



Review Article

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RECENT ADVANCES IN AYURVEDIC MANAGEMENT OF CEREBRAL PALSY AFFECTED CHILDREN

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ABSTRACT

Cerebral palsy (CP) is a common chronic motor disability among developmental disorder prevalent world widely among pediatric population. It is a group of condition characterized by motor dysfunction due to non progressive brain damage in its early developing period; often accompanied by other features like seizures, behavioral disorder, cognitive impairment and other secondary musculoskeletal problems restricting them from normal independency. The condition poses considerable therapeutic challenge to the treating physician. Currently there is no specific treatment available in modern medical science to counteract the brain insult leading to motor dysfunction in CP. The available therapeutic interventions are mostly symptomatic and costlier. Recent advances in Ayurvedic clinical research have shown increased understanding of etiopathogenesis, its causal pathway of presentation and the value of specific intervention strategies in the management of Cerebral palsy. Ayurveda recommends multiple treatment options for intervention at various levels. Out of them, Panchkarma therapy and oral herbal formulations with properties of anti spasticity, anti convulsant, neurogenerative capacity etc. plays a vital role in the management. This paper provides a critical review on the effect of currently available treatment modalities of Ayurveda that can efficiently manage Cerebral palsy.

Keywords: Ayurveda Management, Cerebral Palsy, Panchkarma Procedures, Oral Herbal Drugs, Neuroplasticity.

INTRODUCTION

Cerebral palsy (CP) is a static neurologic condition resulting from brain injury that occurs before cerebral development is complete. Because brain development continues during the first two years of life, cerebral palsy can result from brain injury occurring during the prenatal, perinatal, or postnatal periods.¹ Even though the primary lesion, anomaly or injury is static, the clinical pattern of presentation may change with time due to growth and developmental plasticity and maturation of the central nervous system.² CP is a common problem, the worldwide incidence being 2 to 2.5 per 1000 live births.³ The etiological aspect of CP is very diverse and multifactorial. Due to recent advances in neonatal and obstetric care, the cases of CP are increasing with increasing survival of the very preterm and very low birth weight infants. Prenatal factors account for 75 % - 80 % of the cases while birth trauma or asphyxia constitute for less than 10 % of cases.⁴

Magnitude of problem and recent challenges

Spasticity is the most common clinical feature found in 70 – 80 % cases of CP.⁵ Affected limbs demonstrate increased deep tendon reflexes, tremors, muscular hypertonicity, weakness, and a characteristic scissors gait with toe-walking. Chronic spasticity can lead to muscular stiffness, contractures, atrophy and fibrosis; thereby making prone to orthopedic problems like scoliosis, equines deformity, subluxation and dislocation of joints etc. Mental retardation (MR) is common in CP in up to 60 % of the cases.² Visual impairments and disorders of ocular motility are common (28 %) in children with CP. There is an increased presence of strabismus, amblyopia, nystagmus, optic atrophy, and refractive errors.² Hearing impairment occurs in approximately 12 % of children with CP. This occurs more commonly if the etiology of

CP is related to very low birth weight, kernicterus, neonatal meningitis, or severe hypoxic ischemic insults.⁵ Epilepsy is common in children with CP and 35 % to 62 % of children develop epilepsy.² In an Indian study; it was found that 35 % had epilepsy. 66 % of children with spastic hemiplegia, 43 % of spastic quadriplegia and 16 % of children with spastic diplegia had seizures as an associated feature.⁶

Need of Alternative treatment

CP poses considerable diagnostic and therapeutic challenge to the physician. The primary injury to the central nervous system produces positive features such as spasticity, hyper-reflexia and co-contraction of muscles while negative features includes weakness, loss of selective motor control, sensory deficits and poor balance. The conventional medicine is usually focused on the positive features because it is possible to treat features like spasticity but it is the negative features which determines when or if a child will walk.⁷ Reduction of spasticity is the most important facet of overall management of CP. Oral medications used to decrease spasticity has untoward side effects such as drowsiness, sedation, fatigue and cognitive decline etc. As the CP is associated with other problems, more other drugs are added to prescription to combat for problems like epilepsy, mental retardation, etc making more complexity of treatment. The available treatment options are highly expensive and out of reach from low Income groups. Hence parents and families are always in a search of better, economical and more effective alternative therapeutic options. In a survey complementary and alternative medical treatment was found to be of great interest to families of affected CP children with a prevalence of 56 %; massage therapy was most accepted.⁸ Currently there is no specific treatment in conventional

system for these brain insults leading to motor dysfunction in CP. However the effects of brain damage may become more pronounced as the child grows. It has been shown that without intervention, detrimental changes will occur in gait and function over time span as short as 1.5 years.⁹ There is no specific disorder similar to CP mentioned in Ayurvedic texts. According to Ayurvedic etiopathogenesis, CP is viewed as a disease with vitiation of all three Dosha with predominance of Vata or more precisely Shiromarma Abhigatajanya Vata Vikara manifesting clinically all over the body (Vatadhika Sannipata Sarvanga roga) with the site of lesion in brain (Mastishka). It manifest with clinical presentation like Monoplegia (Ekanga Vata), Hemiplegia (Pakshaghata), Diplegia (Pangu), Quadriplegia (Sarvanga Vata) etc. The feature depends upon the site of lesion in Mastishka. Snehana, Swedana and Basti form the classical triad of management of Vatika disorder. Basti due to its controlling and regulating mechanism over nervous system is considered to be preemie in the management of Vatika disorders. Procedures like Abhyanga and Shastik Sali Pinda Sweda has a potential to reduce spasticity of muscles and providing nutrition to muscular tissue thereby preventing from atrophy and detrimental changes in muscles. The patients are usually associated with seizures (that are often resistant to antiepileptic drugs), Mental retardation and behavioral problems like hyperactivity, irritability etc. Shiordhara definitely help in regulating the neurochemical changes at the level of brain. Similarly Oral herbal compound containing ingredients with Medhya, Balya, Brimhana and Vatahara properties would bring out normalcy of Vata including other associated doshas, providing better nourishment to dhatus, and importantly changes at higher level of brain with concept of regeneration of neurons and improving cognition deficit in CP affected children. Thus drugs having anti spasticity, anti convulsant, neuroprotective, neuro – regenerative and cognition enhancer capacity should be employed in the treatment module of CP. Due to multiplicity of problems associated with the CP, the different routes of Ayurvedic treatment should be specified according to involvement of areas and dominance of symptoms.

Principle of Treatment

- To improve quality of life in CP affected children.
- To facilitate for early rehabilitation.
- To enhance or improve the functional capacity of the child in order to make him/ her selfdependent.
- To decrease the complications of CP

Areas for Intervention

- To relieve muscle spasticity
- Control of seizures, since most of them are resistant to conventional antiepileptic medications
- Prevention of orthopedic problems like hip subluxation, scoliosis, equines deformity etc.
- To improve cognition, learning and memory for better acquisition of skilled movements

Oral Herbal Drugs

CP is a sequel of brain injury and a growing body of evidence demonstrates that the brain is capable of recovery after an injury because of the ability of neurons and other brain cells to alter their structure and function (plasticity) in response to external and internal pressures.¹⁰ Neuroplasticity refers to structural and functional changes in the brain that are brought about by training and experience. The brain is organ that is designed to change in response to experience. According to Canadian Neurologist Donal Hebb, when cells are active together synapses are strengthened and preserved. This strengthening and preservation of neurons are very much activity dependent. The neuron and synapses that are activated repeatedly are preserved while those that are not activated are pruned. Hence early experience has great impact on brain development. Plasticity of brain is maximal in first few years of life but continues at reduced rate throughout the life. Hence early intervention in early period of life can have greater impact on brain.¹¹ Thus drugs with neuro-regenerative, neuro-protective and nootropic properties can make a micro environment in the brain for brain plasticity. Review of various clinical and experimental studies of Ayurvedic herbs with aforementioned properties are discussed as below.

Anti Spasticity / Skeletal muscle relaxant

Vidarikanda

In a study *Pueraria tuberosa* DC. (Vidarikanda) isoflavanoids and their active metabolites daidzin, daidzein and genistein were studied for muscle relaxant activity in mice. Methocarbamol and dantrolene sodium; which were used as positive controls. A high dose (100 mg/kg, i.p.) of equal, the reductive metabolite of had significant muscle relaxant activity at 15, 30 and 45 minutes after administration and its potency been moderate. Potent muscle relaxant activity was observed in vivo with p-ethylphenol (100 mg/kg, i.p.), the degraded metabolite of genistein.¹²

Bansh

The leaf extract of *Phyllostachys bambusoides* was investigated for skeletal muscle properties by testing the effects of the extract on wistar rat using rota-rod apparatus model, inclined screen test, climbing test. The extracts contain glycosides, carbohydrates, tannins, proteins and flavonoids. Diazepam in a dose of 4 mg/kg (s.c.) was used as a standard. Chloroform extract at the dose level of 200 mg/kg body weight showed significant skeletal muscle relaxant activity¹³

Tvak

In another study, the aqueous extract of *Cinnamomum zeylanicum* bark was studied for skeletal muscle relaxation property orally at a dose of 50, 100, and 200 mg / kg. It was divided into five groups consisting of six animals each. Group I served as the control, which received normal saline 10 ml / kg, Group II received the standard drug Diazepam, at a dose of 10 mg / kg, p.o., Group III, IV, and V received the aqueous extract of *Cinnamomum zeylanicum* bark orally at a dose of 50, 100, and 200 mg / kg. Three different doses of aqueous extract

of *Cinnamomum zeylanicum* bark (50, 100, and 200 mg / kg p.o.) showed a dose-dependent increase in muscle relaxation, that is, 62.3, 77.7, and 79 %, respectively, when compared to the control. Maximum muscle relaxation was observed with 200 mg / kg of *Cinnamomum zeylanicum*.¹⁴

Karvira

Similarly to above study, the aqueous extract of *Nerium oleander* leaves was studied for skeletal muscle relaxation property orally at a dose of 50, 100, and 200 mg / kg. Maximum muscle relaxation was observed with 200 mg / kg of aqueous extract of *Nerium oleander* leaves.¹⁵

Anti convulsant Activity

Shankhpushpi

It was observed that the animals treated with the methanolic extracts of stem callus, leaf callus and whole plant (200 mg/kg oral) of *Convolvulus pluricaulis* Choisi (Shankhpushpi) showed significant protection against tonic convulsion induced by transcorneal electroshock, which was also comparable with that of the standard drug phenytoin¹⁶

Jatamansi

Ethanol extract of the roots of *N. jatamansi* was studied for its anticonvulsant activity and neurotoxicity, alone and in combination with phenytoin in rats. Pretreatment of rats with phenytoin at a dose of 12.5, 25, 50 and 75 mg/kg in combination with 50 mg/kg of *N. jatamansi* root extract resulted in a significant increase in the protective index of phenytoin from 3.63 to 13.18. The dose response studies of phenytoin alone and in combination with *N. jatamansi* extract on the serum levels of phenytoin clearly demonstrated the synergistic action of both the drugs.¹⁷

Ashwagandha

Co-administration of a sub-effective dose of *W. somnifera* (50 mg/kg, po) with a sub-protective dose of either GABA (25 mg/kg, ip) or diazepam (0.5 mg/kg, ip) increased the seizure threshold. The results suggested that the anticonvulsant effect of *W. somnifera* against PTZ seizure threshold paradigm involved the GABA_Aergic modulation.¹⁸

Tagar

Valeriana wallichii possess anticonvulsant activity in maximal electroshock seizures in mice after intra peritoneal administration as indicated by reduction in duration of tonic hind limb extensor phase with a dose dependant increase in potency (450 mg/kg, 900 mg/kg).¹⁹

Yasthimadhu

Anticonvulsant effects of aqueous extract of *G. glabra* were investigated in mice. *G. glabra* extract, diazepam and normal saline were injected intraperitoneally at 50-300 mg kg⁻¹, 0.5-1 mg kg⁻¹ and 10 mL kg⁻¹, respectively, 30 min before pentylenetetrazole (90 mg kg⁻¹, i.p.). Aqueous extract at a dose of 300 mg kg⁻¹ delayed the onset time of the seizure and decreased the duration of seizure significantly compared to the control. The duration of seizure was also significantly decreased at

doses 60-200 mg kg⁻¹. In conclusion, the aqueous extract of glycyrrhiza root possesses anticonvulsant activities which may be effective in the management of petit mal seizure.²⁰

Neuroregenerative Drugs

Mandookparni

Axonal regeneration

It is important for functional recovery following nerve damage. Male Sprague-Dawley rats given *Centella asiatica* ethanolic extract in their drinking water (300-330 mg kg⁻¹ daily) demonstrated more rapid functional recovery and increased axonal regeneration (larger calibre axons and greater numbers of myelinated axons) compared with controls, indicating that the axons grew at a faster rate.²¹

Enhancement of Amygdaloid Neuronal Dendritic Arborization

The rat pups (7-days-old) were fed with 2, 4 and 6 ml/kg body of fresh leaf juice of *Centella asiatica* (CeA) for 2, 4 and 6 weeks. After the treatment period, the rats were killed, brains removed and amygdaloid neurons impregnated with Silver nitrate (Golgi staining). Amygdaloid neurons were traced using camera lucida and dendritic branching points (a measure of dendritic arborization) and intersections (a measure dendritic length) quantified. The data were compared with those of age-matched control rats. The results showed a significant increase in dendritic length (intersections) and dendritic branching points along the length of dendrites of the amygdaloid neurons of rats treated with 4 and 6 ml/kg body weight/day of CeA for longer periods of time (i.e. 4 and 6 weeks). The study concluded that constituents/active principles present in CeA fresh leaf juice has neuronal dendritic growth stimulating property; hence it can be used for enhancing neuronal dendrites in stress and other neurodegenerative and memory disorders.²²

Ashwagandha

Induction of Axon or Dendritic outgrowth

Extension of dendrites and axons in neurons may compensate for and repair damaged neuronal circuits in the dementia brain. The methanol extract of Ashwagandha (roots of *Withania somnifera*; 5 µg/ml) significantly increased the percentage of cells with neurites in human neuroblastoma SK-N-SH cells. The effect of the extract was dose and time-dependent mRNA levels of the dendritic markers MAP2 and PSD-95 by RT-PCR were found to be markedly increased by treatment with the extract, whereas those of the axonal marker were not. The results suggest that the methanol extract of Ashwagandha promotes the formation of dendrites.²³

Neuritic regeneration and synaptic reconstructions

Treatment with Withanolide A (WL-A) 1 micro M induced significant regeneration of axons and dendrites, in addition to the reconstruction of pre and post synapses in the neurons. WL-A (10 micro mol kg⁻¹ day⁻¹), for 13 days) recovered α beta (25-35) induced memory deficit in mice. At that time, the decline of axons,

dendrites and synapses in the cerebral cortex and hippocampus was almost recovered.²⁴

Neuroprotective

Ashwagandha

The neuroprotective effects of *W. somnifera* were studied on stressed adult female Swiss albino rats. Experimental rats were subjected to immobilization stress for 14 h and were treated with a root powder extract of *W. somnifera* available as Stresscom capsules. Control rats were maintained in completely, non stressed conditions. Thionin stained serial coronal sections (7 μ m) of brain passing through the hippocampal region of stressed rats (E₁ group) demonstrated 85 % degenerating cells (dark cells and pyknotic cells) in the CA₂ and CA₃ sub-areas. Treatment with *W. somnifera* root powder extract significantly reduced (80 %) the number of degenerating cells in both the areas. The study thus demonstrates the anti stress neuroprotective effects of *W. somnifera*.²⁵

Jatamansi

Study was conducted in middle cerebral artery occlusion model of acute cerebral ischemia in rats. All the alternations induced by ischemia were significantly attenuated by 15 days pretreatment of *N. jatamansi* (250 mg/kg, orally) and correlated well with histopathology by decreasing neuronal cell death following occlusion and reperfusion.²⁶

Vacha

Administration of *Acorus calamus* at its ED₅₀ dose of (185 mg/kg) significantly potentiated the anticonvulsant action of phenytoin by reducing its ED₅₀ value from 13.5 mg/kg to 9.25 mg/kg; further, it potentiated the anticonvulsant action of phenobarbital by reducing its ED₅₀ value from (8 mg/kg to 5 mg/kg). This study provides evidence for significance of neuroprotective herb *Acorus calamus* as an adjuvant in antiepileptic therapy.²⁷

Nootropic activity (Improvement in cognition, learning and memory)

Brahmi

Double-blind randomized placebo control study was done in 76 adults aged 40-65 years to examine various memory functions and level of anxiety. 3 testing sessions were decided at prior to trial, after 3 months of trial and 6 wks after completion of trial. The results show a significant effect of Brahmi on a test for retention of new information, in follow-up test the rate of learning was unaffected. Task assessing attention, verbal and visual short term memory and retrieval of pre-experimental knowledge were affected. Questionnaire measures of everyday memory function and anxiety levels were also unaffected.²⁸

Protection from Phenytoin-induced cognitive deficit

Bacopa monniera (BM) was evaluated alone and in combination with phenytoin for its effect on (a) passive-avoidance (PA) task; (b) maximal electroshock seizures; and (c) locomotor activity in mice. Phenytoin (PHT, 25 mg/kg po \times 14 days) adversely affected cognitive function in the PA task. BM extract (40 mg/kg \times 7 days),

given along with phenytoin in the second week of the two-week regimen, significantly reversed PHT-induced impairment. Both acquisition and retention of memory showed improvement without affecting its anticonvulsant activity. The results provide evidence for potential corrective effect of *Bacopa monniera* in cognitive deficit associated with PHT therapy.²⁹

Mandookparni

Fresh plant extract was given orally to rat pups with a dose of 2 ml/ka/day for 6 wks, subjected to learning tests in T-maze and passive avoidance test. The result indicates a correlation between improved learning capacity and increased dendritic arborization in amygdaloid nucleus. This may be the neural basis for enhanced learning in *C. asiatica* treated rats.³⁰

Jatamansi

Three doses (50, 100, and 200 mg/kg, p.o.) of an ethanolic extract of *N. jatamansi* were administered for 8 successive days to both young and aged mice. The 200 mg/kg dose of *N. jatamansi* ethanolic extract significantly improved learning and memory in young mice and also reversed the amnesia induced by diazepam (1 mg/kg, i.p.) and scopolamine (0.4 mg/kg, i.p.) It also reversed aging-induced amnesia due to natural aging of mice. As scopolamine-induced amnesia was reversed, it is possible that the memory improvement may be because of facilitation of cholinergic transmission in the brain.³¹

Panchakarma Procedures

Various Panchakarma procedures are shown to be beneficial in motor dysfunction of CP. Main procedures are Abhyanga (massage), Shashtika shali pinda sweda (sudation with specific rice), Shirodhara, and Matra basti.

Abhyanga and Shastik shali pinda sweda

The procedure which causes unctuousness, fluidity, softness and moistness in the body is Snehana or Oleation therapy. The main part of Abhyanga procedure is the mechanical stimulation more precisely the pressure application during massage therapy. Pressure application done in proper way can help in reduction of motor neurone hyperexcitability by reducing the alpha motor neuron activity. The way of mechanism of action is not clearly understood and the amount of pressure to be given to stimulate deep tendon receptors or superficial mechanoreceptors is still not properly understood. A study reported that in hemiparetic subjects the H-reflex was depressed during both continuous and intermittent tendon pressure. Intermittent pressure was more effective than continuous.^{33,34} In a study, cerebral palsy symptoms in children were decreased following massage therapy but the mechanism behind it was not explained in the study.³⁵ Shastika shali pinda sweda (SSPS) is the sudation performed by bolus of boiled Shashtika shali (*Oryza sativa* Linn.) with Balamoola kwatha (decoction of *Sida cordifolia* Linn.) and milk. The heat provided by bolus of Shastik sali dipped in Balamula kwatha with Godugdha may increase the blood flow locally, relieve muscle spasm, increase tendon extensibility and provide pain relief. Thus Abhyanga and Shastik Sali Pinda Sweda

cumulatively help in reduction of spasticity and facilitate free movement of joint preventing from deformities and contractures.

Matra Basti

It is a sub type of Anuvasana basti in which oil or ghee is given by rectal route in a small quantity. When medicated oil reaches rectum and colon, presence of short chain fatty acids in oil allows direct diffusion of drugs from epithelial cells into capillary blood villi and showing its generalized effect. Gastrointestinal tract is richly supplied by network of nerve fibers and works in synergism with central nervous system. Hence Basti pacifies the vitiated Vata at root level of its origin thereby normalizing and influencing its sub- doshic level at other distant sites too. Thus the effect of Basti at gastrointestinal system will definitely affect other system thereby achieving a level of homeostasis. Thus it helps to control and regulate symptoms of CP. In a clinical study, effect of Samvardhana ghrita by oral and rectal route were assessed. 40 children suffering from CP were included and randomly distributed in two groups with 20 patients each. Group A (Samvardhana ghrita orally) was treated with 5 g. of Samvardhana ghrita twice daily with honey as anupana for 48 days. In group B (Samvardhana ghrita as matra basti) 20 ml of Samvardhana ghrita was administered through Basti for 48 days. The study reports promising results. Oral route was found to be more effective in language and performance while Basti group had shown better improvement in fine and gross motor development.³⁶

Shirodhara

The chemical constituent of Shirodhara may modulate the secretions of various neurotransmitter and hormone at brain cellular level. Thereby controlling seizures, cognitive impairment and behavioral problems like anxiety, attention – deficit hyperactivity disorder etc. associated with CP.³⁷ In a randomized controlled clinical trial, the multiple Ayurvedic interventions in form of oral herbal formulation (Syp. Ayurvedic Compound) and Panchkarma procedures (Abhyanga, SSPS, shirodhara and Matra Basti) along with Physiotherapy was studied on CP affected children of age group 1- 12 years for six month duration. Group A (n = 14) received Physiotherapy (control group); group B was treated with Panchakarma procedures and Physiotherapy while group C received Syp. Ayurvedic Compound, Panchkarma procedures and Physiotherapy. The study observed the maximum efficacy of treatment modality in Group B and C on standard assessment criteria – Gross motor function classification system, Modified Ashworth spasticity scale and Modified Barthel Score index- Activities of Daily Living (ADL). The study concluded that the effect of physiotherapy treatment was potentiated by an oral herbal formulation (Syp. Ayurvedic Compound) and Panchkarma procedures for better reduction in spasticity, joint deformities and prevention of contractures and thereby facilitating for early achievement in areas of ADL by improving muscle tone, gain in muscle strength and intelligence for better skilled performance.^{37,38}

CONCLUSION

Cerebral palsy is a common chronic motor disability prevalent among pediatric population. Recent studies show that Ayurveda through its principals can effectively manage different types of CP along with its associated problems. Recent advances concludes that Ayurvedic treatment modality through its principals can induce structural reorganization or repair of damaged neurons and stimulation for forming new synapses for better functional recovery in Cerebral palsy affected children. The maximum benefit can be achieved if Ayurvedic treatment modality is incorporated with physiotherapy in early intervening period

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