1979.tb02942.x> PMID:484204
23. Dorph C, Nennesmo I, Lundberg IE. Percutaneous conchotome muscle biopsy. A useful diagnostic and assessment tool. *J Rheumatol* 2001; 28: 1591–99.
24. Sushruta, Sushruta Samhita, with Dalhanacharya. In: Acharya YT, ed. Nibandha Sangraha, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2009. p. 113-114.
25. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 321-22
26. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2009. p. 617
27. Sharma A. Madhavanidanam with Madhav vimarshini Commentary. 1st edition, Varanasi: Chaukhambha Sanskrit Pratishthan; 2007. p. 198-99.
28. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2009. p. 368
29. Shastri K, Chaturvedi G. Charaka Samhita with Vidyotini Commentary. edition, Varanasi: Chaukhambha Bharati Academy; 2011 reprint. p. 793.
30. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2009. p. 620
31. Vagbhata, Astanga Hridaya, with Arundatta. In: Kunte AM, ed. Sarvangasundari, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 225
32. Vagbhata, Astanga Hridaya, with Arundatta. In: Kunte AM, ed. Sarvangasundari, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 28
33. Vagbhata, Astanga Hridaya, with Arundatta. In: Kunte AM, ed. Sarvangasundari, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 223
34. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 25
35. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 88
36. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 651
37. Mukherji P K et al. Acorus calamus: Scientific Validation of Ayurvedic Tradition from Natural Resources Pharmaceutical Biology, 2007;45(8): 651-666
38. Vagbhata, Astanga Hridaya, with Arundatta. In: Kunte AM, ed. Sarvangasundari, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 267
39. Jain Mukesh D, Yoga Annapurna and Pandey MP 2002 Preliminary Study of Integrated Approach of Panch karma, Yoga and Ayurvedic Medicine in the Management of Muscular Dystrophy : 46 Patients. *World Health Review*, 1:1 33-35
40. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 731-32
41. Vagbhata, Astanga Hridaya, with Arundatta. In: Kunte AM, ed. Sarvangasundari, Commentary. Reprint ed. Varanasi: Chaukhambha Orientalia; 2011. p. 594
42. Govind Das, Bhaisiya ratnavali, In: Ambika datta shastri, ed. vidyotini commentary.reprint ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2001.p. 301
43. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 621
44. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 26
45. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 678
46. Govind Das, Bhaisiya ratnavali, In: Ambika datta shastri, ed. vidyotini commentary.reprint ed. Varanasi: Chaukhambha Sanskrit Sansthan; 2001.p. 396-97
47. Chakara pani Datta, Chakra Datta, In: Jagdishvara prasad Tripathi, ed. Bhavarthsadipani fifth ed. Varanasi: Chaukhambha Sanskrit series; 1983.p. 199
48. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 90-91
49. Agnivesa, Charaka Samhita, with Chakrapaanidatta. In: Acharya YT, ed. Ayurved Dipika, Commentary. Reprint ed. New Delhi: Chaukhambha Surbharati Parkashan; 2008. p. 89
50. Martini FH. Fundamentals of Anatomy and Physiology chapter 5. 4th ed. New Jersey: Prentice Hall Inc. Simon & Schuster; 1998. p. 148-155.
51. Martini FH. Fundamentals of Anatomy and Physiology chapter 5. 4th ed. New Jersey: Prentice Hall Inc. Simon & Schuster; 1998. p. 162.

Cite this article as:

Chaturvedi Ashutosh, Rao Prasanna N, U Shailaja, M Ashvini Kumar. Role of panchakarma in Duchenne muscular dystrophy. *Int. J. Res. Ayurveda Pharm.* 2013; 4(2):272-275

Source of support: Nil, Conflict of interest: None Declared