



ISSN 2456-3110

Vol 4 · Issue 6

Nov-Dec 2019

Journal of
**Ayurveda and Integrated
Medical Sciences**

www.jaims.in

JAIMS

An International Journal for Researches in Ayurveda and Allied Sciences



Charaka
Publications

Indexed

Cerebellar Ataxia and its management - An Ayurvedic Approach

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ABSTRACT

The cerebellar ataxia is a heterogeneous group of disorders clinically characterized by the presence of cerebellar dysfunction. These results from the involvement of cerebellum and its afferent and efferent path ways including spinocerebellar pathway, and the frontopontocerebellar pathways. Etiology of cerebellar ataxia can be classified based on the onset, progression, location of lesions, based on distribution, hereditary. In Ayurveda by seeing etiology and symptoms can be correlated to shiromarmopaghata. Due to indulgence in vataja ahara and vihar, abhighata does the vata and raktapradushana leading to shiromarmopaghata. In cerebellar ataxia shiromarmopagata Chikitsa can be adopted. Since we don't get direct reference about this disease based on hetu vishesha and sthana vishesha treatment can be adopted.

Key words: Cerebellar ataxia, Shiromarmopagata, Vatavyadhi.

INTRODUCTION

The cerebellum (Latin word means "little brain") is the largest part of hindbrain. It is infratentorial structure that coordinates voluntary movements of the body. ATAXIA- "lack of order" (Greek word)"A"-(negative article); "taxia" - (order). It is defined as impaired coordination of voluntary muscle movement. The cerebellar ataxia are a heterogeneous group of disorders clinically characterized by the presence of cerebellar dysfunction. These results from the involvement of cerebellum and its afferent and efferent path ways including spinocerebellar pathway,

and the frontopontocerebellar pathways.^[1] Symptoms and signs of ataxia consist of gait impairment, unclear speech, and visual blurring due to nystagmus, hand incoordination and tremor with movement.^[2]

Ataxia can have an insidious onset with a chronic and slowly progressive clinical course (e.g., spinocerebellar ataxias [SCAs] of genetic origin) or have an acute onset, especially those ataxias resulting from cerebellar infarction, hemorrhage, or infection, which can have a rapid progression with catastrophic effects. Sub acute onset, as from infectious or immunologic disorders.^[3]

In Ayurveda we don't get much information about cerebellar Ataxia but by seeing its etiology, signs and symptoms we can correlate to shiromarmopagata.

Physiological or functional division of cerebellum

The cerebellum and its afferent and efferent connections, the vestibular system, and the proprioceptive sensory pathway are all involved in ataxia. Hence knowing the functional division of the cerebellum helps in localization of the lesion.

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Submission Date: 08/11/2019 Accepted Date: 25/12/2019

Access this article online

Quick Response Code



Website: www.jaims.in

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Functional division of the cerebellum

	Contents	Afferent Connection	Efferent Connection	Functions
Vestibulo cerebellum	Flocculonodular lobe	Vestibulocerebellar tract	Cerebellovestibular tract Fastigiobulbar tract	Regulation of tone, posture and equilibrium By receiving impulses from vestibular apparatus
Spinocerebellum	Lingula, Central Lobe, Culmen, Lobulus simplex, Declive, Tuber, Pyramid, Uvula, Paraflocculi & medial portions of cerebral Hemispheres.	Dorsal spinocerebellum tract, Ventral spinocerebellum tract, Cuneocerebellar tract, Olivocerebellar tract, Pontocerebellar Tract, tectocerebellar tract, trigeminocerebellar tract	Cerebelloreticular tract Cerebello-olivary tract Fastigiobulbar tract	Regulation of tone, posture and equilibrium By receiving impulses from proprioceptors in muscles, tendons and joints, tactile receptors, visual receptors and auditory receptors
Corticocerebellum	Lateral portions of cerebral hemispheres	Olivocerebellar tract, Pontocerebellar Tract,	Dentatothalamic tract Dentatorubral tract	Regulation of coordinated movements Damping action Control of ballistic

				movement :Timing and programming of movement :Servomechanism :Comparison or function
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Etiology of Cerebellar Ataxia

Based on onset etiology can be classified as acute, sub-acute and chronic

Onset	Causes
Acute (hours to days)	<ul style="list-style-type: none"> Intoxication: Alcohol, lithium, phenytoin, barbiturates. Infections: Acute Viral cerebellitis, cerebellar abscess Vascular: Infraction (AICA, PICA syndromes), hemorrhage, subdural hematoma.
Sub Acute (days to weeks)	<ul style="list-style-type: none"> Intoxication: Mercury, Solvents, Glue Nutritional: B1 & B12 deficiency Infection: HIV Demyelinating: Multiple Sclerosis Neoplastic: Glioma, Metastases
Chronic (months to years)	<ul style="list-style-type: none"> Autoimmune Causes: Paraneoplastic syndromes. Hypothyroidism Infections: Syphilis (tabes dorsalis) Congenital Lesions: Arnold-Chiari and Dandy Walker Syndromes. Inherited: SCA, Friedreich's Ataxia,

Based on progression

Progression	Causes
Progressive Ataxia	Spinocerebellar ataxia (SCA)
Static Ataxias	Vascular causes

Reversible Ataxia	<ul style="list-style-type: none"> ▪ Infectious causes ▪ Thyroid ▪ Drugs ▪ Toxins
Intermittent Symptoms	Episodic Ataxias (inherited etiology)

Based on location of lesion

Cerebellar Hemispheric Syndrome (unilateral)	Ipsilateral limb ataxia, tremor, hypotonia	infarct, neoplasm
Rostal Vermis Syndrome (anterior lobe and vermis)	Gait and postural Ataxia	Chronic Alcoholism
Caudal Vermis Syndrome (vestibulocerebellum)	Trunk Ataxia, dysarthria and nystagmus	Medulloblastomas
Pancerebellar Syndrome (bilateral hemispheres+vermis)	Truncal & bilateral limb ataxia, dysarthria, oculomotor disturbances.	Neurodegenerative diseases, acute alcoholic intoxicatio.
Cerebellar Peduncles	Dramatic cerebellar symptoms	

Based on Distribution

Focal Ataxia	Symmetrical Ataxia
Vascular causes	Intoxication
Multiple sclerosis	Nutritional
Cerebellar Abscess	Infectious
Cerebellar Giloma	Hypothyroidism
HIV	
Congenital causes	

Clinical Features

- Gait Ataxia (Truncal Ataxia of walking): unsteady walking with tendency to fall and compensatory wide-based stances. Gait deviates and falls to side of the lesion or may be so severe that patient cannot walk (ABASIA).
- Postural Ataxia (Truncal Ataxia of stance & sitting): Stance usually is on broad base, feet

several inches apart or patient may be unable to sit or stand without support (ASTASIA).

- Limbal Ataxia (Ataxia of extremities): its more marked in upper limbs than lower limbs, in complex movements than in simple movements, in fast movements than in slow movements and when change of direction is required.
- Dysmetria; disturbance of trajectory during active movement(due to inability to control distance, direction, speed power)
- Dysdiadokokinesia: inability to perform rapidly alternating movements(e.g. forearm pronation-supination)
- Hypotonia: decreased muscle tone
- Rebound phenomenon
- Dysarthria (Ataxia of bulbar muscles): slurred (articulatory impreciseness), slow speech, increased variability of pitch and loudness, sing-song quality, increased separation of syllables
- Ocular Ataxia (Ataxia of Extra ocular muscles): Gaze-evoked Nystagmus.
- Tremor: intention tremor. Static Tremor develops if patient attempts to maintain limb in fixed position.

Examination

Neurological examination of the patient helps in proper diagnosis of cerebellar ataxia. Lesion in cerebellum do not affect on Mental Status (Cognition, Memory, consciousness etc.), sensory status, autonomic functions and muscle strength.

Cranial Nerve Examination

Ipsilateral loss of corneal reflex and eighth cranial nerve dysfunction may suggest a cerebellopontine angle tumor. Facial and tongue fasciculations may be a prominent sign of SCA3, and severe tongue atrophy and fasciculations are signs of SCA36. Examination of extraocular movements.

Vestibular Signs

Ataxia from the vestibular system is almost always associated with vertigo and slow nystagmus with or

without change of position. Hearing loss should be further evaluated to rule out inner ear issues.

Cerebellar Signs

1. Finger-to-nose test
2. Finger-to-finger test
3. Heel-shin test
4. Dysdiadokokinesia
5. Rebound phenomenon
6. Tandem walking
7. Romberg's test.

Reflex-hyporeflexia

Extrapyramidal signs. It is not uncommon for chronic progressive ataxia to be associated with extrapyramidal signs. In hereditary ataxias, extrapyramidal signs are often the indication of spreading of an underlying neurodegenerative process beyond the cerebellum and brainstem.

Proprioceptive sensory system. Loss of sensory input from spinocerebellar tracts to the cerebellum may cause sensory ataxia. Any impairment along the proprioceptive pathway may cause sensory loss (for example, Friedreich ataxia, ataxia with vitamin E deficiency, acquired sensory ataxias related to ataxic polyneuropathies [e.g., paraneoplastic sensory neuronopathy], Sjögren syndrome, diabetes mellitus, vitamin B₆ toxicity, Miller Fisher syndrome) This can be tested by examining vibration and proprioception at the great toe.

Investigations

- CT head: Head CT may detect a mass in the posterior cranial fossa and is extensively used in the clinical evaluation of acute stroke, especially for the rapid exclusion of intracerebral hemorrhage
- MRI Brain: for the structural lesion in the cerebellum or brainstem MRI is more appropriate. It is especially useful for ischemic stroke and infratentorial structural lesion evaluation.
- Vit.E, B12 levels
- Total cholesterol level, thyroid hormones

- NCV and EMG studies
- Toxicology screen(includes phenytoin level)
- Serology screen(for autoantibodies)
- CSF analysis: Syphilitic infections.
- Genetic Analysis

Management

The most important goal in the management of patients with ataxia is to identify treatable entities like as lesion must be recognized promptly and treated appropriately^[4] Malabsorption syndromes leading to deficiency of vitamin E, Vitamin B₁&B₂ levels in serum should be measured and vitamins should be administered. Ataxia due to hypothyroidism is easily treated. Patient with syphilitic infection, tabes dorsalis, Lymedisease appropriate antibiotic therapy should be instituted^[5] there is no proven therapy for any of the autosomal dominant ataxias.

CEREBELLAR ATAXIA IN AYURVEDA

In Ayurveda we don't get direct correlation for cerebellar ataxia but based on some signs and symptoms it can be correlated to *Vatavyadhi* and *Shiropaghata*.

Nidanas

Vayu gets aggravated by the following; Intake of Ruksha (unctuous), sheeta (cold), alpa (scanty), and laghu (light) food. Ati vyavaya (Excessive sexual indulgence), Ati prajagara (remaining awake at night).

Vishama upachara (inappropriate therapeutic measures); Ati Doshasruksravana (administration of the therapies which cause excessive elimination of Dosh and blood); langhana (keeping fast in excess); plavana (swimming in excess); Ati adhva (walking excess); Ati vyayama (excessive exercise); vichesta (other physical activities in excess); dhatu kshaya (loss of dhatu); excessive emaciation due to chinta (worry), shoka (grief) and due to vyadhi (affliction by diseases). Dukha shayya asana (sleeping and sitting over uncomfortable bed); krodha (anger); divaswapna (sleeping during day time); vegasandharanat (suppression of natural urges); due to formation of ama; abhigata (trauma); abhojana (abstention from food); marmabhighata (injuries to marmas). Riding

over gaja (elephant), ustra (camel), ashwa (horse) or sheegrayana (fast moving vehicle) and patamsanat (falling down from the seats on these animals and vehicles).^[6]

Any injury to Shiro Marma due to upaghta (external injuries) or affliction by vayu etc.^[7]

Samprapti

Vataja Ahara and Vihara, abhigata leads to Vata Prakopa this prakupita vata fills the Rikta srotas leads to vata vikara (vata guna like laghu, chala guna vrudhi).^[8]

Lakshanas

Injury to shiras give rises to manya-stambha (torticollis), Ardhita (facial paralysis), Chakshu-vibhrama (agitation of eyes), moha (unconsciousness), udvestana (cramps), ceshta-nasha (loss of motor activities), kasa (cough), shvasa (dyspnea), hanu-graha (lock-jaw), mukatva (dumbness), gadgadatva (lulling speech), akshi-nimilana (closure of eye lids), ganda-spandana (twitching of cheeks), jrumbana (yawning), lala srava (excessive salivation), svara hani (aphasia), vadana jihmatva (twisting of face).^[9]

Samprapti Ghataka

- Dosh: Vata Pradhana (Prana Vata, Udana Vata and Vyana Vata)
- Dushya: Rasa, Rakta, Mamsa, Medas, Majja
- Adhistana: Sarvasharira
- Srotas: Rasavaha, Raktavaha, Mamsavaha, Medavaha
- Sroto Dusti Prakara: Sanga
- Vyadhi Svabhava: Ashukari/Chirakari,
- Sadhyaasadhya: Kruchra Sadhya/Yapya.

Chikitsa (Shiropaghata Chikitsa)^[10]

- Abhyanga - Vatanashaka taila (Mahanarayana Taila, Mahamasha Taila, Masha Taila, Ashwagandha Bala Lakshadi Taila, Ksheerabala taila)
- Svedana - Shastikashali Pinda Sweda, Patrapinda sweda
- Ushnaupanaha

- Snehapana
- Nasyakarma - with Brumhananga tailas
- Avapidana Nasya with Lashuna or durva (in condition like avarana)
- Mrudu Shodana by giving snehapana (arohanartha) with Ashwagandha Ghrita or Ksheerabala Taila or brahmi ghrita. Followed by Sarvanga Abhyanga with Balaashwagandhlakshadi Taila followed by Mrudu swedana.
- Mrudu virechana - Satala ghrita, Tilavaka ghrita and eranda taila + ksheera. Gandharvahastadi taila (30-180ml according to patients strength)
- Snigdha, amla, lavana and ushna ahara Vatanulomaka dravyas
- Niruha basti prayoga-durbala, avirichya (dosha nirharana)
- Deepana Pachana dravya prayoga
- After virechana - after samasarjana krama jataragni vrudhi-again give snehana and svedana.^[11]
- Repeated use of madura, amla, lavana and snigdha ahara.
- Dhoomapana

Ghrita	Ashwagandha Ghrita Brahmi Ghrita Kalyanaka Ghrita Mahakalyanaka Ghrita
Asava/Arista	Dhanwantharishta Ashwagandharista Balarista Saraswatarishta
Kashaya	Vidaryadi Kashaya Sahacharadi Kashaya
Taila	Mahanarayana Taila Mahamasha Taila Masha Taila

	Ashwagandha Bala Lakshadi Taila Ksheerabala taila
Rasoushadi	Yogendra rasa Brihat vata chintamani Rasa Brahmi vati
Rasayana	Ashwagandha Rasayana Ajamamsa Rasayana Brihat Chagaladi Ghrita

DISCUSSION

The cerebellar ataxia syndrome is a heterogeneous group of disorders clinically characterized by the presence of cerebellar dysfunction. It is characterized by gait impairment, unclear speech, visual blurring due to nystagmus, hand incoordination and tremors with movement. Etiology of cerebellar ataxia can be classified based on the onset, progression, location of lesions and based on distribution. In Ayurveda by seeing etiology and symptoms it can be correlated to Shiromarmopagata. Due to indulgence in vataja ahara, vihara and abhighata, there is vata and raktapradushana leading to vata vyadhi.^[12] Hence in Shiromarmopagata we should adopt Samanya Vatavyadhi Chikitsa by using Virechana, Yapana basti, Nasyakarma and Murdni taila. Virechana acts on masthiska by acting on Pittadarakala due to its similarity with Majjadarakala. Yapana basti which does ayusho yapana (rejuvenation of brain cell) which is also mamsa-bala janana, vishama Jwara hara, sholahara and rasayana.^[13] Nasya karma and Murdni taila does the poshana of masthulunga and helps in correcting the pathology in the cerebellum.

Abhyanga, Upanaha and Shastikashali Pinda Sweda help in relieving the symptoms like hypotonia due to its balya and brihmana properties.

CONCLUSION

According to Hetu Vishesha and Sthana Vishesha we should adopt the treatment hence in cerebellar ataxia where there is abhighata to Shiro Marma by infection, vascular accidents, alcohol etc. by seeing the causes, treatment is adopted accordingly. Cerebellar ataxia caused due to reversible causes has to be identified and treated accordingly such as

alcohol and intoxication caused ataxia. This has to be treated according to Mada/Madatyaya Chikitsa. If due to infections like Acute viral cerebellitis Sannipataja/Vishama Jwara Chikitsa is to be adopted and when caused due to nutritional deficiency Santarpana Chikitsa is adopted. Cerebellar ataxia caused due to hereditary and autosomal dominant ataxia can only be managed by adopting Samanya Vatavyadhi Chikitsa. If it's caused due to vascular causes or local lesion is treated according to Shiromarmopagata Chikitsa.

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How to cite this article: Dr. Ashwini A., Dr. Rajashekhar C.V. Cerebellar Ataxia and its management - An Ayurvedic Approach. J Ayurveda Integr Med Sci 2019;6:108-114.

Source of Support: Nil, **Conflict of Interest:** None declared.
