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An Overview on Pharmacological Action of *Sariva* (*Hemidesmus indicus* R.Br.) w.s.r. to Cerebral Palsy

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Abstract

It is always said that childhood period is like a mirror, which reflects in after life the images first presented to it. When debility is considered particularly in children, near about quarter of chronic childhood disorders are neurological in origin. Cerebral Palsy is defined as a non-progressive disturbances in the developing fetal or infant brain which is accompanied by disturbances of sensation, perception, cognition, communication, behaviour as well as by epilepsy and secondary musculoskeletal problems. In modern medicine, there is only symptomatic treatment for neuro-motor disability but actually there is no effective treatment has been formulated at present which can totally cure this disorder. Hence, there is a need for the development of a well-planned ayurvedic approach in present era. So, we are proposing *Sariva* (*Hemidesmus indicus* R.Br.) as best choice in cerebral palsy. *Sariva* possesses *madhur* and *Tikta rasa*, *Madhura vipaka*, *Sheeta veerya* and *Guru*, *Snigdha guna*. *Acharya Sushruta* in *Garbhinivyakaranasharir adhyaya* has mentioned that on first day of *Nalchhedana*, the child should be sprinkled with cold water and then *Madhu* and *Ghrita* mixed with *Ananata* (*Sariva*) *choorna* should be given in small quantity three times in a day. It helps in promoting general and healthy growth, strength and intellect of children. It is also used in treating the conditions like *Pangu*, *Mooka*, *Ashruti* and *Jada*. In this article we have try to discuss about the pharmacokinetic of *Sariva* w. s. r. to cerebral palsy in children.

Keywords: *Sariva*, Cerebral palsy, Neuro-motor disorder

Introduction

Childhood is the happiest period of life and it creates beautiful memories that can't be replaced. But some diseases occur at childhood period and last throughout the life. Some of them are life threatening. So it gives grief to all family members. According to *Ayurveda*, the root of these

diseases are from *vikrit shukra* and *shonita* of father and mother respectively. One of these diseases is Cerebral Palsy which occurs at gestational period and persists in perinatal and after birth of child. It is the most ordinary form of childhood disability.

According to W.H.O. (2011), the worldwide incidence of Cerebral Palsy is approximately 2-
2.5 cases per 1000 live births. In India near about 3 cases per 1000 lives were found. [1] With reference to the prevalence of CP, more than 15 million population worldwide and more than 25 lakh people in India are suffering. [2]

The word "Cerebral" means controlling the brain. The word "Palsy" means difficulty or troubles with body movements. Cerebral Palsy is an umbrella term used for neuromotor symptoms. It is defined as a non- progressive disturbances in the developing fetal or infant brain which accompanied by disturbances of sensation, perception, cognition, communication, behaviour, as well as epilepsy and by secondary musculoskeletal problems. [3] [Nelson]. It is a range of non-progressive syndrome of posture and motor impairment, causing activity limitation. Bax et al concluded that the cerebral palsy often accompanied by other neurodevelopmental disorders such as specific cognitive or visual deficits. [4] CP is classified into topography-based subtypes into quadriplegia, diplegia, hemiplegia or extrapyramidal disorders which often result from various abuses to the different areas within the developing nervous system, occurring in utero, during delivery or after birth during the first 2 years of life.

[5] As said earlier that CP occurs at the gestational period, so the vulnerability of different brain
structures and types of disability related with CP are strongly influenced by the gestational age at which brain development is altered. There is urgently need to lead the brain protection strategies and prevention of CP. In modern medicine, there is only symptomatic treatment for such diseases and the permanent cure is not possible. Ayurvedic remedy is helpful for such condition from very long decades. Various *Acharyas* have told that *shuddha artava* and *shuddha shukra*, *pathya sevana* by women after conception and *jatkarma samskara* support the healthy child development. After all these precautions, if disease happens we have to move toward herbal medication. The drug *Sariva* is useful in cerebral palsy and it is more potent, price effective and easily available in market.

In Ayurveda, according to *Acharya sushruta, vyadhi* has been categorised into *Aadibala pravrutta, Janmabala pravrutta* and *Doshabala pravrutta*. [6] *Aadibala pravrutta vyadhi* are due to *dushti* in *beej* and *beejabhaga avayava* arising from *shukra* and *shonita dushti*. *Janmabala pravrutta vyadhi* are *rasakruta a*nd *dauhrida apacharakrita*. Due *to Apathya ahara* and *Vihara sevana* and *Apachara* by the pregnant woman leads to the *Andhatva, Badhirya, Pangu, Mooka, Minmina* and *Vaamana* in the child. *Doshabala pravrutta vyadhi* are *Sharira* and *Manasa vyadhi* which leads to the physical as well as mental impairment in development of child. All these *vyadhi* i.e. *Aadibala, Janmabala* and *Doshabala vyadhi* are commonly take part in pathogenesis of cerebral palsy.

According to Marret et al, in modern

science, the pathophysiology of cerebral palsy be like this. In first part of pregnancy (until 24 weeks of gestation) cortical neurogenesis takes place and is characterised by proliferation, migration and organization of neuronal precursor cells takes place and then formation of neurons. These can be altered by genetic deficit or acquired impairment. In second part of pregnancy, growth and differentiation events (axonal and dendrite growth, synapse formation, and myelination) as well as stabilization processes (neural cell apoptosis, neurite regression, redundant synapse elimination) and specialization of circuitry are predominant and persist after birth and are maximal during the first 2 years of life. Environmental factors like hypoxia-ischemia are involved in the occurrence of Cerebral Palsy at this step of brain development. [7]

By nelson, CP is usually classified into topography-based subtypes (quadriplegia, diplegia, hemiplegia, or extrapyramidal disorders) which often result from various insults to different areas within the developing nervous system, occurring in utero, during delivery, or after birth during the first 2 years of life. [8] It was previously considered that, the interruption of the oxygen supply to foetus or birth asphyxia were classically the main causal factors explaining later cerebral palsy. But in human epidemiological studies and in animal experiments, it is to be found that clinically defined birth asphyxia and birth injury account for minority cases of cerebral palsy. Several non-ischemic factors have now been recognised. In CP, causal factors do not act separately, but they are in co-operation to create the disturbance in the central nervous system. A set of predisposing factors, acute or sub-acute antenatal factors and postnatal aggravating factors act together in the brain development in the foetus and new born to alter the brain maturation and create the cerebral palsy. Pre-disposing factors include genetic factors, infections, toxic factors, multiple gestation, vascular disease of pregnancy, preterm birth, post-term birth, maternal factors, etc. [9,10,11] Acute antenatal factors include an umbilical arterial PH

$\leq$ 7, a moderate or severe neonatal encephalopathy and a later quadriparetic or diskinetic CP.

[12] Postnatal factors include stress and separation of mother/baby, nutrition and extra uterine growth retardation, nosocomial infections, Enterocolitis, or drugs. [13]

From the above pathophysiology of cerebral palsy, it is clearly noticed that CP is caused by numerous factors; but it results from a common pathological mechanism i.e. excitotoxicity. The key factors triggering the excitotoxicity in cerebral palsy are excessive production of pro- inflammatory cytokines, oxidative stress, and deficiency of growth factors, extracellular matrix modification and excessive release of Glutamate.

Role of Glutamate-

Glutamate is the primary neurotransmitter of central nervous system, which is released in synaptic cleft. It is then bound with Glutamate receptors and due to removal of Glutamate by Glutamate receptors, the propagation of action potential takes place. This Glutamatergic effect is mediated by N-methyl D aspartate, of which the density peaks in early neonatal period. Due to this calcium mediated excitotoxicity occurs. Moreover, the excitotoxicity is also due to failure of

energy-dependent reuptake of Glutamate. So, the glutamate receptors fail to bind with Glutamate which results into increase in Glutamate at synaptic terminal. It leads to fail in propagation of action potential and finally results to brain ischemia or brain damage. [14] [15]

Role of Acetyl-choline esterase-

We well know that, cholinergic system plays an important role in memory and learning function of central nervous system. The neurotransmitter Acetyl choline helps in the propagation of impulse from post synaptic membrane to pre synaptic nerve. But due to calcium influx, there is increase in activity of Acetyl choline esterase. Due to this, the catabolism of acetyl choline increases; so there is reduction in acetyl choline synthesis which ultimately leads to reduction in memory and judgement function of cerebrum. [16] [17]

Role of Free radicals-

Increase in calcium influx promotes the production of free radicals, which leads to cell membrane damage. Due to this increase in free radicals, dysfunction of mitochondria occur, which mediates the activation of genes involved in apoptosis. [18]

Review of literature-

Acharya Sushruta has mentioned that [19] At first day of child birth, *Anantamoola choorna* should be given with *Madhu* and *Ghrita* in 3 times a day.

SARIVA

Latin name – *Hemidesmus indicus* R. Br.

Family – Asclepiadaceae

Synonyms – *Gopawalli, Gopakanya, Gopavadhu, Gopi, Ananta, Utpalasariva, Sharadi, Chandansariva, Bhadravallika* [20]

***RASAPANCHAKA* –**

***Rasa* –** *Madhura, Tikta* ***Vipaka* –** *Madhura* ***Veerya* –** *Sheeta*

***Guna* –** *Guru, Snigdha* [21]

***Sariva* (*Hemidesmus indicus* R.Br.)**

Chemical Constitutions-

Coumarino - lignoids hemidesminine - hemidesmine 1 and hemidesmine 2, beta-sitosterol, alpha-amyrin, beta-amyrin, beta-amyrin acetate, lupeol, lupeol acetate, lupeol octacosanoate, 2-hydroxyl-4-methoxy-benzaldehyde**,** drevogenin-beta-3-O-beta-D-oleandropyranosyl, (1-4)-beta-D-oleandropyranoside (desinine). [22]

Discussion-

From the pathophysiology of cerebral palsy, it is clearly noticed that even if CP is caused by several factors; it results from a common pathological mechanism. The key factors triggering the excitotoxicity in cerebral palsy are excessive release of

Glutamate, increase in activity of Acetylcholine esterase and oxidative stress.

The drug *Sariva* has *Tikta* and *Madhura rasa*. The *panchabhautika* constitution of *rasa* is like-

Panchabhautika constitution	*Tikta rasa*	*Madhura rasa*
Prithvi	-	+
Aap	-	+
Tej	-	-
Vayu	+	-
Akash	+	-

From the table, it is cleared that *Tikta rasa* has predominance of *Vayu* and *Akash Mahabhuta* (Hydrophobic), while *Madhura rasa* has predominance of *Prithvi* and *Aap mahabhuta* (Hydrophilic).

The Glutamate is the non-essential amino acid which is hydrophilic in nature. The *Tikta rasa* with predominance of *Vayu* and *Akash mahabhuta* counteract the effect of Glutamate. So there is decrease in amount of glutamate at the synapse and ultimately helps in propagation of action potential. Acetylcholine esterase is hydrophobic in nature. So, *Madhura rasa* with predominance of *Prithvi* and *Aap mahabhuta* counteract the effect of Acetylcholine esterase. Due to this, there is normal synthesis of Acetylcholine which ultimately helps in transmission of impulses from one synapse to other. Thus, maintain normal memory and learning function of the brain.

D. Sivaraman and P. Muralidharan have experimentally proved that the Methanolic extract of *Hemidesmus indicus* root bark showed the antagonized property against the release of excess Glutamate and thus reduces the level of Glutamate. It also increases the level of Dopamine which is already lowered in neurodegenerative condition. It helps in decrease of Acetylcholine esterase and release of Acetylcholine at the nerve terminals, which restore the normal memory function. [23]

M. N. Ravishankara et al, in his in vitro study, it has been proved that the presence of tannins and phenolic compounds along with flavonoids like Hemidesmin 1 and Hemidesmin 2, 2- hydroxy-4-methoxy benzoic acid of methanolic extract of Hemidesmus indicus root bark shows antioxidant activity by scavenging DPPH radical, harmful superoxide radical and a moderate nitric oxide scavenging activity. [24]

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