"A CLINICAL STUDY ON KAMPAVATA (PARKINSON'S DISEASE) AND ITS MANAGEMENT WITH TRIGUNA RASA"

By

Vijayamahantesh.s

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ABBREVIATIONS

C.S. - Charaka samhita

S.S - Sushruta Samhita

A.H. - Astanga Hrudaya

A.S. - Astanga sangraha

B.P. - Bhavaprakasha

B.R. - Bhaishajya Ratnavali

M.N - Madhava Nidana

Sha.S - Sharangadhara Samhita

Y.R. - Yoga Ratnakar

R.R.S - Rasaratnasammuchhaya

C.D. - Chakradutta

V.S. - Vangasena

Bh. S - Bhela samhita

R.J.N - Rasa Jala nidhi

R.T - Rasa Tarangini

S.K.D - Shabda kalpa drruma

Ba.Ra - Basavarajeeyam

S.Y - Sahasrayoga

BT - Before Treatment

AT - After treatment

UE - Upper extremity

LE - Lower extremity

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Abstract: ***A Clinical study on kampavata (Parkinson's disease) and its management with Triguna rasa.***

Key words; Kampa vata, Parkinson's disease, kampa, stambha, chesta sangha, Avanamana,vak vikruti, gatisanga, tremor, pill rolling, cog wheel rigidity, Bradykinesia dopamine, Basal ganglia.

Kampavata is a slow progressive disorder of late adult life and is one of most prevalent neurological disorder. Parkinson's disease, known in Ayurveda as "Kampa Vata," is a neurological disorder affecting 1% of the population over age 65 and is the fourth most common neurological degenerative disorder found in the elderly. In Charaka Samhita vepathu has been described as one of the eighty types Vata nantmaja vyadhi and Kampa has been mentioned one among them. There are many vata vyadhis commonly seen but Kampavata is one of the rare mentioned under vata vyadhis because of its crippling nature and non availability of curative treatment this disease has remained a great problem in the ageing society. The present study objective is to evaluate the efficacy of Triguna Rasa in Kampavata. A simple random single group observational study is adopted here. Kampavata expresses the signs and symptoms as Karapadatale Kampa, Dehabhramana, Nidrabhanga and Matiksheena. Due to the etiological factors, as mentioned the Vata gets aggravated by its chala, ruksha and sheeta properties. Prana, Udana and Vyana are most affected among the five types of vata, which in turn vitiate the Mastulunga Majja in the Shiras; these vitiated doshas selectively affect the Vatavaha srotas in the Mastishka. This vitiation of Vatavaha Srotas impairs the motor functions of the body leading to Kampa, Dehabhramana, Matiksheena etc. according to contemporary understanding it is said to be Parkinson's disease, most common

neurological degenerative disorder found in the elderly, where the nerve cells containing melanin, called substantia nigra, are concentrated in the part of the brain called basal ganglia which secrete dopamine. As these cells deteriorate, the production of dopamine that carries messages within the brain falls, and the result is the characteristic symptoms as pill rolling tremors, rigidity, Bradykinesia etc is seen. However, most patients do not develop all of the symptoms associated with the condition. In most cases, primary symptoms include slow movements (bradykinesia), tremor, rigidity, and parkinsonian gait. Symptoms of Parkinson's usually begin on one side of the body.

In Sahasrayoga Triguna Rasa is directly indicated for Kampavata. The main ingredients of this yoga are Haritaki and Kajjali. As Hartaki choorna is indicated in conditions like mastishka dourbalya & best in all types of Vatavyadi. Parada & Gandhaka (kajjali) mitigates all types of rogas & even tridoshas. Thus an effort is made to evaluate the efficacy of internal administration of Triguna Rasa in Kampavata.

Present study registers 20 patients after fulfilling the criteria of diagnosis in a single group. All the patients were examined before and after the trail, according to the case sheet format. Data before the treatment and after the treatment recorded and at the end of study both were compared for assessment.

In the present study Kampa was seen in all patients, gatisanga was seen in 19 patients, 18 patients had vakvikruti, 17 patients had sthamba, all patients had Chestasanga and only 5 patients had Avnamana.

Objective parameters like, Tremor was seen in all 20 patients, Rigidity was seen in 17 patients, 19 patients had Bradykinesia, 16 patients had Gait impairment, 18 patients had Dressing difficulties and 11 patients had problems with postural stability.

Statistical analysis showed the treatment is more highly significant in the parameters, Kampa, Gatisanga, Stamba, Chestasanga, Tremors and Rigidity. In the parameters *Stamba* and *Chestasanga* have same effects with positive correlation between before and after treatment. In the parameters *Gait, Vakvikruti* and *Bradykinesia* treatment has less significant. In the parameters *Avanamana and Postural stability* treatment not significant even they positive correlation before and after treatment. This may be because of the involvement of whole vertebral column which cannot be corrected with medical management.

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Chapter 1 ***Introduction***

Ayurveda is the science that imparts all knowledge of life. It defines health and factors responsible for its maintenance and promotion. It was the science which did not start with fundamental understanding but developed from observation of phenomenon which were then classified, analysed and systematised. One of such science is the science of life, "Ayurveda"

Health is essential for enjoyment of all the worldly pleasures in a righteous manner. Ayurveda defines a useful, harmful, happy and unhappy life and provides knowledge which is beneficial to life in short it discusses all aspects of human life. Ayurveda is the everlasting supreme science of medicine because it deals with every aspects of life, particularly of human being since time immemorial.

The 'Vata' which is the motivator and controller of other two doshas, is responsible for the manifestation of almost all diseases. Vataja nanatmaja vikaras are limited to eighty in various classics, but when we group all the disorders of vata mentioned in various classics, the number exceeds eighty.

Major neurological problems come under vata vyadhis. Kampavata as one among them manifests with "Dehabhramana" (postural instability), "Karapada tale kampa" (tremors in hands and legs), "Matiksheena" (dementia), and "Nidrabhanga" (sleeplessness).

There are many vata vyadhis commonly seen but Kampavata is one of the rare mentioned under vatavyadhis because of its crippling nature and non availability of curative treatment, this disease has remained a great problem in the ageing society which usually affects after the age of 50 years. The disease is increasing in its frequency with the world population showing an incidence of 1-2 per 1000 population and has an equal sex distribution.

Historcial background of the disease is suggestive of the fact that though, in vedic period the disease 'Vatikrit' was known but the typical clinical entity identical to Kampavata was not mentioned.

According to Ayurveda, Kampavata is a Vata Nanatmaja vikara. During the period of Charaka and Sushruta clinical manifestations of kampavata like kampa, sthamba, chestasanag, vakvikriti etc was not explained as one disease instead explained under various contexts majority of the symptoms of kampavata were found in kaphavrita udana and kaphavrita vyana but no single avarana process completely covers the symptoms of kampavata . Actually many of the experts tried to provide a suitable Ayurvedic nomenclature for the Parkinson's disease e.g. - sakamp-paksaghata and vepathu etc.

Sakamp-paksa-ghata was the nomenclature suggested in view of the synonym of Parkinson's disease as paralysis agitans. The term *vepathu* was considered in view of acceptance of Charak followed by Madhavkar as a separate clinical entity. Both of the above terms need to be appropriately elaborated. Parkinson's disease has its three types Viz,

- Idiopathic form
- Arteriosclerotic form
- Post-encephalitic form

The idiopathic form is known as the true Parkinsonism and paralysis agitans, if we consider the sakamp-paksaghata as synonym of paralysis agitans then remaining two forms of the disease or not explainable. Further sakamp-paksa-ghata has no classical basis. The term vepathu though has been widely accepted as alternative term for Parkinson's disease but in view of the following point.

(A) This does not explain the other symptoms of Parkinson's disease except tremor.

(B) Vepathu has been used in different contexts for different meanings eg. in vatika jvara for rigors in mahaswasa for giddiness and so on. It initiates to an endless debate.

It is therefore suggested that in view of classical reference, a complete clinical entity having symptoms, signs etc. the term **Kamp-vata** the most appropriate term, for the first time explained by Basavarajiyam with clinical features similar to that of Parkinson's disease.

Direct reference to the Parkinson's disease in the ancient Ayurvedic literature is sparse and refers only to related symptoms including tremors. Thus, the condition is referred to the modern ayurvedic literature by various names for kampavata (tremors due to vata), vepthu (shaking), prevepana (excessive shaking), shirakampa (head tremor), spandin (quivering) and kampana (tremors).

This disease doesn't have or knows no boundaries with regards to race, rationality, gender or social class. As this can interfere with activities of daily living, there is need of assistance with more routine activities of daily life such as grooming, bathing, dressing and feeding.

Parkinson's disease is characterised by abnormalities of motor function, slowness of movement and an inability to start a movement are the hallmarks of the disease. The motor disturbance also results in diminished facial expression and decreased rate of blinking. The second important manifestation is the stiffness and rigidity so that the person encounters raised resistance when attempting to move a limb and a joint. The third manifestation is a tremor that may be quite asymmetrical, occurring in one just one hand, or may involve both hands and the trunk. As the disease

progresses, problems with balance become quite limiting, and falls may occur frequently. Alternatively, with disease progression, episodes of freezing may occur, during which voluntary movement becomes almost impossible. Finally some individuals suffer from dementia, which appears to be an integral part of Parkinson's disease process. The basic pathologic change is degeneration of a group of nerve cells deep within the centre of the brain in an area called substantia nigra. These cells use Dopamine as their neurotransmitter to signal other nerve cells. As these cells degenerate and, stop functioning, dopamine fails to reach the areas of the brain that affect motor functions. From the scientific point of view Parkinson's disease is of interest because of the insight it gives into the process involved in translating thoughts and intentions into the appropriate actions of their clear expression. It produces a number of behavioural changes stemming from a disruption of the brain mechanisms that mediate these processes.

Therapy for Parkinson's disease is aimed at replacing dopamine. Since the blood brain barrier prevents dopamine from entering the brain from blood stream, a precursor of dopamine (L-dopa) that will enter the brain is given.

Parkinson's disease remains the only neurodegenerative disorder that has demonstrated significant responsiveness to therapeutic intervention. However the treatments which are present now have a little evidence that this treatment changes the course of disease. In this regard newer targets are being explored.

Kampavata (Parkinson's disease) is slow progressive disorder of late adult life and is one of the most prevalent and common neurological disorder occurs with more or less equal frequency in all countries around the world. In present era there are many such neurological disorders which are rising in their incidence day to day, such neurological disorders can be considered under the concept of vatavyadhi.

Kampavata (Parkinson's disease) being one of them having the pathology of degeneration in a part of the brain. Nearly two centuries have elapsed since disease Parkinson's is known, better treatment are still being sought.

Parkinson's disease is more treatable than other neurodegenerative disorders. Nevertheless many features of Parkinson's disease are resistant to treatment. In spite of advancing medical sciences, the treatment of Parkinson's disease remained symptomatic no curative treatment is available for the disease.

In modern medical science lot of research works have been done but still no radical therapy available for Parkinson's disease. So there is continuous search for safer, convenient, effective and economical remedy for Parkinson's disease.

Because of its crippling nature and non availability of curative treatment Parkinson's disease has remained a great problem in aging society. However the treatments which are present now have a little evidence that the existing treatment can change the course of disease.

In Ayurveda, Snehana, Svedana, Niruha basti, Virechana, nasya, Anuvasana basti and Sirobasti been indicated in management of Kampavata. The management of Kampavata can be done by the oral administration of Triguna Rasa which is explained in Sahasra yoga as it contains Haritaki ,Parada and Gandhaka, the karma of Haritaki over nadivaha samsthan is balya and medhya indicated in conditions like mastishkya

dourbalya, nadidourbalya and best in all vata vyadhis, kajjali (parada and gandhaka) mitigates all types of roga and even tridoshas.

The present study intended to focus on the disease evaluation i.e. kampavata vis-a-vis Parkinson's disease and its management with Triguna rasa.

AIMS AND OBJECTIVES OF STUDY

1) To evaluate the efficacy of Triguna Rasa in Kampavata

2) To evaluate the efficacy of Triguna Rasa in Minimizing effects of Parkinson's disease.

3) To study the literary work in Kampavata.

4) To study the literary work in Parkinson's disease.

HISTORICAL REVIEW

Records of the past events always enlighten us regarding the depth of subject to understand it scientifically. The methodical record of the past events about Ayurveda begins from the Vedas. Hence it is always must to go through record of the past events before moving into the subject Kampavata and its management.

The history of Kampavata can be reviewed under following kala.

1. Veda kala (2500 BC - 1000 B.C)
2. Samhita kala (1000 BC - 100 A.D)
3. Sangraha kala (100 A.D - 800 A.D)
4. Nighantu kala (800 A.D. - 1700 A.D)
5. Adhunika kala (1700 A.D. onwards)

1. Vedic Period

Vedas are considered as the oldest recorded documents of knowledge and root of all available knowledge. Description of kampavata begins from veda where it has been mentioned in Rigveda that Indra had suffered from vepathu[1]and in Atharvaveda some description regarding vepathu[2] is also available but word denoting kampavata is not available as a separate entity in Atharvaveda.

Samhita kala

Charaka samhita

In Charaka samhita kampavata is found by the name vepathu and has been included in the Nanatmaja vata vyadhi[3] some symptoms of kampavata are kampa and sthamba these symptoms are found in some pathological conditions of vatavyadhi, as Charaka explains decrease of pitta and increase of vata and kapha causes symptoms like vepana (kampa) and sthamba.[4] Even Charaka further explains the increase of vayu when

affects marma leads to shareera kampa, [5] this concept provides the pathology of tremors relating to brain (marma).

Many other references regarding kampa are found in name of vepathu, ,vepana, etc kampa is also one of symptom of many other diseases like vataja jwara,[6] vataja unmade,[7] ananthavata,[8] vishamasannipatika jwara[9] and vatika pandu.[10]

Sushruta samhita

Acharya Sushruta has mentioned the symptoms like chestasanga, sthamba and gurugatrata in the condition of kaphavrita vyana.[11] He has described vata vyadhi kills the patient when accompanied with complication such as tremors.[12] Tremors have been mentioned as the updrava of prameha.[13]Vepathu described as symptom in sthavaravisha vignyana.[14] Use of Avi ghrita is mentioned for management of kampa.[15]He has explained Tremors as one of the complication of ardita.[16]

Kashayapa samhita

Vepathu has been listed under the vata nanatmaja disorders.[17]

Bhela samhita

In this treatise, shirakampa has been mentioned to occur due to regular intake of ruksha diet and a person who has got the tendency to develop udavarta is more prone to this disease.[18] Snehapana, [19] nasya and anuvasana basti are the treatment suggested for shira kampa. Rasnataila has been indicated in the management of gatrakampa.[20] kampa as a symptom is seen in Astimajjagata vata[21]

Sangraha kala

Astanga sangraha and Astanga hridaya

In these treatise of Vaghabata, explanations and few references regarding kampavata is available. Kampa as a symptom of parkupita vata [22] is mentioned and also eventually explained kampa as one of symptom of sarvanga vata, [23]even kampa is

mentioned in kpaha kshaya, pitta kapha kshaya and rasakashya conditions. [24]Sthamba as a symptom mentioned in condition of mamsagatavata and medhogata vata[25.]

Madhava nidana

Acharya madhava explained the disease vepathu in vatavyadhi for the first time, which is characterised by sarvanga kampa (tremors all over the body) and shirokampa. The commentator vijayrakshita has further explained that in shirokampa the tremors of limbs can also be included which indicates the crucial picture of kampavata (Parkinson's disease) [26]

Sharangadhara samhita

Sharangadhara has mentioned kampa under vatajaroga[27] and treatment for sarvanga kampavata with maharasnadi kwatha is.[28]

Medicine like maharasnadi kwatha should be taken with food or after food in case of kampavata has been mentioned in his purva kahanda.[29]Shira kampa been explained under shiroroga.[30]

Various medicines have been explained like Devadarvadi kwatha in kampa, [31] Dhattur taila in shirakampa, [32] Varuni taila in hasta and shirakampa, [33] Mashadi taila in shaka kampa and shira kampa.[34] Shirobasti for seven days will cure even dreadful vata disorders and shirakampa. [35]

Bhavaprakasha

Acharya Bhavamishra has explained symptoms of sthamba and kampa in condition of snayugata vata. [36] He explains even in excessive use of tikta rasa will lead to kampa.[37]

In panchakarma vidhi adhaya, explaining bruhana nasya guna, acharya explains bruhana nasya helps in treating tremor.[38]While describing sankhya of vatavyadhi, kampa and sthamba are also explained.[39] Some scattered references

regarding kampa is found as a feature while explaining kalayakhanja[40]and sarvanga vata lakshana.[41]

Yogaratnakara

He has adopted the description mentioned same of Madhav nidana, he has also explained sarvanga kampa and shiro kampa under the disease vepathu in vatavyadhi chapter. [42]

Chakradatta

In Chakradatta, the treatise of treatment of diseases, the conditions like bahu kampa, shira kampa and hasta kampa can be treated with recipes like Dwitya masha taila and maha masha taila.[43]

Vangasena samhita

He explained vepathu as sarvanga kampa under vatavyadhi.[44]Prime importance given regarding the treatment principles of kampavata, treatment like abhyanga, sweda, nasya, niruha basti, anuvasana basti, virechana and shirobasti which are useful.[45] Masha taila[46]and Mahamasha taila[47] have been indicated in the management of hastakampa, shirakampa and gatrakampa.

Basavarajeeyam

A more detailed diagnostic approach with illustration for the first time provided by the author with explaining the symptoms of kampavata viz karapade tale kampa ,dehabrhamana,nidra bhanga,ksheenamathi. He has also indicated masha taila and karpasataila for the treatment of bahukampa.[48]

Bhaishajya ratnavali

In this treatise the remedies like nakula taila, nakula ghrita, mahamasha taila, vijayabhairava taila and sarvanaga kampari rasa etc have been recommended for kampavata.[49]

HISTORICAL BACKGROUND OF PARKINSON'S DISEASE

James Parkinson was born on April 11, 1755, to John and Mary Parkinson. They resided at number 1 Hoxton Square in the Parish of St. Leonards of Shoreditch, Middlesex County, where Parkinson lived his entire life. He qualified as a surgeon in 1784 at the age of 29 Parkinson published his medical classic *An Essay on the Shaking Palsy* in 1817 at the age of 62. This was a comprehensive treatise containing 5 chapters and 66 pages on the subject (which he called "paralysis agitans"). The review includes his experience with 6 patients.

Parkinson indicates that in order to understand the natural history of the disease one needs to either observe patients as they evolve or see patients at various stages of the disease or receive a correct history of its symptoms even for several years. The first two chapters are dealt with, Chapter *1*: Definition– History–Illustrative Cases and in Chapter *2* he has narrated about Pathgnomonic Symptoms. He notes that the tremor and the gait disorder, the most visually dramatic features, are the pathognomonic symptoms of the disease the tremulous agitation, and the almost invincible propensity to run, when wishing only to walk, He indicates quite succinctly that the resting tremor is the characteristic feature and one that could differentiate paralysis agitans from other forms of tremors. "The propensity to lean forward becomes invincible, and the patient is thereby forced to step on the toes and fore part of the feet, impelled to take much quicker and shorter steps, and thereby to adopt unwillingly a running pace." In the very late stages he indicates that "the trunk is almost permanently bowed, muscular power is more decidedly diminished, and the tremulous agitation becomes violent. With regard to sleep the sleep becomes much disturbed." "The power of articulation is lost," He vividly describes the features of a masked face and drooling by indicating that the chin is now almost immovably bent down upon the sternum. Constipation, a feature that is present

in a large percentage of patients, *Chapter 3* provides the differential diagnosis for the shaking palsy, In *Chapter 4* Parkinson ventures a guess on the location of the lesion and the aetiology of the disease process within the brain. Finally, in *Chapter 5*, he addresses possible treatments. He is the first person to have discussed neuro-protective therapies. Throughout the world this illness is now called "Parkinson's Disease." [50] To this disease Marshall Hall in 1841 given the name "paralysis agitans" which is a Latin translation of the term shaking palsy.

French Neurologist Charcot (1825 to 1890) considerably increased the knowledge of the disease. He pointed out rigidity as if their joint were soldered and spoke of difficulty in performing movement and emphasized that there was slowness in performance, rather than genuine reduction of muscular power. He claimed that experiments with dynamometers showed no real weakness.

When this disease was described affection of brain was not possible to examine. In the mid-nineteenth century several patients who had died from Parkinson's disease were examined but no specific abnormality could be recognized in the nervous system. However in 1893, a patient who suffered Parkinson's disease during his life time and which was limited to one side of the body, post mortem examination of him disclosed a small tumour pressing on the opposite basal ganglia. In the same year Prof. Brissaud suggested that focal damage to the substantia nigra might cause Parkinsonism. Inspired by his suggestion a young doctor called Tretiakoff (1919) meticulously examined the substantia nigra in the nine patients and was able to conclude that the principal symptoms of Parkinson's were due to damage within the pigmented nucleus. Later Hassler (1937) and Greenfield (1958) confirmed Tretiakoff's earlier findings.

After the Second World War Indian physicians reported role of Rauwolfia in high blood pressure. So its active ingredient reserpine was introduced and within a year

of its introduction a most spectacular side effect was noticed. A condition indistinguishable from Parkinson's disease developed in few patients, and when the drug was discontinued the signs of Parkinson have disappeared. This exciting discovery opened up a new era. Arvid Carlson (1958) a young Swedish scientist found that when he injected reserpine into rats a state of rapid immobility was produced resembling the severe form of Parkinson's disease. He was able to demonstrate that in the brains of affected animals there had been a severe depletion of certain chemical substances, including dopamine. He then found that after giving levodopa injection, the chemical precursor of dopamine the immobilized rats recovered rapidly. 3 years later in Vienna, Oleh Hornykiewicz (1960) discovered that Parkinson's brains contained very little amount of dopamine in the basal ganglia. In 1967, Cotzias in New York gave large amounts of levodopa and then true potential of this form of therapy was realized.

In 1976, a young college student abused meperidine analogue MPTP as heroine substitute for 6 months. He developed severe Parkinsonism. Autopsy revealed that loss of basal ganglia was limited to substantia nigra. In summer of 1982 MPTP drug abusers began arriving in hospitals of California with what appeared to be advanced, unalloyed Parkinsonism. Langston (1985) showed that this compound is selectively toxic to substantia nigra.[51]

VYUTPATTI AND PARIBHASHA

Description regarding vyutpatti and paribasha is very important before advancing into the disease kampa vata.

Kampavata comprises of two words, Kampa and vata. Individual meaning regarding these components are as follows.

Kampa

The word kampa belongs to masculine gender. It is derived from the root *Kapi* and suffixed by *ghan* which gives the meaning 'to move' or to 'shake.'

Gatradi chalanam,[52] - that which produces shaking or movements in the body. The word kampa conveys the meaning of shaking or tremor.[53]

Vata

The term vata belongs to masculine gender. It is derived from the root *Va* and suffixed by *ktha*.[54]

"*Va-gatigandhanayoho*"[55] Vata is one of the three humours of the body. Gati and gandhana are the two important functions of vata i.e., all the motor and sensory functions in the body are governed by vata.

By this one can define the term kampavata as one of vataja disorder which has cardinal sign of kampa. In total the term Kampavata means the disorder of impaired vata, in which the prime clinical manifestation is kampa.

Definition of Parkinson's disease

Marsden (1994) has defined Parkinson's disease as "a clinical syndrome dominated by a disorder of movement consisting of tremor at rest, rigidity, elements of bradykinesia, postural and gait abnormalities associated with a distinctive pathology, consisting of degeneration of pigmented brain stem nuclei including the dopaminergic substantia nigra, pars compacta, with the presence of lewy bodies in the remaining cells."[56]

PARYAYA

In classics paryayas of kampa vata is not found.

Synonyms of Parkinson's disease

Synonyms used in contemporary science for Parkinson's disease are as follows.

Shaking Palsy – In 1817, James Parkinson an English Physician for the first time explained this disease as shaking palsy.

Paralysis Agitans – To Parkinson's disease Marshal Hall in 1841 gave the name Paralysis agitans which is a Latin translation of term shaking palsy.

DISEASE REVIEW

Before starting any research on a disease, the researcher should have the knowledge of Anatomy and physiology of related disease. Explanation regarding aetiology, pathogenesis and available different treatment modalities of the disease along with necessary information available in other system of medicine shall be collected and correlated.

Parkinson's disease is a progressive disorder of the central nervous system. It is a neurodegenerative disorder affecting the dopaminergic neurons of the substantia nigra in the basal ganglia.

Shareera Rachana and Kriya Vivechana in Ayurveda

Charaka explaining the importance of Shiras explain head as the Uttamanga where vital breath and all sense organs are located and considers as best of all organs.[57]Here chakrapani opines shiras as seat of prana or vayu. Charaka accepted Shiras as seat of indriyas, indriyavaha and pranavaha srotas.[58]Here pranavaha srotas are channels which convey prana. Shiras is chief working seat of pranavayu.[59] Pranavayu supports buddhi and manas.[60] Chakrapani also opines that seat for vayu (pranavata) is Shiras. Acharya Vaghabhata has compared shiras with root of plant, as for well being and nutrition of plant root is very important, likewise head is also an important part of body for normal functioning, as diseases of shiras attack the functional root i.e. head of human, they should be treated as early as possible.[61]Mastishka in the shiras is nothing but the majja as said by chakrapani, and mastishka develops from prasada bhaga of asrik and sleshma.

Certainly after knowing matishka it is important to know the relation between vayu and mastishka.

Functions of the body are controlled by the vayu at mastishka which is situated at the top.[62] Charaka in sutra sthana while explaining the nidana of shiro roga mentions due to vatakara nidana sevana, vata gets vitiated and causes diseases of shiras.According to acharya Bhela Shiras is the seat for vayu and manas. Vata and manas work with each other like milk and water.[63]

ANATOMY OF BASAL GANGLIA

THE **NERVOUS SYSTEM** is the most complicated and highly organized of the various systems which make up the human body, it is the mechanism concerned with the correlation and integration of various bodily processes, the reactions and adjustments of the organism to its environment.

Parkinson's disease is a progressive motor disorder resulting from the selective death of a very tiny group of neurons in the brain called the substantia nigra. These neurons may be few in number, but they do something very important. They secrete a neurotransmitter called dopamine into a part of the brain called the basal ganglia.

Lesions of corpus striatum in basal ganglia cause Parkinson’s disease because the basal ganglia work kind of like a switch that is involved in choosing to initiate motion. When brain is considering initiation of motion, a signal goes to the basal ganglia. The basal ganglia make a computation, and then a signal is sent back either encouraging or discouraging the activation of that motion, so the concept of basal ganglia as follows.

The basal nuclei are subcortical intracerebral masses of grey matter forming important parts of the extrapyramidal system. Ganglia are the collection of neurons in the peripheral nervous system. The structures that comprise “Basal ganglia” are neurons within the central nervous system.

The basal ganglia are a collection of nuclei that have been grouped together on the basis of their interconnections. The nuclei play an important role in movement and disorder of movement. Basal ganglia generally include the structures like Corpus striatum, Substantia nigra and sub thalamic nuclei. Brief description regarding the functional anatomy of the basal ganglia system as follows**:**

The corpus striatum has received its name from the striped appearance which a section of its anterior part presents, in consequence of diverging white fibres being mixed with the gray substance which forms its chief mass. A part of the corpus striatum is imbedded in the white substance of the hemisphere, and external to the ventricle it is termed the **lentiform nucleus** the remainder, however projects into the ventricle, and is named the **caudate nucleus.**

Caudate nucleus is a **c** shaped nucleus which is surrounded by the lateral ventricle the concavity within it encloses the thalamus and the internal capsule. The nucleus has head, body and tail.

The *head* forms the anterior horn of the lateral ventricle and the medial wall forms the anterior limb of the internal capsule.

The *body* forms the central part of the lateral ventricle and lies medial to the posterior limb of the internal capsule.

The *tail* forms the roof of the inferior horn of the lateral ventricle, and ends by joining the amygdaloid body at the temporal pole.

Lentiform nucleus is a large lens shaped nucleus forming the lateral boundary of internal capsule. It is divided into two parts by a large part called **putamen** and **globus pallidus** which is made up of large motor cells.

Morphological divisions of corpus striatum

Corpus striatum morphologically divided into **paleostriatum** represented by palladium. The **neostriatum** it is represented by caudate nucleus and the putamen.

Connections of Corpus striatum

The caudate nucleus and the putamen are afferent nuclei, while the globus pallidus is the efferent nuclei of the corpus striatum.

Functions of Corpus striatum

It regulates the muscle tone and thus helping in smoothening voluntary movements. It controls the automatic associated movements of the body and co ordinate movements of different parts of the body for emotional expression. Lesions of the corpus striatum give rise to hyper tonicity or lead pipe muscular rigidity, involuntary movements, like tremors and result in Parkinson's disease.

Substantia nigra

It is one of component of basal ganglia which is made of small un-pigmented and large pigmented cells. Substantia nigra serves as centre for integration of sensations, between general body surface and special senses of hearing, sight and smell. The substantia nigra consists of two components dorsal cell rich portion reffered to as the *pars compacta* and ventral cell sparse portion denoted the *par reticulate*. Most of the neurons in the pars compacta of the substantia nigra in the humans are pigmented because of the presence of neuromelanin. These cells contain the neuro transmitter dopamine.

Sub thalamic nucleus of Luys.

It is a biconvex in nature and is situated dorsa-lateral to the red nucleus and it appears to be the important site for integration of a number of motor centres. Discrete

lesions of this area in humans lead to *Hemiballismus* which is characterised by violent involuntary abnormal movements on one side of the body involving mostly the arms.

Connections of the Basal ganglia

Unlike most other components of the motor system, the basal ganglia do not make direct connections with the motor neurons in the spinal cord. Their influence on motor activity is exerted indirectly through their connections with the motor cortex.

Functions of basal ganglia

It is concerned with motor activities, the various functions of basal ganglia is as follows.

- Control of voluntary motor activity
- Control of reflex and muscular activity
- Control of muscle tone
- Control of automatic associated movement

Neurotransmitters of Basal ganglia

The entire nervous system is made up of individual units called nerve cells. Nerve cells actually serve as a 'communication network' within the body. To communicate with each other, nerve cells use a variety of chemical messengers called neurotransmitters which is a chemical mediator responsible for the transmission of impulse through the synapse. Neurotransmitters also allow nervous system to communicate with body's muscle and translate thought into action.

The functions of basal ganglia on motor activities are executed by some neurotransmitters released by nerve endings within the basal ganglia. The neurotransmitters released in basal ganglia are

- Dopamine
- Gama-aminobutyric acid (GABA)

- Acetylcholine
- Noradrenaline.

Among all these neurotransmitters, Dopamine and GABA are inhibitory neurotransmitters.

One special important, neurotransmitter is dopamine, which is released by dopaminergic fibers from substantia nigra to corpus striatum (nigro strial fibers). Dopamine is very crucial for human movement and transmits messages to the striatum which in turn initiate and control the movement and balance. These dopamine messages make sure that muscles work smoothly, under precise control and without unwanted movement. Acetylcholine is another neurotransmitter which is released by fibers from cerebral cortex to caudate nucleus and putamen; it works with dopamine to carry out smooth movements.

Dopamine Metabolism

Dopamine is an inhibitory transmitter in the basal ganglia. Dopamine is one of the three catecholamine neurotransmitters, the others being nor adrenaline and adrenaline. Dopamine synthesis involves two enzymes – Tyrosine hydroxylase (TH) and Dopa decarboxylase. Dopamine is metabolized by two enzymes – Catechol-o-methyl transferase (COMT) and Monoamine oxidase B (MAO-B).

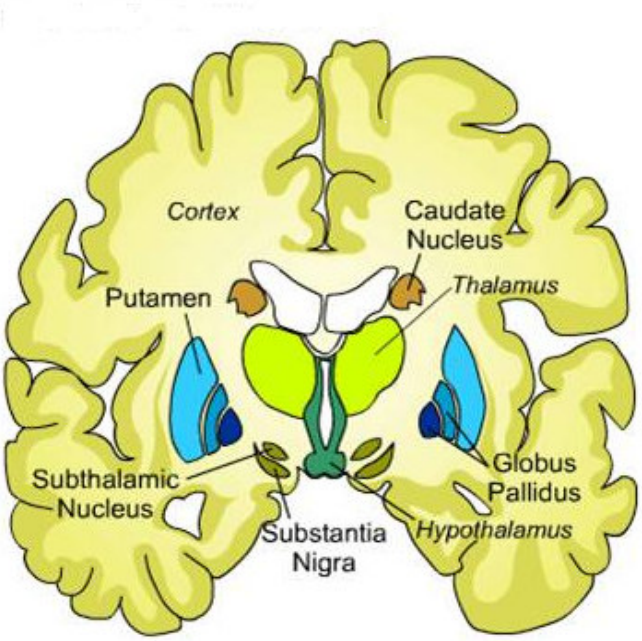


Figure No 1 .Coronal section of brain showing basal ganglia

Concept of Vatavyadhi

The word vata is derived from the verb *va* which means movement. The biological element vata is derived from the combination of space and vayu. The biological actions of vata is it controls the movement of molecules, cells as well as the body and division of cells, it is responsible for the organization of all the tissues of body. By bringing together *kapha* molecules and cells, it helps in regeneration and conjugation of tissues. By stimulating movement and activity it increases the catabolism of the body. By controlling the speed of action, it controls the metabolic process in the body. It is the leader among the tridoshas. It is responsible for origin, maintenance and destruction of life. Strength and life depends on vata.

Action of vata in the nervous system

Vata controls the action of the prefrontal lobe, motor cortex and spinal cord. It is responsible for smooth and co-ordinate movements. It carries all sensory impulses to their centers and maintains the efficiency of sense organs. Sound and touch together

with their sense organs originates from vata. It stimulates all sensory as well as motor centers.[65]

Many of diseases explained under the concept of vatavyadhi appear to have much more similarities with diseases of nervous system. If sensory tracts are affected symptoms like shoola (pain), supti (numbness), are produced. If motor tracts are affected then abnormal movements like stambha (stiffness), akshepa (convulsions), kampa (tremor), sankocha (contractures) will result. If nerve tracts are concerned with the perception of light, sound, taste, smell and touch various diseases like – Anosmia (gandha nasha), optic atrophy, tinnitus, vertigo, loss of taste will result. When motor tracts in connection with sense organs are involved, many deficiencies in their movements such as facial paralysis (ardhita), hemiplegia (pakshaghata) etc. will occur. Majority of the vatavyadhis can be compared to nervous system disorders on the basis of clinical features.

- Pakshaghata - Himiplegia
- Ekangavata - Monoplegia
- Sarvangavata - Quadriplegia
- Ardhangavata - Paraplegia
- Ardhita - Facial paralysis
- Manyastambha - Cervical spondylosis
- Gridhrasi - Sciatica
- Apabahuka - Brachial neuralgia
- Kampavata - Parkinson's disease, Chorea

Vata Sthana

After understanding vata and vatavyadhi, it is necessary to know the sthana of vata. A critical search of samhitas provide the information, mastishka is the sthana of vata and manas. The functions of vata can be observed in sarva shareera, main karyalaya is mastishka.

According to Atharvaveda, Hridaya and Shiras are intimately connected. On this relationship, it is explained that vayu is located in the upper portion of mastishka and controls everything. Charaka opines, Shiras is the seat for all vital centers and senses, it is best among all parts of the body and it is the sthana of vayu.[66]

With all the above quoted references, it can be concluded that, brain is the seat for vata, and every function of the body is due to proper functioning of vata or nervous system.

Nidana

Acharya Charaka defines Nidana are the factors, which cause the disease. Treatment becomes easier by knowing the causative factors of a disease. In this light it has been clearly stated that 'Nidana Parivarjanam' is one type of Chikitsa. According to Ayurveda, consideration of aetiological factors is important for the diagnosis, prognosis and line of treatment.

Nidana refers to all the causative factors which are responsible for the initiation and progress of the disease process. Nidana is defined as; *"Vyadhi utpatti hetu nidanam"*[67] That is, nidana is the main cause for occurrence of the disease.

The etiological factors of vatavyadhi in general have been described in our classics, but separate nidanas for Kampavata not explained. Kampavata is one of the vatavyadhi and it is also told that, *Na kampo vayuna vina*[68] without vata, there is no manifestation of kampa.

As specific nidanas are not being mentioned for kampavata so, the general nidanas explained for vatavyadhis can be considered here.

Table No. 1. Showing Samanya Vataprakopaka Nidanas

Nidana (**Aharaja Nidana)**	CS	SS	AH	MN	YR	BP	HR
Guna Pradhana							
Alpa	+		+	+	+	+	
Laghu	+			+	+	+	
Rooksha	+	+	+	+	+	+	+
Sheeta	+	+		+	+	+	+
Rasa Pradhana							
Katu		+				+	+
Kashaya		+	+			+	+
Tikta		+	+			+	
Dhanya Varga		+					+
Adhaki		+					+
Harenu		+					+
Kangu		+					+
Koradoosha		+					+
Kalaya		+					+
Masoora		+					+
Mudga		+					+
Neevara		+					+
Nishpava		+					+
Raktashali		+					+
Rajamasha		+					+
Shyama		+					+
Yavagu							+
Harita Varga							
Gunjana							+
Kalinga							+
Kandashaka							+

Palandu							+
Shushkashaka							+
Mamsa Varga							
Vallura		+					
Varaka		+					
Viharaja Nidana							
Vishamashana	+	+		+	+		
Atibhukta							+
Abhojana			+				
Langhana	+				+		
Adhovata rodha		+					+
Mutra rodha		+					+
Pureesha rodha		+					+
Ratri jagarana	+		+	+	+	+	+
Ati vyavaya	+	+	+	+	+		+
Ati vyayama	+	+	+	+	+		+
Ati adwa	+	+		+	+		+
Atibhashana			+				+
Ashwayana	+			+	+		+
Ushtrayana	+			+	+		+
Rathayana							+
Gajayana	+			+	+		
Plavana	+			+	+	+	
Aticheshta	+			+	+		
Vegadharana	+	+	+	+	+		
Sheegrayana	+			+	+		
Abhighata	+	+		+	+	+	
Shayyasana	+				+		
Manasika Nidana							
Chinta	+		+	+	+	+	
Bhaya	+						+
Dukha	+						

Krodha	+				+		
Shoka	+		+	+	+	+	
Panchakarma Apacharajanya							
Asamyak vamana					+		
Asamyak virechana					+		
Kriyati yoga			+				
Vishama upachara	+			+		+	
Kalaja							
Sheeta dine							+
Durdine							+
Aparahne							+
Varsha ritu					+		
Jara	+				+		
Greeshma ritu		+					
Ahoratri		+					
Nishante	+						
Divasante	+						
Varshante	+						
Anya Nidana							
Dhatukshaya	+			+	+	+	
Marmabhighata	+			+		+	

Adibala Pravrita vyadhi

Kampavata can also be considered under the heading of Adibala Pravrita vyadhi as Sushruta while explaining vatika kustha mentions kampa as a symptom, though Kampavata not mentioned directly as separate disease but can be justified on the basis of symptom in Adibala Pravrita vyadhi like vatika kusta. [69]In modern medicine, it has been assumed that genetic factors are important because at least 10 – 15% of cases have relatives similarly affected.

In recent years, geneticists have accumulated increasing evidence of genetic defects associated with the development of Parkinson's disease. However, such monogenetic links have been found in very few families, and in the majority of cases Parkinson's disease is not thought to be directly inherited. Nevertheless, it has been estimated that having a parent with Parkinson's disease increases the lifetime risk of developing Parkinson's disease from 2% to 6%.

Kalabala Pravrita

Vagbhata [70]observes tremor and flexion posture as symptoms of aging. Most of the patients develop Kampavata between the ages of 50 – 65. Age related aspects of this disease are also proved. 80% loss of striatal dopamine is required for symptoms to develop. It has been shown that an approximately 5 – 8% cell loss in the substantia nigra occur per decade of life, Suggesting that aging may increase vulnerability to the Parkinson's. In this way Ayurvedic concept of development of tremor in the aged due to the Kala or Svabhava Balapravrita is supported by even the recent researches.

Abhigataja

Charaka while explaining shiromarma abhigata lakshnas mentions symptoms like chestanasha, Spandana (means Kampana), swarahani etc.[71] Vaghabhat states that any trauma to marma will lead to vishama spandana which is abnormal movement.[72] Pranavaha Srotasa Marmaghata causes Vinamana, Bhramana and Vepana[73] repeated forceful blow to head as in boxing can cause numerous small haemorrhage throughout the brain, including basal nuclei, which in turn can cause tremor, rigidity and physical slowness. This is well recognized in punch – drunken boxers who over the course of years have prided themselves on their ability to absorb frequent, intense physical punishment with consequent irreversible brain damage. However, this punch-drunk condition provides some clue to Abhighata theory.

Aharata nidana (Dietetic cause)

Charaka while mentioning guna karma of rasas explained as katu rasa is vayu and agni pradhana excess use of katu rasa will lead to kampa.[74] Similarly Sushruta also mentioned excess usage of katu rasa causes kampa.[75] Even vaghabat mentions katu rasa causes kampa.[76] Excess intake of Kashaya rasa cause for sthambana explained by acharyas which is another symptom of kampavata. [77, 78, 79] Acharya Vaghbhat mentions as excess use of tikta rasa will lead to disorders of vata vyadhi.[80] Usage of ruksha and alpa ahara are causes of vataja disorders [81]ruksha dravya causing vitiation of prana.[82] In Bhela samhita its described that persons who constantly consume dry foods and also patient of udavartha the aggravated vayu gets excited and rises upwards causing for shira kampa.[83] Certain vitamins have been the subject of suspicion for an association with Parkinson's disease, especially an excess of vitamin E. However the evidence for this is so far poor. In contrast, it has been suggested that vitamin C decreases the risk of Parkinson's disease.[84] Recently it has been shown that prisoners of war who were inferred in Far East and suffered great physical and nutritional neglect, in their later life showed an increased vulnerability to develop certain chronic neurological diseases including Parkinsonism, but significance of this remains controversial.[85]Charaka explains suppression of jrumba (Yawning) causes Kampa (tremor), Pravepana (shaking), Vinama (flexion posture), Samkocha[86]even suppression of hikka (eructation) leads to vepathu.[87] udavartha is also attributed to cause shira kampa.[88]

Visha

Charaka while explaining the vishavega mentions vepathu as the symptom of second vega [89] poisoning due to mustaka produces stiffness of body (gatrastambha) and vepathu.[90] Vepathu is a long term complication of dushivisha.[91]From many years, the idea that exposure to environmental toxins such as certain pesticides may cause

Parkinson's disease has received much attention. Indeed, some studies have shown there is a significantly increased risk of Parkinson's disease in people with a higher exposure to pesticides, such as farmers. There is a higher incidence in people who drink water from wells even. MPTP (1-methyl-4-phenyl-1, 2, 3, 6-tetrahydropyridine) has clearly been shown to cause symptoms of Parkinson's disease. Neuronal loss in the substantia nigra was shown at post mortem in those exposed to MPTP. However, evidence confirming any link between the toxic effects of paraquat and the development of Parkinson's disease is lacking.[92]Parkinsonism is reported in chronic manganese intoxication and carbon monoxide poisoning. The drugs like phenothizine (chlorpromazine), butyrophenones (haloperidol), Rauwlfia alkaloids (reserpine), tetrabenzene, and procaine cause reversible Parkinsonism. These above mentioned can be included in Ayurvedic concept of Visha.

Dhumapana

Epidemiologic studies suggest that cigarette smoking is protective against development of PD. Cigarette smoking has been associated with a lower risk of PD in numerous prevalent case-control studies in a wide variety of populations, but whether this association is due to a biologic effect of tobacco or is the result of some other factor remains controversial. Smoking has a dose-response relationship with PD. Current heavy smokers had a lower risk than current light smokers and former heavy smokers who had recently quit had lower risk than those who had quit more than 20 years earlier. Assuming smoking is neuroprotective, one might expect it to delay the onset of PD and improve the course of the disease in people already affected. Hypothesis has yet to be proven.[93] Charaka mentions that Dhumapana prevents many diseases of head.

Vruddhapya

Acharya Vagbhata has explained kampa (tremor) and avanamana (flexed posture) as symptoms of aging.[95] Symptoms of Parkinson's disease most often first appear during a person's 50s or 60s. Age related aspects of this disease are also proved 80% loss of striatal dopamine is required for symptoms to develop. It has been shown that approximately 5-8% cell loss in the substantia nigra occur per decade of life. Tyrosine B-hydroxylase, the rate limiting enzyme for dopamine also diminishes with age, suggesting that aging may increase vulnerability to the Parkinson's disease. Dysfunctional antioxidative mechanisms are associated with old age, which suggests that both acceleration of age related changes in dopamine production may also be a factor in Parkinson's.[96]

Chinta and shoka

As Charaka states chinta and shoka cause vata prakopa leading to manifestation of disease.[97]The stress as crucial trigger for initiation of Disease and chronic anxiety might conceivably cause irreversible change to nerve cells in brain. High incidence of moral rigidity and mild obsession personality traits are observed in Parkinson's patients. High frequencies of depressive illness occurring many years before onset of Parkinson's have been reported in some studies.

Etiology for Parkinson's disease [98]

The cause of Parkinson's disease is unknown. Many researchers believe that combinations of several factors are involved in the development of Parkinson's. These factors include free radicals, accelerated aging, environmental toxins, and genetic predisposition.

It may be those *free radicals*—unstable and potentially damaging molecules that lack an electron—are involved in the degeneration of dopamine-producing cells. Free

radicals add an electron by reacting with nearby molecules in a process called oxidation. This process can damage nerve cells. A chemical called *antioxidants* normally protects cells from oxidative stress and damage. If antioxidative action fails to protect dopamine-producing nerve cells, these cells may be damaged, resulting in Parkinson's disease.

Dysfunctional antioxidative mechanisms are associated with older age, which suggests that the acceleration of *age-related changes* in dopamine production also may be a factor in Parkinson's.

Exposure to an *environmental toxin*, such as a pesticide, that inhibits dopamine production and produces free radicals and oxidative damage may be involved in Parkinson's disease development. In some cases, the use of certain drugs can produce Parkinsonian symptoms (called drug-induced Parkinsonism). These drugs include chlorpromazine and haloperidol, which are prescribed for psychiatric patients, and metoclopramide, which often is used to treat stomach disorders. Changing the medication or adjusting the dosage of the drug moderates or eliminates Parkinson's symptoms in many cases. The only consistent findings have been on increased incidence of drinking water from Well in childhood and low incidence of cigarette smoking has some protective effect or this could be merely reflect the premorbid personality of patients with Parkinson's disease.

Roughly one-fifth of Parkinson's disease patients have at least one relative with Parkinsonian symptoms, suggesting that a *genetic factor* may be involved in the disorder. Several genes that cause symptoms in younger patients have been identified. However, most cases are not thought to be caused by genetic factors alone.

Toxins for Substantia nigra [99]

MPTP (1methyl-4phenyl-1, 2, 3, 6 tetrahydropyridine) is converted by monoamine oxidase to its neurotoxic metabolite MPP^+ and this compound is taken up

actively by dopaminergic neurons it then binds to neuromelanin and selectively destroys the nigro striatal dopaminergic system.

For globus pallidus

- Carbon monoxide
- Carbon disulphide
- Cyanide
- Manganese

To summarise, all the samanya nidanas explained for vatavyadhi can be considered as dosha hetus which are responsible for vataprakopa, in turn leading to Kampavata. It is very difficult to analyse the exact role of these nidanas in the pathology of Parkinson's disease.

Vishishta nidanas like vruddhapya, visha and abhighata have a role in the causation of Kampavata (Parkinson's disease) which are again supported by views of allied science. Chinta, bhaya, shoka etc. manasika nidana definitely provoke the condition, rather than producing the disease.

POORVA ROOPA

Symptoms, which manifest themselves before the appearance of the disease, are known as Poorva roopa (Premonitory symptoms). Many times these Poorva roopa give clue about the forthcoming disease. Premonitory symptoms are of two type viz. 1) Samanya - General or nonspecific to the dosha and 2) Vishista - Specific to the Dosha.

Incited Dosha localized (Sthanasanshraya) in impaired Srotasa marks the beginning of disease and at that stages Poorva roopa are produced but Acharya Charaka has not mentioned specific Poorva roopa for Vatavyadhi. He has considered it to be Avyakta or unmanifested, [100] even though following may be considered as poorva roopa of kampavata,

- Angamardha (body ache),
- Disorientation (Moha)
- Forgetfulness (Smriti hani)
- Paresthesia (Supti)
- Anxiety (Udvega)
- Nervous ness (Avasada
- Tiredness (Klama)

As most of these symptoms are vataja in nature suggestive of vata pradhanata. The early symptoms of PD may be nonspecific, such as fatigue, reduced energy, joint stiffness (especially of the shoulder), muscles cramps, or vague sensory disturbances. Unilateral limb dystonia, especially in the foot, may accompany the sensation of limb stiffness. Dragging of one foot or tripping after walking a distance even in the absence of dystonia may occur. The patient may take longer time to perform daily activities. The eyes may feel dry, with a sensation of burning due to the reduced frequency of blinking, slower and more effortful handwriting, with smaller letters (micrographia) may occur. Patient may complain of hoarseness or a soft voice (hypophonia), especially after speaking for a while. Sleep disturbances are very common in early PD. Further early nonmotor manifestations include constipation seborrheic dermatitis, decreased perception of smell (hyposmia), and including bladder dysfunction.[101]As specific Poorva roopa are not explained for Kampavata, the premonitory symptoms told for Parkinson's disease can be considered here.

ROOPA

The manifestation (vyaktavastha) of signs and symptoms of disease is known as Roopa. Disease expresses itself in the form of laxanas only after the completion of dosha dushya sammurchana. The signs and symptoms produced in an individual as a

result of sequential changes in the disease process can be studied under the heading "Roopa".

Basavarajeeyam has explained the symptoms of Kampavata [102] as

- ❖ Karapadatale Kampa (i.e. tremor in hands and feet),
- ❖ Dehabhramana (Rombergism),
- ❖ Nidrabhanga (disturbed sleep) and
- ❖ Matiksheena (dementia).

Certain symptoms like

- ❖ Stambha (Rigidity)
- ❖ Cestahani (Slowness of the movement)
- ❖ Vinaman (Flexed posture)
- ❖ Vakvikriti (Speech disorders).

Are other symptoms mentioned in some pathological conditions of Vata vyadhi which can also be grouped under the features of Kampavata.

In modern science other than these above mentioned symptoms they have described as[103]

- Rigidity
- Bradykinesia
- Monotonous speech
- Postural instability
- Depression
- Hallucinations
- Dementia
- Nocturia
- Constipation

Though the above said symptoms are not explained in our classics in the context of Kampavata, they can be considered in this context as they are being observed in patients of Parkinson's disease.

Kampa – Tremor

Kampa is a cardinal symptom of Kampavata. Increased movements are denoted as Vepathu, Kampa, Spandana, Sphurana etc as mentioned in Ayurvedic texts. Vepathu or Kampa is enumerated in Vata nanatmaja vikara.[104] Kampana is Chalana (increased movement) of any part, Spandana is shaking of mild degree,[105] Sphurana is continuous or repeated shaking.[106] Kampa is also symptom of many diseases like Jarashosha,[107]Urustambha,[108]VatikaVisarpa,[109]Madatyaya,[110]VatikaJwara.[111]Madahvaka ra explained kampa as separate disease which manifested all over the body and head. Acharya Basavarajeeyam opines in kampavata the chief manifestations are karapadatale kampa (tremors in hands and legs) respectively.

To understand regarding tremors Acharya Charaka in *Kiyanthashirasiya adhaya* explains the cause for veapana in body due to diminution of kapha and pitta with increase of vata which causes trembling of body. Acharya Vaghbhat opines Kampa as a symptom of Provoked Vata. [112] As Vyana Vata is responsible for all the movements in the body, disturbance in the normal functioning will lead to kampa which signifies vyana vata vikruti will lead to Kampa, with Chala guna vriddhi of Vata is seen in increased movements.

Explanation of kampa depending upon the site is explained by acharyas. Charaka while explaining disease of head opines tremors in head is disorder caused due to vikruta vata.[113] Sushruta mentions as symptom of ardita [114] Bhela mentions shiro basti is best remedy for shirah kampa.[115] Hasta kampa is an another type of kampa where Charka has enlisted this in demerits of basti given with hasta kampana.

Pada kampana is mentioned by Acharya Bhavaprakash in the context of kalaya khanja where the person has tremors in his legs at the commencement of walking. [116] Kampa is even noted in disorders of snayu. [117,118] Tremor is a main symptom associated with Parkinson's disease. However, contrary to popular belief, it is not universal and approximately one-quarter of patients do not have tremor. The involuntary rhythmical shaking normally occurs at rest and tends to reduce or stop when the affected part is used for some activity, for example if the hand is reached out to take hold of something. However, sometimes the patient also has an 'action tremor', similar to that seen in patients with essential tremor. The tremor of Parkinson's disease is quite coarse with a frequency usually between 4 and 6 Hz. Although the hands are often affected, some patients experience tremor of the jaw or foot. The tremor affecting the thumb and first finger produces the commonly called 'pill rolling' effect.[119]

Stambha (Rigidity)

The term Stambha is originated from masculine gender and Stambha + Ach means to fix firmly/stiff [120] Stambha means to make stiff or rigid.[121] [i]Stambha is a sign of Snayu prapta Vata.[122] Charaka explains disorders of Snayu produce Stambha.[123] Hemadri noted Stambha as inactivity.[124]Acharya Dalhana has defined Stambha as immobility.[125] Disordered Snayu produce Stambha. Charaka explained Stambha under Vataja disorders and Stambha of different parts is observed by Charaka like Grivastambha, Manyastambha, urustambha[126] etc. Stambhana is also sign of Avarana of Vyana by Kapha.[127] It is also a sign of Sarvangatavata.[128] Avarana of Vyana by Udana causes Stabdata.[129]The Rasa like Kashaya produces Stambha.[130]Dushyas like Sira, Snayu are important in production of Stambha.[131] Rigidity is an important feature of Parkinson's disease rigidity is actually hyper tonicity of muscles. In rigidity muscles become continuously or intermittently firm and tense. Rigidity is detected clinically by

resistance to passive manipulation of the limbs, neck or trunk, the rigidity, or muscular stiffness occurring with Parkinson's disease exacerbates the problems with movement resulting from hypokinesia and bradykinesia. All muscle groups can become affected the increase in muscle resistance occurs when there is passive movement; the resistance to passive movement is constant throughout the range of movement, unlike spasticity where sudden relaxation can occur after movement has begun. If the patient also suffers with tremor, the so-called cog wheeling effect can be seen. This jerky movement results from the tremor superimposed on top of the rigidity. The rigidity associated with Parkinson's disease is also often asymmetrical at onset.

Chestasanga – Bradykinesia

As the word chesta signifies to move [132] and sanga means obstruction combining both words makes reduced movements or obstructed movements. Vyana vayu actually carries out all the movements. Disturbance in the function of Vyana vayu leads to Chestasanga. Bradykinesia is defined as slowness or poverty of movement with loss of automatic stereotyped movements. It is perceived by the patient as a slowing of his ability to perform the usual activities of daily living such as bathing, dressing, rising from chair, turning over in bed, making buttoning, shaving etc. Hesitation on initiation of movement and early fatigue are also features of bradykinesia. Bradykinesia accounts for many of the characteristic features of Parkinson's disease such as the expression less face (mask face), a decrease and subsequent loss of blinking, reduced arm swing while walking, small cramped hand writing (micrographia), monotonous speech, decrease in the frequency of swallowing (resulting in accumulation of saliva in the mouth and drooling) and loss of expressive gestures of hand.

Flexed Posture – Avanamana

Avanama means to bow, to bend down.[133]Acharya Vagbhata mentioned avanamana as a sign of aging [134] with Vata being the dominant dosha in old age and ruksha guna of Vata is particularly important in this respect. Prana is particularly important in maintenance of balance and posture. Patients with Parkinson's disease develop a characteristic flexed posture resulting especially from flexion of the knees and hands. The posture of Parkinson's disease patient involves flexion of the head, trunk and extremities. As the course of Parkinson's disease progresses, postural instability becomes a more troublesome feature. A steady posture is normally maintained by the nervous system making continuous reflex adjustments. Impairment of these mechanisms leads to a reduced ability to maintain balance, making the patient less steady when walking and particularly while turning

Gait Abnormalities – Gatisanga

The word 'Gati' is used for gait and related movements. The term 'Gati' originates from 'Gam' meaning to move, to go. [135] Gati is a function of vyana vayu. In Kampavata the functions of vyana vayu are impeded resulting in characteristic gatisanga. Avarana of Vyana, Udana and Prana by Kapha manifest with symptom of restricted movements.[136] Prana by supporting all sensory and motor organs, Udana by Prayatna Urja and Vyana by imparting five types of Cheshtas, play role in Gati. This function is assisted by mamsa dhatu and snayu and asti dhatu which maintains the erect posture,[137] when these vata ie prana,udana and vyana gets avarana by kapha respective functions gets impaired resulting in gatisanga .Gait symptoms are a common feature of Parkinson's disease, but usually occur around five years after initial diagnosis which is abnormal in nature. In addition to the postural abnormalities and loss of arm swing, the Parkinson's disease patient generally takes small shuffling steps, difficulty in beginning

to walk and to stop walking once started leads to the so called "festinating gait" or "hurrying gait" i.e. invincible propensity to run, when wishing only to walk. The forward leaning of trunk moves the centre of gravity forward, thus patient feels as if he is driven forward (propulsion). Patient are often unable to lift their feet off the floor as if they are stuck to it exhibiting another characteristic gait abnormality "start hesitation" or "freezing".

Monotonous Speech – Vak vikriti

Vak is the function of udana vayu. Rooksha guna of vata is responsible for obstructed low, dry, and broken voice. Disturbance in functions of Prana and Udana may interfere with fluency of speech. Avarana of Udana by Kapha manifests with symptom of vaksawara graha as mentioned by charaka.[138]There are many ways in which speech may be adversely affected by Parkinson's disease, decrease in muscle movement of the larynx can reduce the volume and articulation of speech making it difficult for others to understand what is being said. This is compounded by the tendency for phrases to be said in a rush, the patient being unable to control the speed of delivery. Sometimes, long silences occur as a patient has difficulty starting the beginning of a sentence or new phrase.

Dehabhramana – Postural Changes

Meaning of bhramana is chalanam.[139] Chalana is a term used synonymously for kampa. In vachaspatya bhramana is defined as Gatibheda i.e. type of movement. So here Dehabhramana can be considered as generalized tremor which may be seen in the later stage of the Parkinson's disease. Another meaning for bhramana is unsteadiness. Unsteadiness of the body may be seen in Parkinson's disease because of Postural instability.

Nidrabhanga – Insomnia

Here nidra is sleep and bhanga means disturbance. So Nidrabhanga defined as disturbed sleep. Disturbed sleep or Nidrabhanga is a symptom of aggravated Vata. Staying asleep or Insomnia can result from anxiety or depression. Nearly all patients with Parkinson's disease report various disturbances with sleep. In the majority of cases, problems are the result of limb movements, myoclonic jerks or leg cramps. Being unable to turn over in bed during the 'off' period, as well as the tremor associated with Parkinson's disease can also interfere with sleep.

Ksheenamati – Dementia

Buddhi determines the things by shifting out and distilling bad and good probabilities respectively. Buddhi takes rational decision by choosing right objectives Chakrapani [140] Pranavayu supports Buddhi [141] Udana is related with Dhi(Buddhi) and Manobhodhana.[142] Sadhaka pitta is responsible for activities of Buddhi, ahankara and utsaha.[143] Thus impairment in the functions of Pranavayu, Udana vayu and Sadhaka pitta can lead to Ksheenamati in the patients of Kampavata. Serious cognitive impairment occurs in about one-fifth of patients with advanced Parkinson's disease. Aspects of mental function such as reduced short-term memory, confusion, adverse effects on judgement and reasoning, and visual hallucinations are key features of dementia.[144]

Depression – vishada

Charaka mentions vishāda as one of the nanatmaja vikara of vata[145] tendency of increase in the strength of disease increase with vishadata in person.[146] Sadhaka Pitta is essential for the desired action Shukra Dhatu is concerned with harsha[147]or elation. Udana is related with functions of mind. Prana supports all Indriyas. Vyana defiles due to Vishada.[148]Thus derangements in the functions of Prana, Udana and Kshina Sadhaka

Pitta and impaired Dhatus like Rasas Shukra and Ojasa leads to Vishada. Nearly one-half of Parkinson's disease patients suffer with depression, and quality-of-life assessments have shown this symptom to be a major factor in reducing quality of life. Depression can be caused by chemical changes in the brain or it can be a reaction to having a disabling disease. Depression can further contribute to memory loss and confusion.

Impairment in Memory – Smritihani

Smriti is ability to remember the things already seen or heard or experienced.[149] Smriti is dominating function of udana vayu.[150] so smritihani is result of impairment in function of udana vayu. Some patients of Parkinson's disease suffer significantly with memory loss. The exact cause for this is not known.

Constipation – Vibandha

Dushti of apanavata leads to formation of vibandha in Kampavata. The main gastrointestinal symptom associated with Parkinson's disease is constipation, which affects a large proportion of patients. This is the result of reduced stool transit in the colon; Pelvic floor muscle dystonia may affect the rectum and anus, which, instead of relaxing when trying to pass a stool, goes into spasm. But it is usually attributed to the combined effects of sedentary life and diminished food and fluid intake.

Other Features of Parkinson's Disease

Dysphagia (difficulty in swallowing) is a common problem in up to one half of patients, especially those in the more advanced stages of Parkinson's disease. Micrographia, writing that is very small sometimes to the point of being unreadable, writing becomes smaller and smaller the longer the patient writes.

Nocturia (frequent urination at night) is often the first indication that Parkinson's disease is affecting the bladder. Excessive sweating, greasy skin,

seborrhoea of the scalp and fore head Sensory symptoms such as numbness, coldness, burning sensation and occasional pain are among the other features of Parkinson's disease.

Table no 2. Showing the Comparison between Kampavata and Parkinson's Disease

Lakshanas of Kampavata	**Symptoms of Parkinson's disease**
Kampa	Tremor
Matiksheena	Dementia
Vibandh	Constipation
Dehabhramana	Postural changes
Nidrabhanga	Insomnia

Lakshanas of Kampavata explained in the classics such as kampa, dehabhramana, nidrabhanga and matiksheena are very vague and are also seen in many other neurological disorders other than Parkinson's disease. So it is very difficult to diagnose the Parkinson's disease very accurately only with these lakshanas.

Diagnosis of Parkinson's disease is based on clinical features; there are no objective parameters like laboratory findings that can confirm the diagnosis.

So the symptoms explained in allied science for Parkinson's disease can also be considered under the heading of roopa for better understanding.

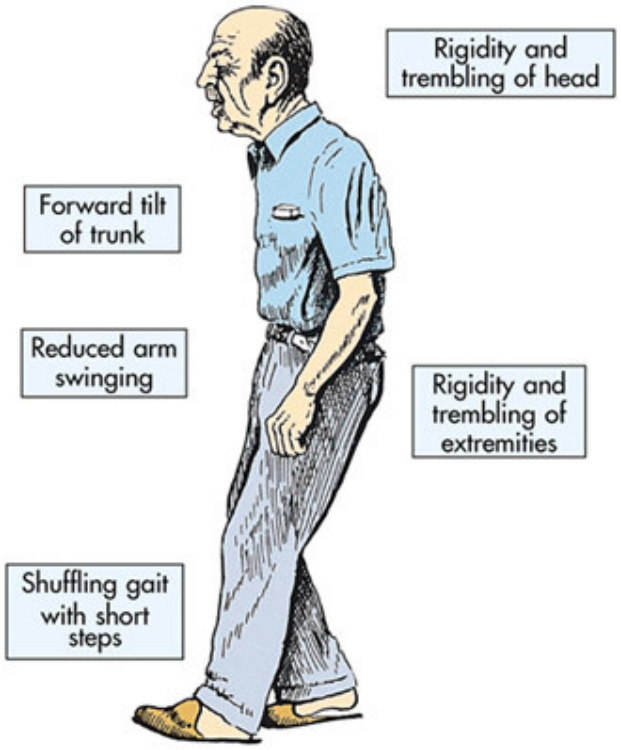


Figure No 2 Showing Picture of Parkinson's disease patient

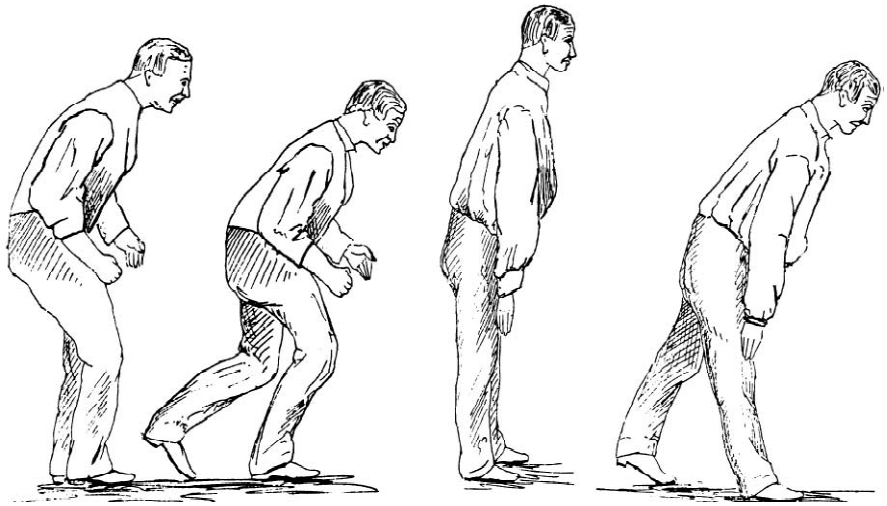


Figure No 3. Showing Short steeping gates in Parkinson's disease

Figure No 04. Showing Flexed posture in Parkinson's disease

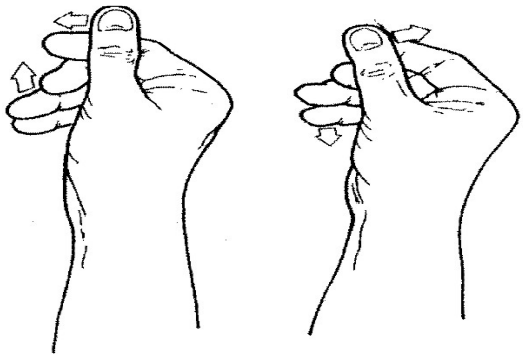


Figure No 05. Showing Pill rolling posture of hand

UPASHAYA AND ANUPASHAYA

Specific references regarding upashaya and anupashaya of Kampavata in classics are not found. So, all the factors that aggravate vata can be considered as anupashaya and that which pacifies vata can be considered as upashaya.

SAMPRAPTI

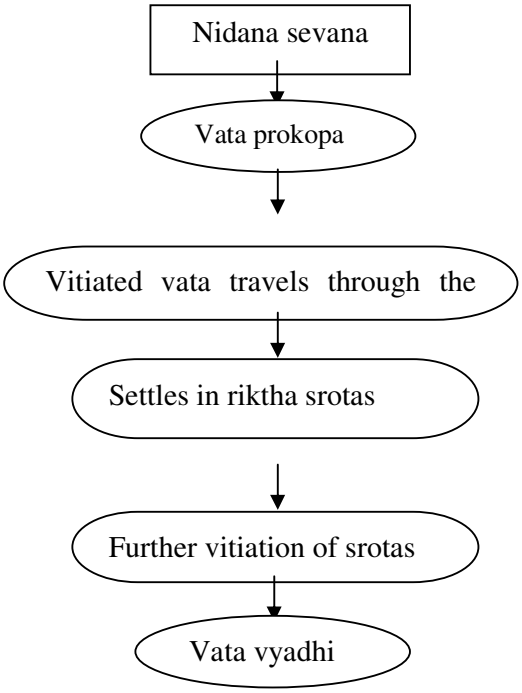
Samprapti' means the complete procedure of manifestation of diseases. Samprapti is most vital in understanding of the disease and its management as goal of therapy is to contravene samprapti. Because samprapti explains the development of morbid conditions more specifically the doshic events and the reaction of other pathologic factors occurring in the disease.

Samprapti of vatavyadhi is complex process to understand. Acharya Madhava while stating the 'vatavyadhi' has explained "*vikruta vatajanito asadharano vyadhihi vatavyadhi,*"[151] vatavyadhi is manifested due to vikruta vata and is asadharana in nature.

Though Kampa is mentioned under the heading of vataja nanatmaja vikaras, samprapti for Kampavata is not explained separately, so the general samprapti of vatavyadhi can be considered here.

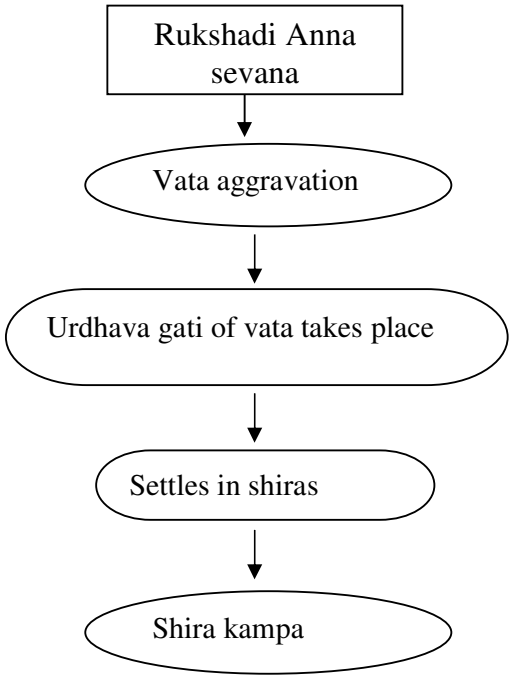
Illustration No.1 Samanya Samprapti of vatavyadhi

According to Acharya Charaka and Acharya Vagbhata



Acharya Bhela has mentioned regarding shirakampa with its samprapati and nidanas [152]

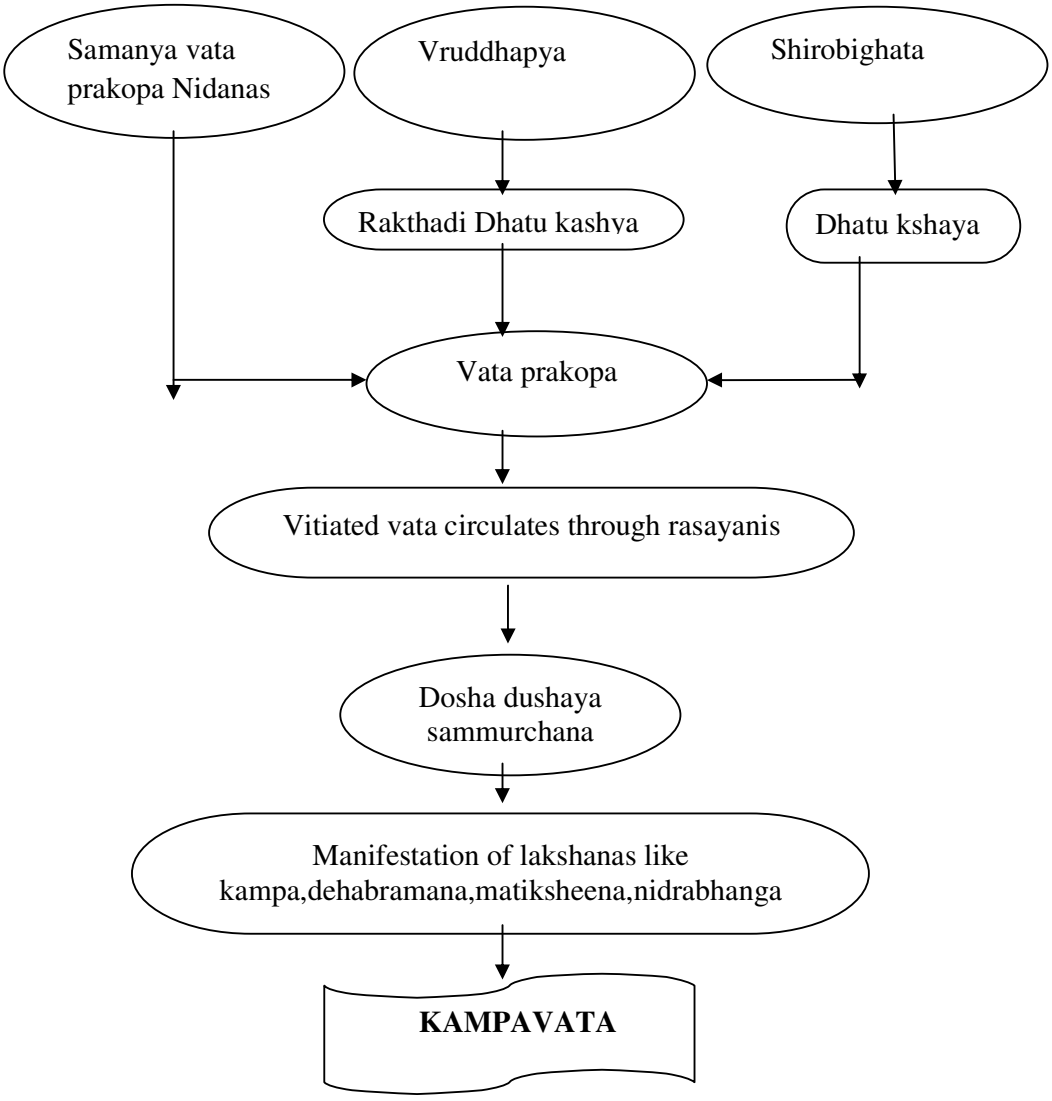
Illustration No.2 Showing the Samprapti of Shirakampa



Vruddhavastha contributes for Dhatukshaya which is the prime cause for Vata prakopa and in turn responsible for the manifestation of Kampavata. Abhighata to Shiras leads to raktadidhatu Kshaya in turn leading to Vata Prakopa. Acharya Vagbhata has mentioned Kampa and Avanamana as symptoms of Vruddhavastha.[153]

Due to the etiological factors, as mentioned the Vata gets aggravated by its chala, ruksha and sheeta properties. Prana, Udana and Vyana are most affected among the five types of vata, which in turn vitiate the Mastulunga Majja in the Shiras, because of Srotovaigunya present at Shiras. As Mastishka is Sneha pradhana, vitiated Vayu impairs this Sneha by its ruksha, sheeta and laghu guna. These vitiated doshas selectively affect the Vata vaha srotas in the Mastishka leading to impairment in the motor functions of the body leading to Kampa, Dehabhramana, Matiksheena etc.

Illustration No.3 Showing the Samprapti of Kampavata



Samprapti Ghataka

Dosha	:	Vata (Prana, Udana, Vyana)
Dushya	:	Mastulunga majja, Snayu
Srotas	:	Vatavaha
Srotodushti	:	Atipravritti
Udbhavasthana	:	Pakvashaya
Adhishtana	:	Mastishka

Sancharasthana	:	Rasayani
Vyaktasthana	:	Sarvashareera
Vyadhi Marga	:	Madhyama .

Patho Physiology of Parkinson's disease

Hallmarks of the Pathology of Parkinson's disease are [154]

- Degeneration of Substantia nigra.
- Loss of at least 60% of dopaminergic neurons.
- Presence of lewy bodies in surviving neurons of the Substantia nigra.

Parkinson's disease results from degeneration of the dopaminergic pathway from the substantia nigra to the corpus striatum. Voluntary movement is controlled by the basal ganglia, which are a group of sub cortical nuclei consisting of the:

- Striatum (caudate and putamen)
- Globus pallidus (externa and interna)
- Substantia nigra (pars compacter and reticularis)
- subthalamic nucleus.

The essential lesion is one of an idiopathic degeneration of the pigmented neurons of the brain stem seen most convincingly in the substantia nigra resulting in depigmentation of these brain stem nuclei. Thus the projection of the nigral neurons to the corpus striatum is destroyed so that their modifying influence on this corpus striatum resulting in the clinical picture of rigidity and tremor.

Control of voluntary motor activity is the main function of basal ganglia. The movements during voluntary motor activity are initiated by cerebral cortex further these movements are controlled by ***basal ganglia***. During lesions of basal ganglia the controlling mechanisms are lost and so movements become inaccurate and awkward. ***Control of reflex muscular activities***, particularly visual reflexes are important in the

maintenance of posture the co-ordination and integration of such impulses for these activities depend upon basal ganglia which will be disturbed in lesions of basal ganglia.

Control of muscle tone**,** the gamma motor neuron of spinal cord is responsible for maintaining the tone of muscles actually the tone is depended upon muscle spindle fibers, Basal ganglia especially the substantia nigra controls the gamma motor neurons and muscle spindle fibers lesions of this area will lead to increased tonicity leading to rigidity.

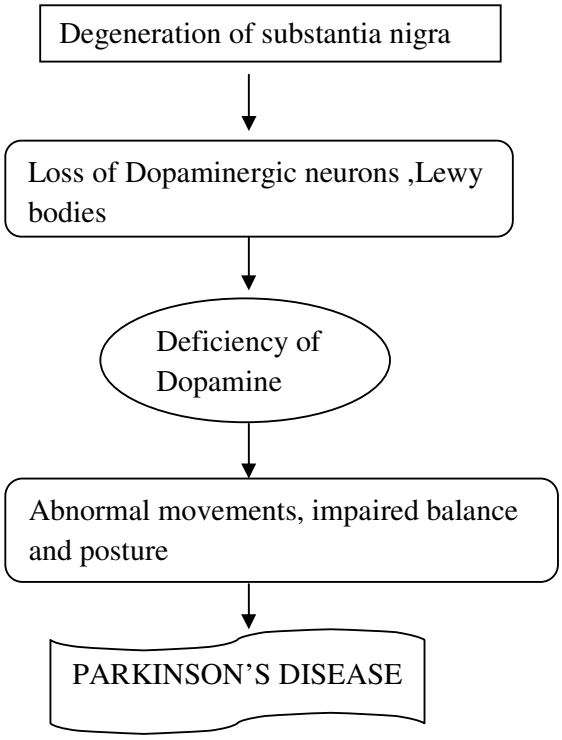
Control of automatic associated movements**,** swinging of arms during walking, appropriate facial expressions and other movements associated with motor activities are called automatic associated movements, thus lesions of basal ganglia cause absence of these movements resulting in poverty of movements, face without appropriate expression i.e. mask like face .

This Pathology is seen as depigmentation of the Substantia nigra in the sections of the midbrain of Parkinson's disease patients. The loss of these neurons causes deficiency of dopamine (neuro transmitter) in the striatum, which is essential for the normal nigral striatal function. It is important to appreciate that the cells of Substantia nigra normally decline with age but patients with Parkinson's disease have an accelerated loss for unknown reasons.

Lewy-bodies

Original description is given by Lewy in 1913, hence the name. Lewy bodies are eosinophilic, intracytoplasmic circular or oval structures. They are made up of granular and filamentous materials they are often multiple and located in the cell body of the affected neurons. Lewy bodies have been invariably recognized as a major pathological feature of Parkinson's disease A diagnosis of Parkinson's disease is rarely confirmed without the presence of Lewy bodies in the substabtia nigra.

Illustration No.4 Showing the Pathology of Parkinson's disease



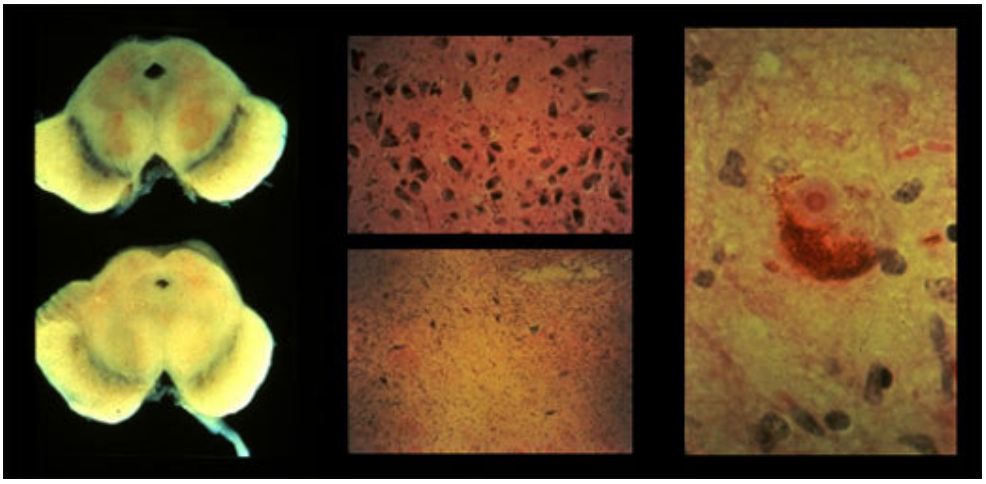


Figure No 06: Substantia nigra and Lewy bodies in Parkinson's disease patient

Sapeksha Nidana

It is necessary to differentiate a disease from others, having similar signs and symptoms which help for the pin point diagnosis and treatment. Kampavata is differentiated from snayugata vata on the basis of lakshanas.

Table No. 3. Showing the Sapeksha Nidana and Vyavachedakata with Kampavata

Kampavata	**Snayugata vata**
Kampa	Kampa
Stambha	Stambha
Dehabhramana	---
Matiksheena	---
Nidrabhanga	---
---	Shoola
---	Akshepa

Table No. 4. Showing the Differential Diagnosis of Parkinson's Disease

Disease	History	Physical Examination	Work up
Idiopathic Parkinson's disease	Gradual onset tremor gait disturbance, slowed movements	Resting tremor (affects limbs more than head) cogwheel rigidity (affects limbs more than neck or spine)	Physical examination is best
Drug induced Parkinsonism	Previous exposure to a drug such as metoclopramide or haloperidol	Similar to idiopathic Parkinson's disease	History and physical examination are best
Essential tremor	Present for many years, positive family history	Tremor with arms raised (postural), head involved	Physical examination is best

Multisystem atrophy	Parkinsonism with autonomic system dysfunction, dysarthria	Orthostatic hypotension skin changes, eg. Seborrhea	Physical examination is best
Progressive supranuclear palsy	Difficulty in reading or driving, stiffness bradykinesia, cognitive or behavioural changes	Gaze palsy (down more than up), axial rigidity (affects neck and spine more than legs)	Physical examination is best
Huntington's disease	Involuntary movements cognitive or behavioral problems	Chorea, loose tone, early dementia	CT or MRI studies to measure caudate nuclei
Normal pressure hydrocephalus	Urinary incontinence memory or cognitive problems	Dementia with frontal lobe features, festinating gait	CT or MRI studies lumbar puncture
Post traumatic Parkinsonism	Repeated head trauma (eg. In boxers)	Bradykinesia evidence of previous trauma	CT or MRI head

Investigations

There are no laboratory biomarkers for Parkinson's disease. Serum ceruloplasmin concentration is obtained as screening test for Wilson's disease. It should be obtained in patient who present with Parkinsonian symptom under age of 40 yrs. As yet, there is no simple test that enables confirmation of an accurate diagnosis of Parkinson's disease. However, a number of techniques and forms of brain imaging may be used, mainly in specialist centres to assist in diagnosis. Imaging techniques have been developed over the years and have contributed much to the current knowledge of the pathophysiology of Parkinson's disease. As research tools they have been valuable and on occasions provide useful techniques for helping with the task of diagnosis.

Computerised tomography (CT) scans appear normal in Parkinson's disease, but may show areas of atrophy in MSA (multi system atrophy). The main value of

performing a CT scan is in excluding other conditions such as hydrocephalus, or small strokes as evidenced by areas of tissue damage.

Magnetic resonance imaging (MRI) scans have a higher resolution and can be valuable in assisting the diagnosis of MSA.

Positron emission tomography (PET) scans are able to give an idea of cell functioning, whereas CT and MRI scans show structural changes that may be present in the brain. PET scans enable uptake of dopamine by the dopaminergic neurons of the nigrostriatal pathway to be measured. A positron-emitting radioactive marker is administered to the patient, such as 18F-6-fluorodopa (18F-dopa). When taken up by presynaptic dopaminergic neurons in the caudate and putamen (corpus striatum) it is metabolised to 18F-dopamine. Emission of positrons by the isotope enables tissue concentrations to be measured. At present, PET scans are mainly used in research and at specialist Centres.

Single photon emission computed tomography (SPECT) scans (also known as Dat scans) are cheaper to carry out and are more readily available in hospitals. Derivatives of cocaine, ^{123I}-β-CIT and ^{123}I-FP-CIT, are most frequently used with gamma-ray-emitting isotope SPECT. These target presynaptic dopamine reuptake sites. The gamma-ray-emitting isotope enables visualisation of uptake in the caudate and putamen, which is reduced in Parkinson's disease.

Diagnosis

Unlike the diagnosis of many diseases, to conclude a patient has Parkinson's disease is not normally the result of assessing specific diagnostic tests. More often the diagnosis is determined from clinical observations, the presenting signs and symptoms, and to some extent the history as outlined on the previous page. Nevertheless, it can be difficult to distinguish idiopathic Parkinson's disease from certain other neurological

conditions. The main difficulty in the diagnosis is to distinguish Parkinson's disease from many Parkinsonian syndromes caused by other degenerative disease and some by medication or toxin. Parkinson's disease is fore more common than any of the syndrome that emulates it. Bradykinesia and rigidity of limbs and axial musculature are shared symptom. But only in Parkinson's disease is resting tremor an early sign and it remaining prominent even late in the illness. *Essential tremor* is very common; with prevalence ten times that of Parkinson's disease. Despite this, many patients with essential tremor are wrongly given the diagnosis of Parkinson's disease. The characteristics of the two conditions are different. Tremor associated with Parkinson's disease mainly occurs at rest and diminishes or stops during an action. In contrast, essential tremor occurs when performing an action .*Drug-induced Parkinsonism* can be produced by a number of medications, unlike Parkinson's disease both sides of body are usually affected equally, and the progression of symptoms is much more rapid. Parkinson's plus syndromes are a group of conditions that have a presentation very similar to Parkinson disease, often making it impossible to differentiate them from Parkinson's disease some of them are multiple system atrophy, striatonigral degeneration, corticobasal degeneration and dementia with Lewy Bodies.

Diagnosis of a Parkinsonian syndrome can be made if one adheres to strict definition of Parkinson's disease bradykinesia and at least one of the following, muscular rigidity, rest tremor postural instability followed by Supportive criteria for Parkinson's disease like unilateral onset, rest tremor present, progressive disorder, persistent asymmetry affecting the side of onset most and excellent response to levodopa.[155]

SADHYASADHYATA

After diagnosing a disease with the help of nidana panchaka, before starting the treatment, it is necessary to consider the prognosis, which helps in adopting the proper treatment. Most of our acharyas consider shuddha vataja vyadhi are asadhya or krichrasadhya. So Kampavata being one of the shuddha vata vyadhi, is also krichrasadhya / asadhya for chikitsa.

Parkinson's disease is a progressive disorder but its rate of progression is variable. The exact prognosis for an individual patient is difficult to predict precisely. Many factors may influence the prognosis of Parkinson's disease, such as age of onset, early clinical pattern and response to the treatment.

Hoehn and Yahr clinical rating scale[156]

This scale is mainly used to indicate the stage of a patient's disease based on the severity of the symptoms they are experiencing

Stage Severity

Stage 1.0 - Tremor or rigidity on one side of the body only (with or without bradykinesia)

Stage 1.5 - Tremor or rigidity on one side of the body and axially (with or without bradykinesia)

Stage 2.0 - Moderate tremor or rigidity on both sides of the body with bradykinesia

Stage 2.5 - Moderate tremor or rigidity on both sides of the body with bradykinesia, but recovery on retropulsion

Stage 3.0 - Significant tremor or rigidity on both sides of the body with bradykinesia and some postural instability

Stage 4.0 - Severe disability, but still able to stand and walk without assistance

Stage 5.0 - Bedridden or wheelchair bound unless assisted (patient unable to function independently.[157]

By this staging we can infer that first 2 stages are curable. 3rd and 4th stages are difficult to cure, where as the last stage is incurable.

Thus Kampavata in the very initial stage will become Sadhya and is Krichrasadhya or asadhya in later stages.

CHIKITSA

In Ayurveda Aushadha is considered as one of the four fold constituents of chikitsa –chatushpada and which has been placed next to the physician. The drug is 'an agent' which a physician employs as an instrument in restoring the equilibrium of the body tissues. In modern ages WHO stresses importance of drug and defines it as a substance or product that is used or intended to be used to modify or explore physiological system or pathological status for the benefit of the recipient. Acahrya Charaka has further amplified the scope of Chikitsa by saying, chikitsa not only aims at the radical removal of causative factors of the disease but aims at the restoration of doshic equilibrium.[158]

Kampavata being one of the Vatavyadhi, general line of treatment which is explained for vatavyadhi can be adopted by considering specific etiology.

For better understanding these principles of treatment are explained under 3 headings.

- Shodhana
- Shamana
- Other Measures

Acharya Vangasena has described specific therapies for the treatment of Kampavata. [159] These specific line of treatment for Kampavata can be summarized here.

- ❖ Abhyanga
- ❖ Swedana
- ❖ Virechana
- ❖ Anuvasana basti
- ❖ Niruha basti
- ❖ Shirobasti

Snehana

- Sneha can be administered in both ways – External and Internal
- Abhyantara Snehas are – Bhojana, Pana, Nasya and Basti
- Bahya Sneha are – Abhyanga, Mardana, Lepa, Moordhni taila etc…

Charaka opines, Snehana is the first line of treatment for all Vatavayadhis, Snehana does Balavardhana, Agnivardhana and nourishes shushka dhatus.

Abhyanga

Abhyanga means to do some gati or movement. Abhyanga ensures softness and unctuousness of skin. Along with different snehas the veerya of sneha will reach uttarottara dhatus and gives the desired effect. Abhyanga done reglurarly clears the srotas, removes tiredness and body pain, builds up stamina, increases blood circulation, prevents old age. It induces sleep and Improves eye sight and complexion of the skin.[160]

Swedana

Swedana is a process by which sweat or perspiration of the body is produced. It cures stambha, guruta and sheetata.[161] Swedana is useful in the conditions like sankocha, āyama, shula, stambha.[162] In the context of vatavyadhi swedanakarma like nadisweda, pindasweda, awagahasweda are mentioned which alleviate vitiated vata dosha.

Virechana

Acharya Charaka mentioned Virechana as a shodhana karma for the treatment of Vatavyadhis. Especially for Kampavata no drugs have been mentioned. Mridu virechana with snehasamyukta oushadhis is advised in all sorts of Vatavyadhis.[163] For Sneha Virechana Eranda taila can be given with milk.[164] Virechana gives bala to indriyas, does agnideepana and koshtashuddhi.

Nasya

Administration of either medicine or medicated oil through the nose is known as Nasya Karma [165]Acharya Vagbhata has stated *"nasa hi shiraso dwaram"* i.e. nose is the easiest and closest opening for conveying the potency of medicines to the cranial cavity. The drugs administered will reach the shringataka marma and spread through the opening of siras of eye, ear and throat etc., and to the head. Acharya Chakradatta and Acharya Vangasena have indicated Nasya karma in Kampavata.

Bastikarma

All of our Acharyas have considered Basti as best treatment for Vatavyadhi. Due to its vast action, it has been considered as the complete or half of the treatment.[166] The appropriate adoption of Bastikarma facilitates expulsion of pureesha, shleshma, pitta, adhovata and mootra and accomplishes strength.[167]

The snehas which are used for Anuvasana basti destroys the rooksha, laghu and sheeta gunas of Vata, by their snigdha, guru and ushna properties. Niruha basti causes elimination of malas and doshas from all the srotases. Basti relives sankocha and stabdhata etc… Basti can be administered in all the age

Shamana Chikitsa

Some of shamana yogas are described for Kampavata are as follows.

- Nakula taila (B.R)
- Vijaya bhairava taila (B.R)
- Rasna taila (Bh. S)
- Sahacharadi taila (S.Y)
- Ksheerabala taila (A.H)
- Varuni taila (Sha. S)
- Dhatturadi taila (Sha. S)

- ❖ Mahanarayana taila (B.P)
- ❖ Nakuladhya ghrita (B.R)
- ❖ Brihat chagaladi ghrita (B.R)
- ❖ Mashataila (Ba. Ra)
- ❖ Brihanmasha taila (Va. Se)
- ❖ Mahamasha taila (B.R)
- ❖ Karpasa taila (B.R)
- ❖ **Rasayogas**
- ❖ Triguna rasa (S.Y)
- ❖ Vatarakshasa rasa (R.Y.S. II)
- ❖ Kanaka sundara rasa (R.Y.S. I)
- ❖ Kalavidhanasano rasa (R.Y.S. I)
- ❖ Gandharva rasa (R.Y.S. I)
- ❖ Chaturbhuja rasa (R.Y.S. I)
- ❖ Nagarjuna vati (R.Y.S. I)
- ❖ Lakshmivilasa rasa (R.Y.S. II)

Treatment of Parkinson's disease [168]

There is currently no form of pharmacotherapy available that has shown to delay the progression of Parkinson's disease. However, there exists a range of drugs that can treat the symptoms of the condition and consequently improve the patient's quality of life. Managing drug therapy in patients with Parkinson's disease can be complex. Although good control is often achieved in the early stages of the disease, as it progresses the drugs usually need careful tailoring with respect to choice of agents and combinations used, and dosage adjustments.

In the very early stages of Parkinson's disease, when functional disability is minimal, the use of anti-Parkinson's drugs is often unnecessary and in fact the potential side-effects may be more of a problem than the condition itself. Once symptoms warrant treatment, this is usually initiated with levodopa combined with a peripheral dopa decarboxylase inhibitor (benserazide or carbidopa), or a dopamine agonist. Levodopa therapy is certainly the most effective. Other drugs used much less frequently as treatments for the early stages of Parkinson's disease include amantadine and anticholinergics. These should not be regarded as first-line forms of therapy, though anticholinergics and beta-blockers may very occasionally be suitable in patients with early-stage disease when tremor is the main feature. Oral administration of levodopa producing widely fluctuating levels of dopamine in the patient, unlike the continuous supply of dopamine that occurs naturally in the healthy brain. Levodopa is primarily absorbed from the small intestine and it is likely that variable gastric emptying rates are the major cause of these fluctuating levels. Various studies have shown there to be a direct correlation between the variation in plasma levels and motor symptoms suffered by patients. After many years of research a gel of co-careldopa has been formulated specifically designed for continuous intraduodenal infusion via an external pump.

Attempts to alleviate the symptoms of Parkinson's disease with surgery date back to the 1940s. This was 20 years or so prior to the introduction of therapy with levodopa, so an effective treatment was anxiously sought. However, the beneficial effects of these early surgical procedures were negligible and were associated with a high rate of mortality and substantial morbidity.

The primary loop in the brain responsible for controlling movement runs from the premotor cortex to the striatum, then on to the globus pallidus and from there to the thalamus, which in turn feeds back to the motor cortex. Activity in this loop is

influenced by one mechanism acting as an accelerator (the substantia nigra), and another acting like a brake (the subthalamic nucleus). In Parkinson's disease, degeneration of the nigrostriatal pathway effectively means the accelerator to the loop is not functioning. This situation can be counteracted and some balance regained in the loop, by reducing the activity of the pathway from the subthalamic nucleus to the globus pallidus (i.e. reducing the braking mechanism). Alternatively, activity in the part of the loop running from the globus pallidus to the thalamus could be reduced, producing a similar effect. In Parkinson's disease there are therefore three areas of over activity in the brain which result in inappropriate activity from the thalamus to the motor cortex slowing the patient down and producing the characteristic symptoms of the disease the subthalamic nucleus the globus pallidus and the thalamus. These three sites therefore form the targets for surgical inactivation. There are two ways in which this inactivation can be achieved. Firstly by lesioning which is achieved by inserting a probe through a burr hole in the skull to one of the sites cited above, and passing an electrical current though the tip which then heats up and destroys the tissue in contact with the end of the probe. In line with the three areas of the brain listed above, this procedure is known as:

- Subthalamotomy
- Pallidotomy
- Thalamotomy.

Another technique is to place a permanent electrode in one of the above areas of the brain and connect this to an electrical stimulator similar to a pacemaker. High-frequency electrical impulses are delivered to the electrode in order to overstimulate the site where the tip is. This causes a depolarising block and thereby reduces conduction in the pathway. In line with the three areas of the brain listed above, this procedure is known as:

- Subthalamic stimulation
- Pallidal stimulation
- Thalamic stimulation.

Other Measures

These modalities include;

Education - Education about disease, its management and progression to patient and their family.

Support - There are many support organizations like American Parkinson's Disease Association, National Parkinson's Foundation, and Local Hospitals etc. Parkinson's disease patients can take help of these organizations by discussing their problems and struggles through the internet.

Exercise – Exercise is an important factor in the medical and psychological well being of patients. Exercise increases the patients overall health and functionality. It has a positive impact on mood and energy levels. All these factors are important in treating chronic disease. Gains from physical therapy only last as long as exercise is maintained, so continuation is essential.

Nutrition – Patients with Parkinson's disease have decreased muscle mass and more weight loss than healthy control subjects, patients should be instructed to eat healthy diet and to take multiple vitamins with calcium if needed.

Yoga and Meditation

Yoga and meditation help to build the resistance and immunity in the body. They also help in the regulation and balance functioning of central nervous system.

PATHYAPATHYA

That which is congenial to the srotas or body is called as pathya, contrary to these are considered as apathya.[169] Pathyapathya play important role in multifunctional effects by helping in avoidance of nidanas relieving the khavaigunya and checking the pathogenesis. There are no particular pathya and apathyas mentioned for Kampavata. Hence pathyas and apathyas of general vatavyadhi can be considered.

Table No.5 Showing the Pathya and Apathyas mentioned for Vatavyadhi

Ahara	Pathya	Apathya
Rasa Varga	Madhura, Amla, Lavana	Katu, Tikta, Kashaya
Shooka dhanya	Godhuma, Raktashali, Shashtikashali	Chanaka, Tarunadhanya
Shimbhi dhanya	Kulatha	Nishpava, Mudga, Kalaya Masha
Harita shakha	Patola Shigru Vartaka	--
Phala	Draksha Pakvamra Jambeera Dadima	Jambu Udumbara Tinduka
Mamsa Varga	Chataka Kukkuta Tittira	All jangala mamsa varga
Jala varga	Ushnajala Shrutasheetajala	Sheetambu Tadajala
Ksheera varga	Godugda Ajadugda Dadhi	Gardhabha
Sneha varga	Sarpi	---

	Vasa Taila Majja	
Mootra varga	Gomutra	
Madhya varga	Dhanyamla Sura	
Vihara	Snana Sambhavana etc.	Shrama Vyayama Jagarana Vegadharana Chankramana
Manasika	Sukha	Chinta Shoka Bhaya
Chikitsa	Snehana Svedana Abhyanga Brimhana Avagaha Shirobasti Tailadroni Nasya Avagaha Agnikarma Upanaha	Vamana Raktamokshana

Drug review

Oral administration of Triguna rasa in arohana and avarohana karma for 41 days indicated directly for kampavata in Sahsrayogam.

Composition of Triguna [170] rasa is as follows. The main ingredients of this yoga are Parada, Gandhaka and Haritaki.

Table no. 6 Showing composition of Triguna rasa

S.N	SANSKIRT NAME	SCIENTIFIC NAME	PROPORTION
1	Parada	Mercury	1 part
2	Gandhaka	Sulphur	8 part
3	Haritaki	Terminalia chebula	9 part

Parada

Parada is the most important and foremost ingredient of compounds of Rasashastra, without which the science of Alchemy – Rasashastra perhaps would not have existed.

Rasa Panchaka of Parad.[171]

Rasa – Shadrasa Veerya – Ushna

Guna – Snigdha, sara Vipaka – Madhura

Karma – Yogavahi, Rasayana, ativrishya, balya, vajkara, vayastambhakara, dehasiddhakara, lohasiddhakara, khe-gatiprada, purushartha chatushtaya- prada, Ayush-kara, Bhukti-mukti-prada, dristi-bala-prada, krimighna, Ropana, Shodhan, Agni-vardhak, Pushtikara etc.

Doshaghnata – Tridoshahara,

Rogaghnata – Tapatrya janyaroga, papaja roga, krimi, vataroga, Akshiroga, sarvarogahara especially ‘sarvakushta nut

Gandhaka

The importance of Gandhaka is due to the basic concept that Parada only with Gandhak becomes highly potent and gains qualities to destroy diseases, old age and death, which is the main purpose of Rasashastra.

Rasapanchaka.[172]

Rasa – Katu	Vipaka – Katu
Guna – Teekshna	Veerya – Ushna

Karma of Gandhak –

Karma – Agni-deepak, Ama-pachaka, Kleda-shoshaka, Visha-nashak, soota-veerya-prada, Ati-rasayan etc.

Doshaghnata – Vata and Kapha-hara, Pitta-vardhaka

Rogaghnata – Kushtaghna, Kandughna, visarpa-hara, Dadru-nashana, Krimi-hara etc.

Sulphur is essential for life. It is the constituent of all proteins. The sulphur content of the average adult human body is about 100 mg. Most of the sulphur present in human body is in the form of three amino acids namely cysteine, cystine and methionine. The rest is in the form of sulphates attached to other substances in body cells. Besides being a constituent of protein, sulphur is involved in the formation of bile acids, which are important for fat digestion and absorption. Sulphur is a component of Vit. B, thiamine and biotin. It plays a part in the reactions that help cells utilize oxygen. The presence of sulphur in human body is also necessary for blood clotting, the functioning of several enzymes, production of hormones and insulin, and formation of blood. Sulphates are important in detoxification mechanisms in the body.

Kajjali

Kajjali is the basic compound of all Rasaushadhis. Kajjali is a type of moorchita parada. Shuddha parad and Shuddha Gandhak or any metal are triturated continuously without adding any liquid till a nischandra (lusterless) black coloured fine powder is obtained. [173]

In Bhavaprakasha,[174] the time of triturating kajjali is told as, till it attains the nishchandra and till black coloured powder is formed i.e. till parada is not seen it must be triturated, which means that until Parada is mixed completely with Gandhak.

Proportions of Shuddha Parada and Shuddha Gandhak is in either of the following ratios [175]_

1. ½: 1 (Ardha)
2. 1:1 (Sama)
3. 1:2 (Dwiguna)
4. 1:4 (Chaturguna)
5. 1:6 (Shadguna)
6. 1:8 (Asta guna)

Kajjali is sagandha, niragni kharaleeya moorchana of parad. Among the 26 bandhas of Parad, Kajjali bandha is told [176] Kajjali when mixed with any formulation it enhances the action of that formulation[177]

Doshaghnata[178]– Tridosha-hara.

Karma and rogaghnata[179]: Kajjali given with different herbal formulations and with different anupanas is sarvamayahara. It is Brimhana, Veerya-vardhaka, virechaka.

Dosage of Kajjali is not mentioned in the Rasashastra texts. Since it forms the basis for Rasa preparations its proportions vary accordingly. Only one reference of single use of kajjali is available in vidradhi chikitsa. The dosage mentioned is one masha with varunadi quatha as anupana. [180]

Haritaki

It is one of the major drugs among alternative or rejuvenations (rasayan) medicines and frequently employed in a large number of formulations. Rejuvenates the body, Increases the bulk of the body. Nourishes the body up to the tissue level Scrapes out the unnecessary deposits and toxins out of the body. Haritaki gives different results according to the mode of taking i.e. when chewed it ignites the digestive fire, when taken after making a paste it ensures the timely evacuation of the feces and when taken after roasting it balances all the three Doshas. **Haritaki** is the drug of choice for rejuvenation for all the seasons since it is called as the "***Ritu Haritaki***". In rainy season it should be taken with *Saindhava* (Rock salt), in autumn with sugar, in early winter with *Shunthi*, in late winter with *Pippali*, in spring season with honey and in summer season it should be taken with *Guda* (Jaggery).

Table no 7 showing the composition of Haritaki

Dravya	**Haritaki**
Kula	Haritaki
Paryaya	Abhaya, Pathya
Latin name	Terminalia chebula
Family	Combretaceae
Utpatti Sthana	Throughout India Especially in Himalayan regions, Assam and Bangal.
Swaroopa	Big tree nearly 50-80 ft height.
Pryojyanga	Phala
Chemical Composition	Tannin, Chebulagic acid, Corilagin
Rasa	Lavanavarjita Pancharasa
Guna	Laghu, Rooksha
Vipaka	Madhura
Veerya	Ushna
Prabhava	Tridoshaghna

Karma	Vatarogahara, Rasayana, Malashodhini, Sangrahini, Kushta, Gulma, Udavarta, Shotha, Pandu, Arsha, Grahani, Vranahara

Rasayana-

It purifies Dhatus and alleviates doshas on them. With clearance of channels, respective elements from ahararasa are transported to Dhatus through rasa. Thus acts as Rasayana and Vayasthapana as the karma of Haritaki over nadivaha samsthan is balya and medhya indicated in conditions like mastishkya dourbalya, nadidourbalya and best in all vata vyadhis.[181]

Uses of Haritaki [182]

- Ignites the digestive fire making even micro nutrients available to the body
- Rejuvenates the body
- Increases the bulk of the body
- A very good nervine tonic
- A tonic for eyes
- Enhances the longevity of the person
- Nourishes the body up to the tissue level
- Scrapes out the unnecessary deposits and toxins out of the body
- Cardiac tonic

Clinical research involves the experimentation of a drug / therapy on a population and recording the feedback based on which postulations are made regarding the usefulness of the drug/therapy in the disease. Before starting any research work it is necessary to list out the materials required and the methods used for research. So in this chapter materials and methods which are required for this clinical study are explained in detail.

Research approach

In the present study, objective was "**A CLINICAL STUDY ON KAMPAVATA (PARKINSON'S DISEASE) AND ITS MANAGEMENT WITH TRIGUNA RASA"** The efficacy was determined by finding out the difference between the baseline data of the parameters to the after treatment data in comparison.

Sources of data

Patient:

Patients are selected from O.P.D of D.G.M.A M.C. and H. after fulfilling the inclusion and exclusion criteria.

Literary:

Required literary information for the intended study will be procured from both the Ayurvedic & contemporary books & updated with recent journals of both faculties.

Trail drug: Internal administration of Triguna Rasa

Preparation of Triguna rasa

Shudda Parada & Gandhaka are mixed and made into kajjali & heated on moderate fire, after melting add equal quantity of Haritaki choorna and mixed. The preparation will be undertaken according to classical text instructions.

Method of collection of data:

Study design:

The patients of KAMPA VATA above the age group of 40 yrs. were selected randomly from O.P.D of D.G.M.A M.C. and H. after fulfilling the inclusion and exclusion criteria irrespective of their sex, occupation and socio-economic status. The size of sample was 20. The present study is a single group observational study where in, patients were assigned in one group. It is a Simple random sampling technique clinical trial.

Sample size:

20 patients were taken for the planed study as a single group.

Inclusion criteria

1. Patients with clinical signs & symptoms of Kampavata vis-à-vis Parkinsonism disease will be selected.
2. Patients of either sex are selected.
3. Patients above 40 years of age.

Exclusion criteria

Patients with other systemic disorder which interfere with the treatment will be excluded such as

1. Diffuse Lewy body disease
2. Jacobs disease
3. Striatonigral degeneration
4. Wilson's disease
5. Huntington's disease (chorea)
6. Alzheimer's disease
7. Drug induced

8. Trauma (pugilistic)
9. Cardio embolic stroke.

Criteria of diagnosis

Diagnosis will be made based on clinical signs & symptom of Kampavata vis-à-vis Parkinson's disease.

Posology:

Triguna rasa orally, starting on day one with 7 ratti and increase of one ratti daily as arohana krama up to twenty one days (i.e.27 ratti) and from twenty second day onwards one ratti will be decreased till to 7 ratti remains i.e. on 41st day as avarohana krama.

Anupana :

Ghrita followed by Rice with milk

Study duration

Internal Administration of Triguna rasa – **41** days

Follow up:

The duration of follow up was 30 days

Assessment of results:

The subjective and objective parameters of base line data to pre and post medication will be compared for assessment of the results. All the results will be analyzed statically for value using pared't' test.

Subjective parameter

1. Chestsanga: slowness and poverty of movements.
2. Kampa (Resting tremor): at least in one limb.
3. Sthamba (Rigidity): In any group of muscles in extremities.

4. Avanamana (Postural changes): Which includes signs like Rombergism.
5. Vak vikruti: Ekshruti (monotony) and Kala (low) speech.
6. Gatisanga: Slow stepped & short stepped gait with proplusion & retropulsions.

Objective parameters:

1. Tremors
2. Rigidity
3. Bradykinesia
4. Gait
5. Dressing
6. Postural stability

Investigations:

The following investigation is undertaken for exclusion.

1. Haemoglobin%
2. Total and differential leucocytes count
3. Erythrocyte Sedimentation rate.
4. Random blood Glucose.
5. Blood urea, Serum creatinine, Serum copper.
6. Complete urine examination.

Assessment Criteria: Assessment of values before and after treatment of subjective and objective criteria is used.

Table no 8 Subjective parametrers:

Grading for Variables

Kampa (Tremor)	**Score**
Bilateral violent tremor along with tremor in tongue and / or in eyelids lips and not suppressed or diminished by willed movement.	-3-
Bilateral tremor	-2-
Unilateral slight tremor present at rest decreased by action, increases by emotion and stress	-1-
No tremor	-0-
Gatisanga:-	
Unable to raise from bed and walk without assistance	-3-
Can walk slowly but need substantially help, shuffling with retropulsion/ propulsion lack of associated movement	-2-
Can walk without assistance slowly but with shuffling gait	-1-
Can walk brisk without aid	-0-
Vakvikriti :-	
Incomprehensive words, monotonous voice, echoing, speaks only on insistence of examiner	-3-
Monotonous voice, spilt consonance but understandable speaks feels with examiner	-2-
Variable tone of voice.	-1-
Normal speech	-0-
Stambha (rigidity)	
Marked rigidity in major joints of limbs, patients maintain abnormal sitting postures, stared eyes	-3-
Rigidity demonstrable on one of major joints	-2-
Cog-wheel rigidity feebly present and on continuos examination vanishes	-1-
No rigidity	-0-

Avanamna	
Complete bend down of body	3
Head bent forward with legs bent at knees	2
Only arm bent at elbows	1
No bending or flexion	0
Chestasanga	
Unable to carry routine activities of daily life	3
Able to perform daily activities with moderate difficulties	2
Able to perform daily activities with less difficulties	1
No difficulties in carrying out activities	0

Objective parameter

International accepted Unified Parkinson Disease Rating Scale (UPDRS) used for functional assessment of the patient before and after the treatment

Grading for Variables

Sl.No	Objectives	Gradations
1	Tremors	**Gr 0 – Absent**
		Gr 1 - Slight and infrequent
		Gr 2 – moderate
		Gr 3 – Marked
		Gr 4 - Marked with all activities
2	Rigidity	**Gr 0 – Absent**
		Gr 1 - Slight and infrequent
		Gr 2 – moderate
		Gr 3 - Severe, interferes with many activities
		Gr 4 - Marked with all activities
3	Bradykinesia	**Gr 0 – None**
		Gr 1 - Minimal slowness
		Gr 2 – Mild slowness and poverty of movement
		Gr3 – Moderate slowness poverty or small amplitude

		Gr 4 - Marked slowness ,poverty,or amplitude
4	Gait	**Gr 0 – Normal**
		Gr 1 - Walks slowly , may shuffle with worst steps no propulsion
		Gr 2 - Walks with difficulty or little assistance or no assistance
		Gr 3 - Severe disturbance no assistance
		Gr 4 - Cannot walk
5	Dressing	**Gr 0 - – Normal**
		Gr 1 - Slow no help needed
		Gr 2 Occasional help with buttons
		Gr 3 - Considerable help required
		Gr 4 – helpless
6	Postural stability	**Gr 0 - – Normal**
		Gr 1 - Recovers unaided
		Gr 2 Would fall if not caught
		Gr 3 - Falls spontaneously
		Gr 4 - Unable to stand

ASSESSMENT CRITERIA FOR OVER ALL EFFECT OF TREATMENT

- Good Response : >75% improvement in clinical parameters
- Moderate Response : 50-75% improvement in clinical parameters
- Poor Response : 25 to 50% improvement in clinical parameters
- No Response : below25 % or No improvement in clinical parameters

Ingredients of Triguna rasa

Parada

Gandhaka

Haritaki

Triguna rasa

Triguna rasa medicine

Present study registers 20 patients and after fulfilling the criteria of diagnosis and inclusive criteria patients were included as a single group for the present study. All the patients were examined before and after the trail, according to the case sheet format given in the annex. Both the subjective and objective criteria were recorded along with validation of disease state. The data recorded are presented under the following headings.

- Demographic data
- Evaluating disease Data and
- Statistical analysis
- Result of the Triguna rasa

Demographic data:

The details of Age, Gender, Religion, etc. of 20 patients are as follows.

Table no 10 Showing age wise distribution of patients

Age	Patients	Percentage
50-55	05	25
55-60	08	40
60-65	03	15
65-70	04	20
Total	20	100%

Here an interval of 05 has considered from the ages 50 to 70. In the study it is revealed that Kampavata is found more in age between 55-60 years .i.e. 40%, and 25% were seen between age group of 50-55, 20% were seen in age group of 65-70 years, and fewer incidences was seen in age group of 60-65 years i.e. 15%.

Figure No 7. Showing Age wise distribution of patients

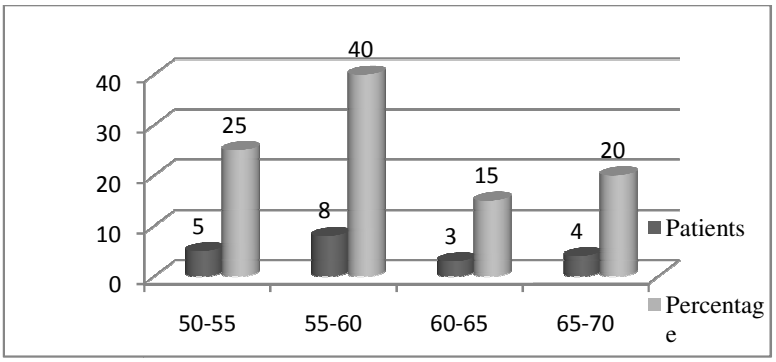


Table No 11 Showing Sex wise Distribution of patients

Gender	Total	Percentage
Male	15	75
Female	05	25

The above table shows that Males are more affected with kampavata than the females. The observations are 15 Patients i.e. (75%) were male and 5 patients i.e. (25%) were female.

Figure No. 8 showing sex wise distribution of patients

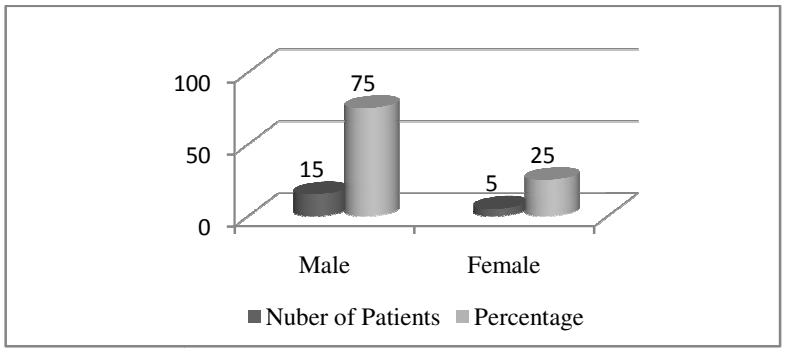


Table no 12 Showing Religion wise distributions of patients

Religion	Total no. of Patients	Percentage
Hindu	19	95
Muslim	01	05
Christian	00	00
Others	00	00

The maximum number of patients are noticed from the Hindu community as the ratio of community at the study area is more i.e. 19 (95%) along with 01 Muslim patient (05%).

Figure No 9 Showing Religion wise distributions of patients

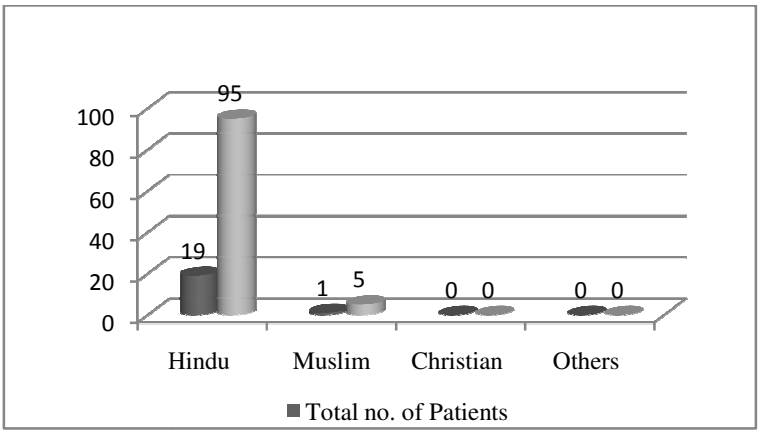


Table no 13. Showing economic status wise distribution of patients

Economical status	Total no. of Patients	Percentage
Poor	03	15
Middle	14	70
Higher class	03	15

Here 03(15%) patients belong to poor class and 15(70%) patients belongs middle class and 03 (15%) patients from higher class.

Figure No 10. Showing economic status distributions of patients

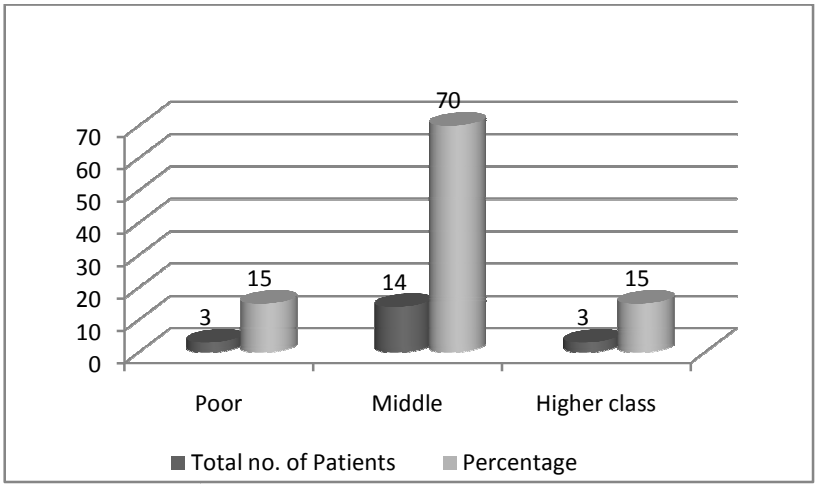


Table no 14. Showing occupation wise distribution of patients

Occupation	Number of patients	Percentage
Sedentary	09	45
Labour	06	30
Active	05	25

In occupation wise distribution 09 (45%) of the patients had sedentary life style where 05 (25%) patients had active occupation and 06 (30%) were patients of labor class.

Figure No 11. Showing occupation wise distribution of patients

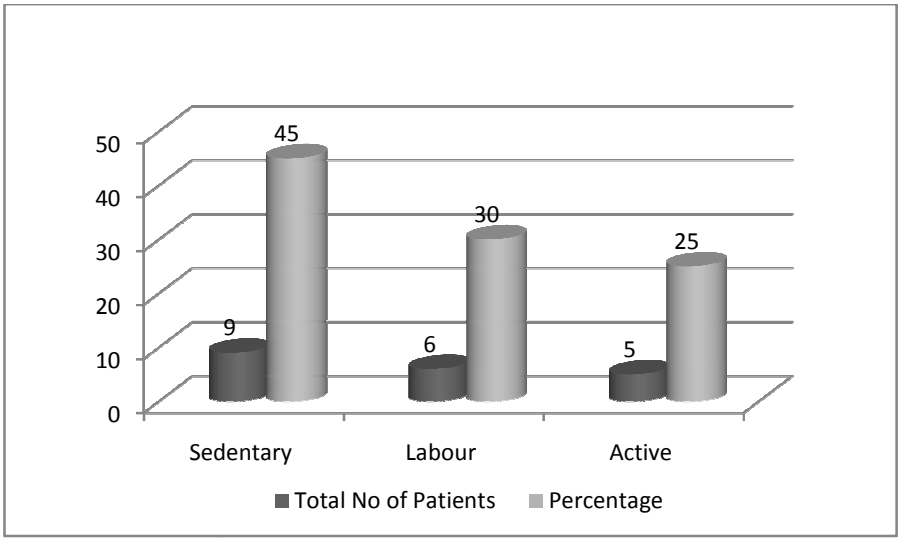


Table no 15. Showing diet wise distribution of patients

Diet	Number of patients	Percentage
Vegetarian	16	80
Mixed	04	20
Stored	00	0

In diet wise distribution of patients 16 (80%) patients were vegetarian and 4 (20%) patients were of mixed diet.

Figure 12 Showing diet wise distributions of patients

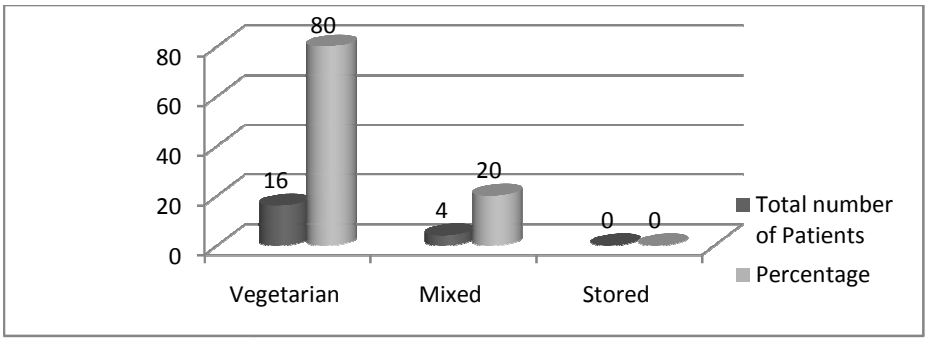


Table-16: Showing Intake of Rasa predominance wise distribution of patients

Rasa predominance	Total no. of Patients	Percentage
Madhura	07	35
Amala	08	40
Lavana	14	70
Katu	17	85
Tikta	04	20
Kashaya	06	30

In this study maximum 17 (85%) patients are habitual of taking Katu Rasa predominantly in their diet, followed by 14 (70%) of lavana Rasa, 08 (40%) of Amla Rasa, and 07 (35%) of Madhura Rasa 06 (30%) of Kashaya rasa and 04 (20%) patients consumed tikta rasa. Excessive use of Katu and kashaya Rasa may be the etiological factors of this disease.

Figure No 13 Showing Intake of Rasa predominance wise distribution of patients

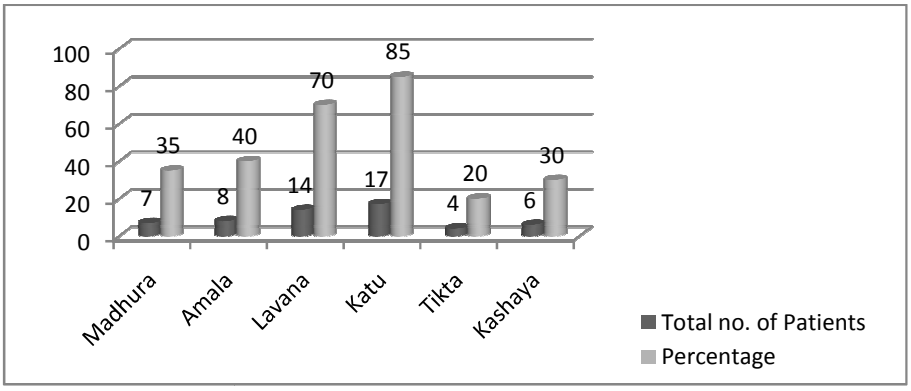


Table no 17. Showing marital status wise distribution of patients

Marital status	Total	Percentage
Unmarried	0	00
married	20	100

All 20 (100%) patients were married.

Figure No 14 showing marital status wise distribution of patients

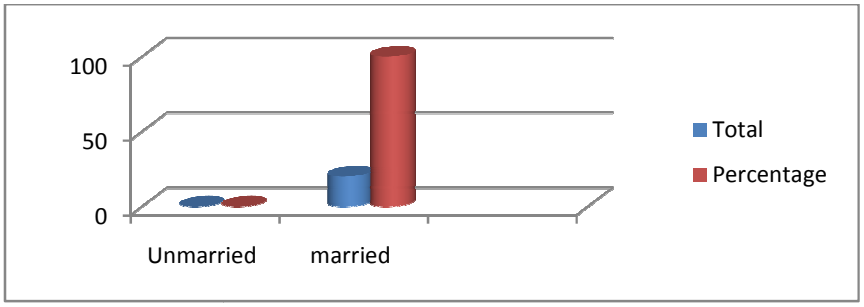


Table no 18 Showing Diagnosis wise distributions of patients

Diagnosis	Total	Percentage
Newly diagnosed	13	65
Previously diagnosed	07	35

In the study maximum patients ie 13 (65%) patients were newly diagnosed and 07 (35%) patients were previously diagnosed.

Figure No 15 Showing Diagnosis wise distributions of patients

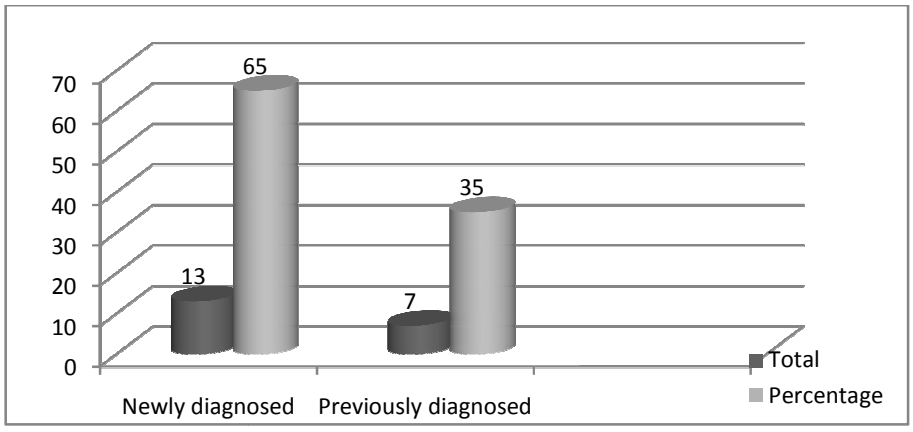


Table 19 Showing previous medication wise distributions of patients

Previous medication	Total	Percentage
Ayurvedic	05	25
Allopathic	02	10

In this study 05 (20%) patients were previously diagnosed and treated with Ayurvedic treatment and 02 (10%) patients were treated with allopathic system of medicine.

Figure No16. Showing previous medication wise distributions of patients

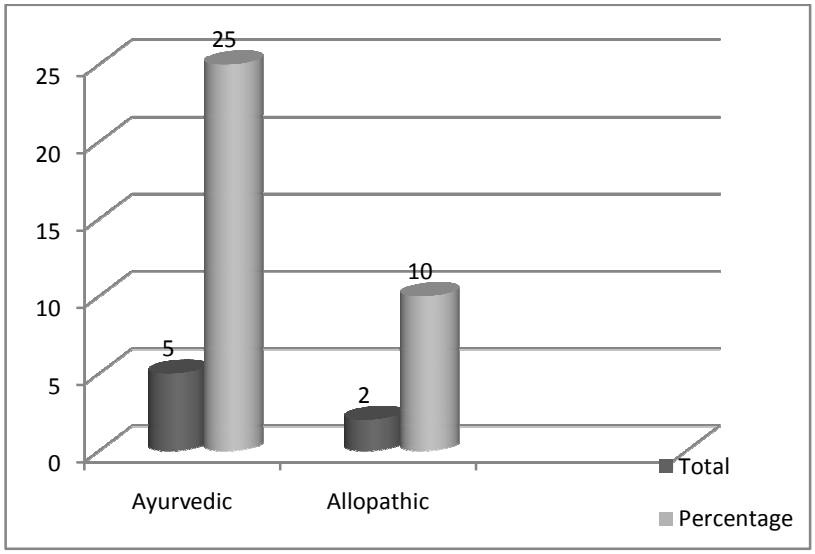


Table 20 showing Addiction wise distributions of patients

Addiction	Total	Percentage
Alcohol	04	20
Smoking	04	20

In this study 04 (20%) patients had habit of consuming alcohol and 04 (20%) patients had habit of smoking.

Figure No 17. Showing Addiction wise distributions of patients

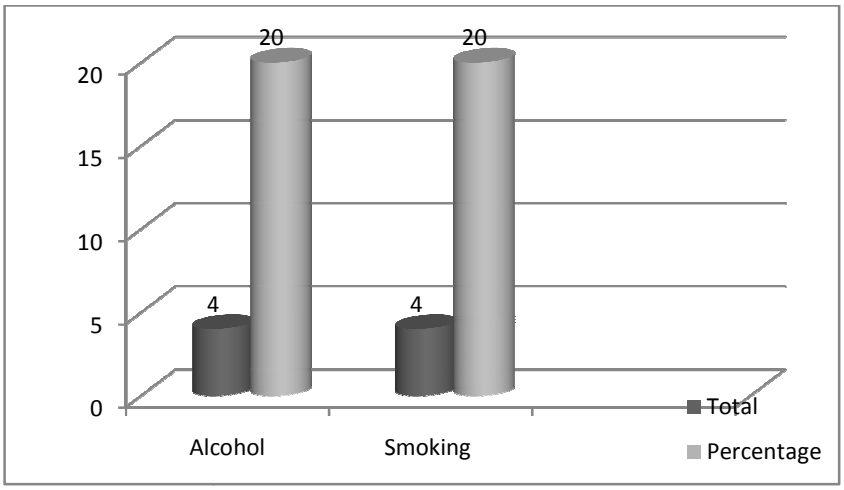


Table no 21. Showing nidra wise distribution of patients

Nidra	Total	Percentage
Normal	09	45
Disturbed	11	55

The present study shows that maximum 11 (55%) of the patients had disturbed sleep and 09 (45%) of the patients had normal sleep

Figure No 18 Showing nidra wise distribution of patients

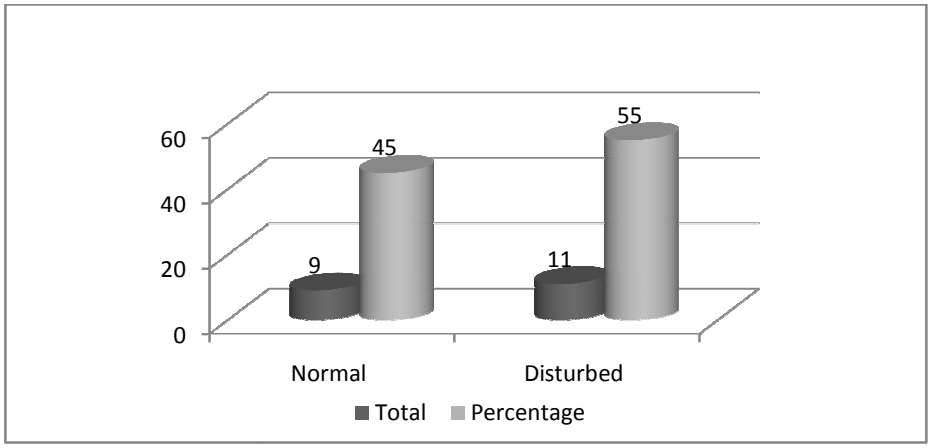


Table no 22. Showing work involving stress wise distribution of patients

Stress	Total	Percentage
Work involving stress	03	15
Work not involving stress	17	85

Here only 03 (15%) patient was suffering from work involving stress and rest 17 (85%) patients dint face any stress at work.

Figure No 19. Showing work involving stress wise distribution of patients

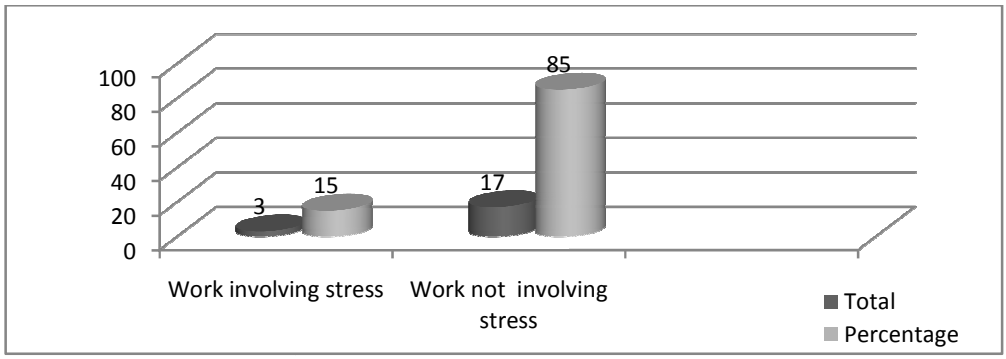


Table no.23 Showing Activity affected wise distribution of patients

Activity affected	Total	Percentage
Writing	08	40
Eating	05	25
Small movements	10	50

In the study 08(40%) people had difficulty in writing, 05(20%) people had difficulty in eating and 10(50%) patients had difficulty in carrying small movements.

Figure No 20. Showing difficulty wise distribution of patients

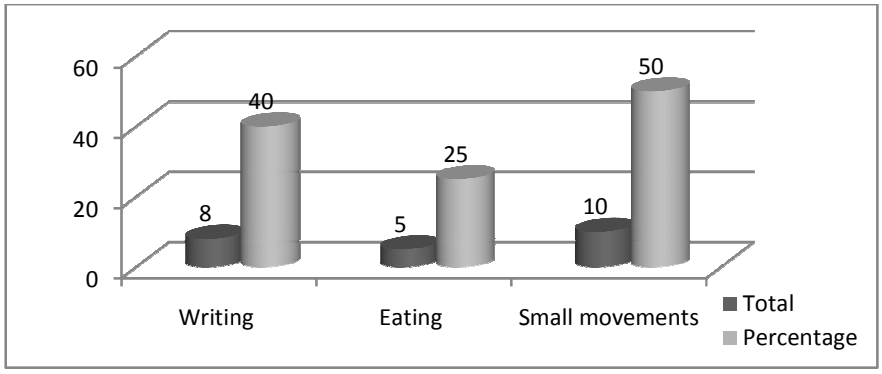


Table no 24. Showing Kosta wise distribution of patients

Kosta	Total	Percentage
Krura	01	5
Mrudu	07	35
Madhyma	12	60

The above table shows 12 (60%) patients had madhyma kosta, 07 (35%) had mrudu kosta, and 1 patient (5%) had krura kosta.

Figure No 21. Showing Kosta wise distribution of patients

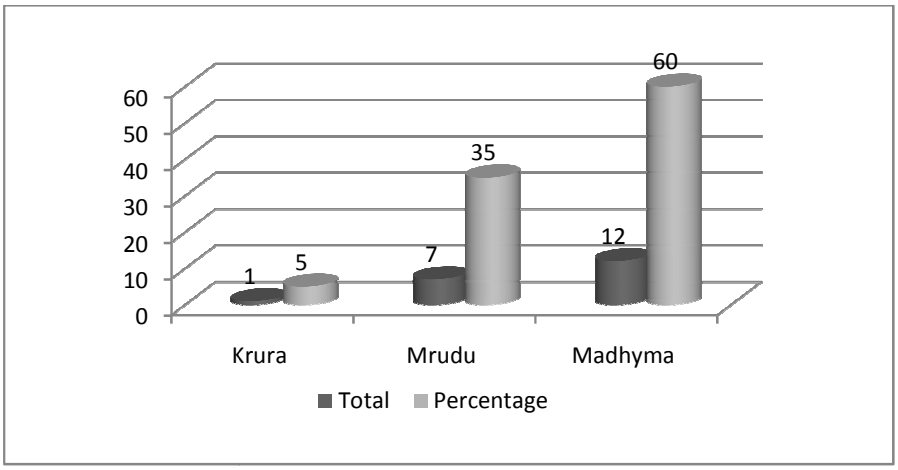


Table no 25. Showing Agni wise distribution of patients

Agni	Total	Percentage
Vishmagni	03	15
Tkishnagni	01	5
Mandagni	08	40
Samagni	08	40

The above data in table shows 03 (15%) patients had vishamagni, 01(5%) patient had tikshnagni, 08 (40%) patients had mandagni, and 08 (40%) of them had samagni.

Figure No 22. Showing Agni wise distribution of patients

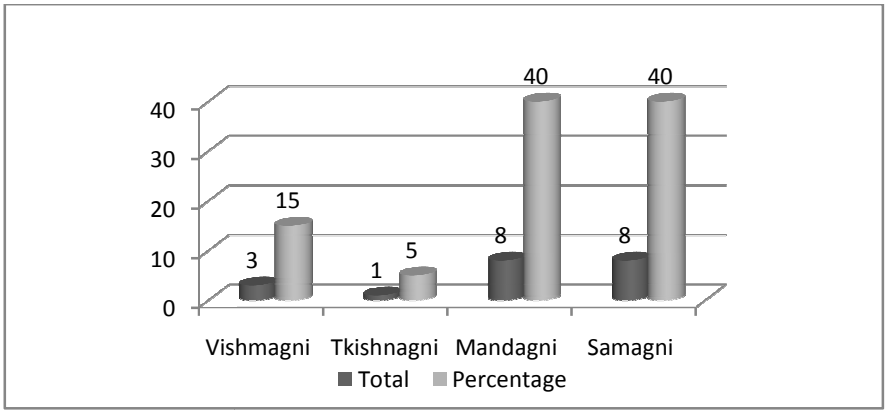


Table no 26. Showing Shareera prakriti distribution of patients

Shareera prakriti	Total	Percentage
Vata pitta	15	75
Vata kapha	04	20
Pitta kapha	00	00
Vataja	01	05
Pittaja	00	00

The study shows 1 (05%) of the patient had vata prakriti, 15 (75%) of the patients had Vatapitta prakriti, 04 (20%) of the patients had vatakapha.

Figure No 23. Showing Shareera prakriti distribution of patients

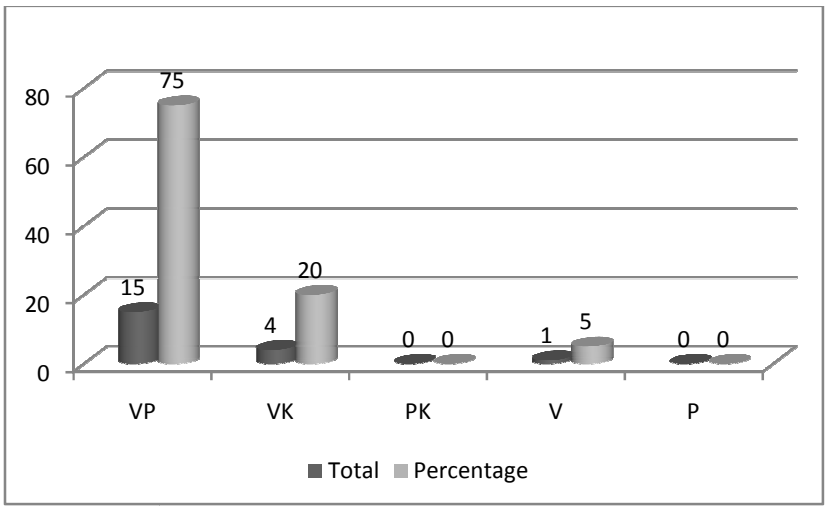


Table no 27. Showing Shareera sara wise distribution of patients

Sara	Total	Percentage
Asti	16	80
Mamsa	02	10
Meda	02	10

Here 16 (80%) patients were of asti sara, 02 (10%) patients of mamsa sara and medha sara people were of 2 (10%) patients.

Figure No 24. Showing Shareera sara wise distribution of patients

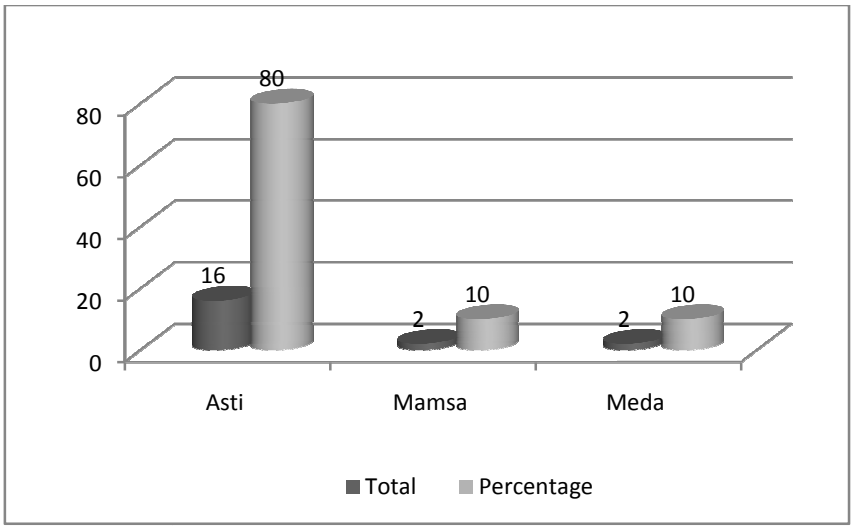


Table no 28. Showing Shareera samhanana wise distribution of patients

Samhanana	Total	Percentage
Susammhita	02	10
Madhyama	17	85
Heena	01	5

In the Study 02 (10%) of the patients were having Susamhata and 17 (85%) of the patients were having Madhyama samhata and 01 (05%) was of Hina samhata.

Figure No 25. Showing Shareera samhanana wise distribution of patients

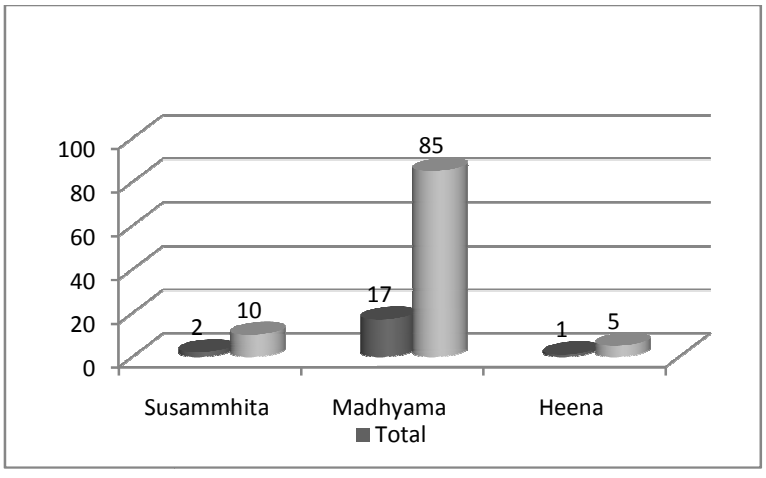


Table no 29. Showing Shareera satmya wise distribution of patients

Satmya	Total	Percentage
Pravara	06	30
Madhyama	12	60
Avara	02	10

In the Study 06 (30%) of the patients were having Pravara satmya and 12 (60%) of the patients were having Madhyama satmya and 02 (10%) were of Avara satmya.

Figure No 26. Showing Shareera satmya wise distribution of patients

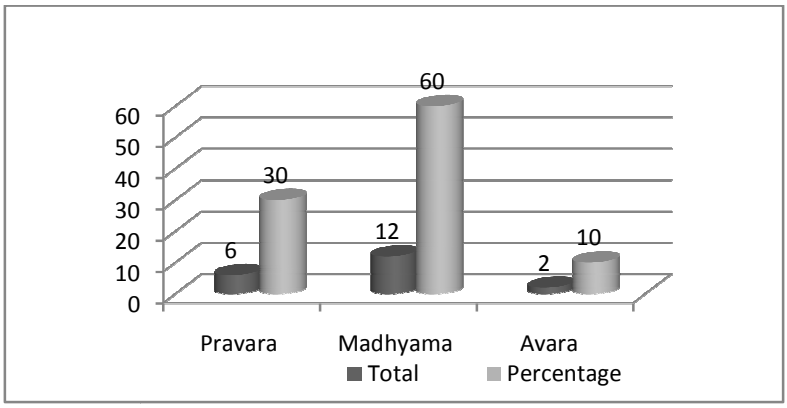


Table no 30. Showing Shareera Satwa wise distribution of patients

satwa	Total	Percentage
Pravara	05	25
Madhyama	13	65
Avara	02	10

In the Study 05 (25%) of the patients were having Pravra satwa and 13 (65%) of the patients were having Madhyama Satwa and 02 (10%) patients of Avara satwa

Figure No 27. Showing Shareera Satwa wise distribution of patients

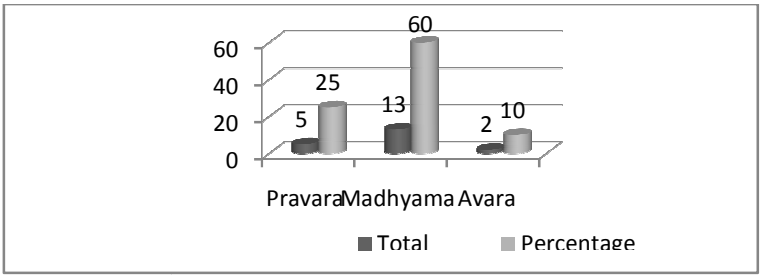


Table no 31. Showing Shareera vyama Shakti wise distribution of patients

Vyama shakti	Total	Percentage
Pravara	02	10
Madhyama	16	80
Avara	02	10

In the Study 02 (10 %) of the patients were having Pravara Vyama shakti and 16 (90 %) of the patients were having Madhyama Vyama shakti and 02 (10%) patients of Avara Vyama shakti .

Figure No 28. Showing Shareera vyama Shakti wise distribution of patients

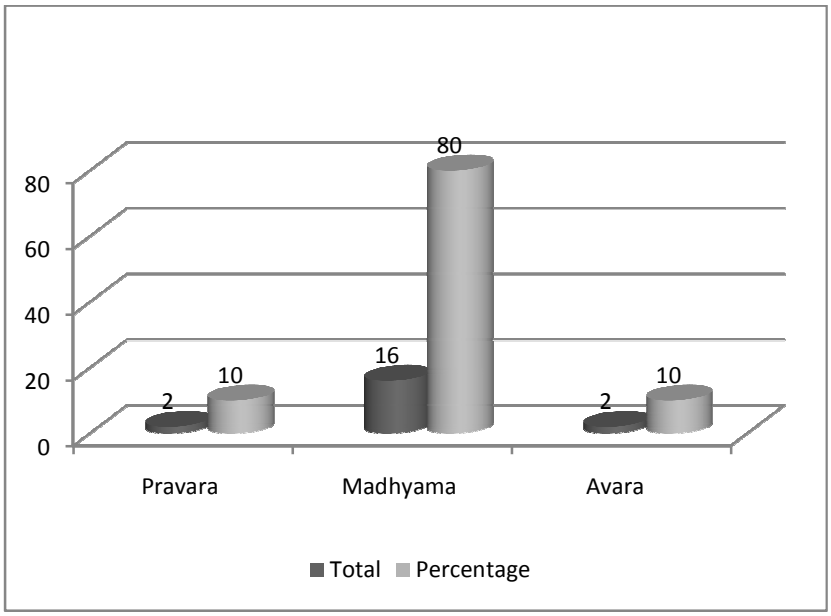


Table no 32. Showing Shareera Vaya wise distribution of patients

Vaya	Total	Percentage
Vrudha	05	25
Madhyama	15	75

In the study 05 (25%) patients were of vrudha vaya and 15 (75%) were of madhyama vaya.

Figure No 29. Showing Showing Shareera Vaya wise distribution of patients

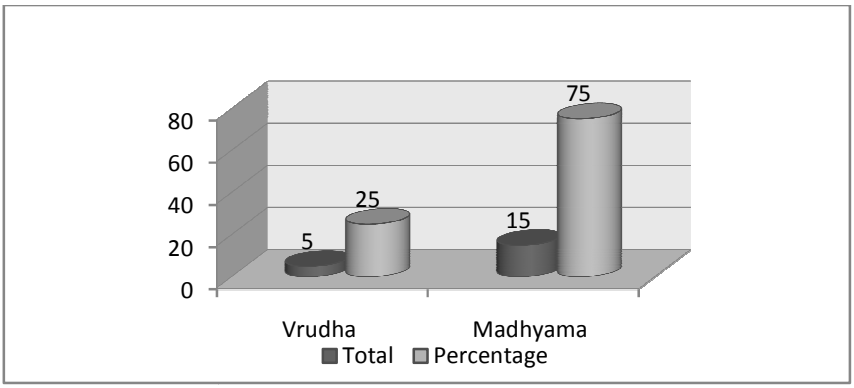


Table no 33. Showing Shareera Pramana wise distribution of patients

Pramana	Total	Percentage
Supramanatha	18	90
Heena	02	10

In the study 18 (90%) patients were of supramanatha and 02 (10%) were of heena pramana.

Figure No 30. Showing Shareera Pramana wise distribution of patients

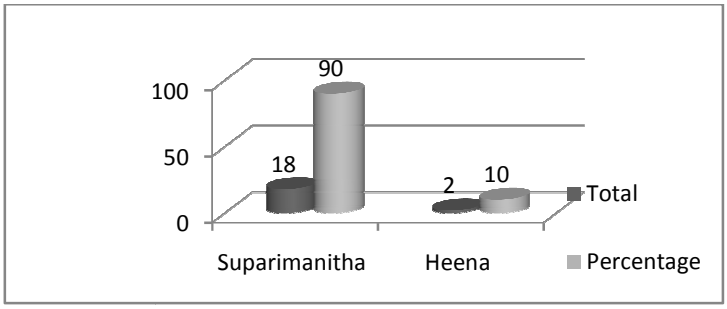


Table no 34. Showing Ahara shakti wise distribution of patients

Ahara shakti	Total	Percentage
Pravara	01	5
Madhyama	17	85
Avara	02	10

In the Study 17(85%) of the patients were having Madhyama and 2 (10%) of the patients were having avara and1 (05%) were having Prvara Aaharshakti.

Figure No 31. Showing Ahara shakti wise distribution of patients

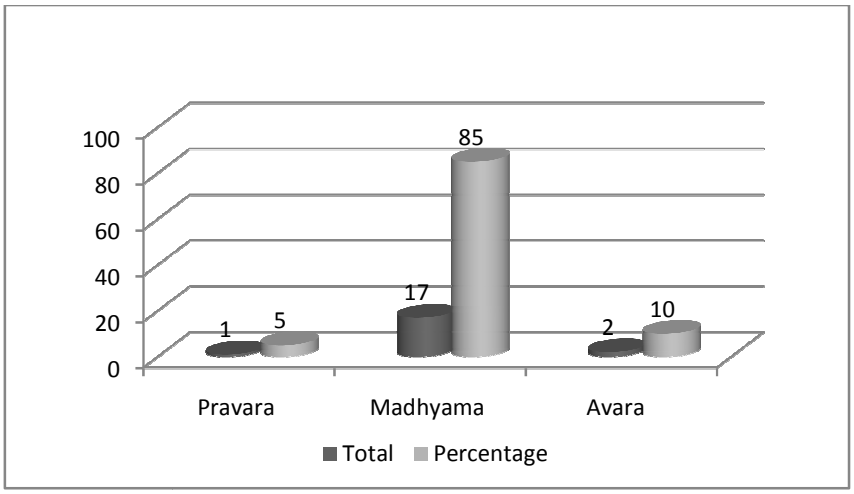


Table no 35. Showing Jarana shakti wise distribution of patients

Jarana shakti	Total	Percentage
Pravara	00	00
Madhyama	19	95
Avara	01	5

In this study 19 (95 %) patients had madhyama jarana shakti 01(05 %) had avara jarana shakti.

FigureNo 32. Showing Jarana shakti wise distribution of patients

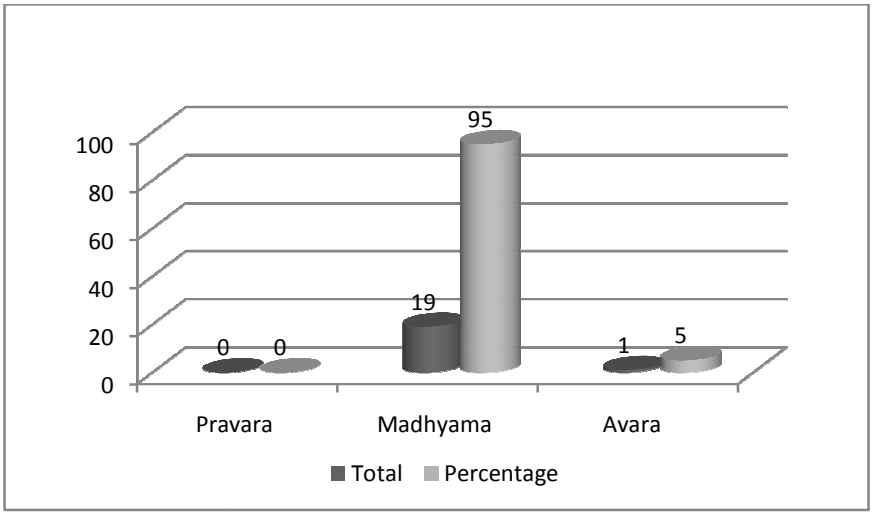


Table no 36. Showing manasika vrittanta wise distribution of patients

Manasika vrittanta	Total	Percentage
Emotional stress	04	20
Anxiety	12	60
Depression	05	25
Unusual laugh	01	05
Aggressiveness	12	60
Mada	02	10
Panic	01	05

In the present study 04 (20%) patients had emotional stress, 12 (60%) patients had anxiety, 05 (25%) patients had depression, 01 (5%) patient had unusual laugh, 12 (60%) patients had samprahara (aggressiveness) in their nature, 02 (10%) patients had mada and 01 (5%) patient was suffering with panic.

Figure No 33 showing manasika vrittanta wise distribution of patients

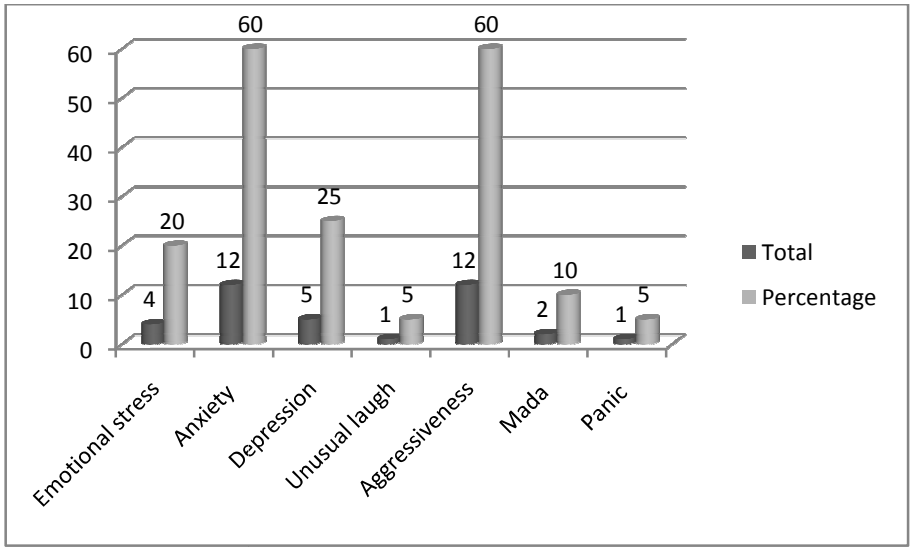


Table no 37. Showing chronicity of Kampa in patients

Kampa	total	percentage
Acute	10	50
Chronic	10	50

In this study 10 (50%) patients were of acute onset of disease and another 10 (50%) were of chronic origin.

Figure No 34 showing chronicity of Kampa in patients

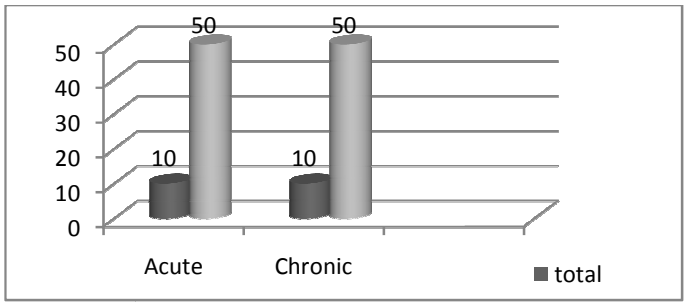


Table no 38. Showing chronicity of Sthamba in patients

Sthamba	total	percentage
Acute	12	60
Chronic	07	35
Not found	01	05

In this study 12 (60%) patients were suffering from sthamba of acute onset and 07 (35%) patients were of chronic onset and 01 (5%) patient dint have sthamba symptom.

Figure No 35 showing chronicity of Sthamba in patients

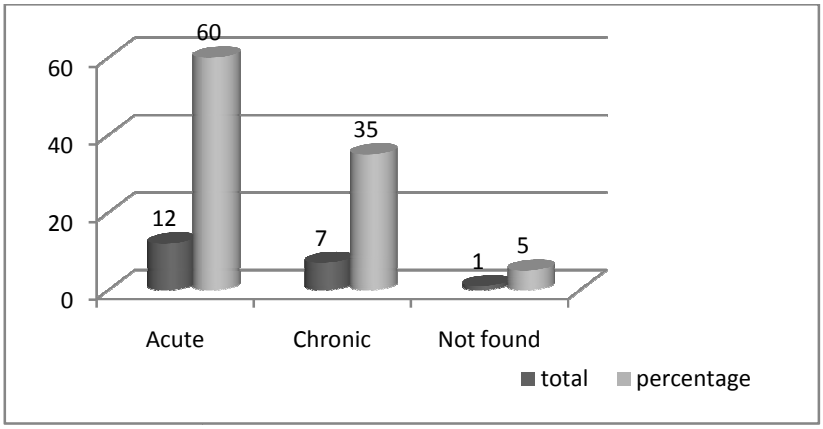


Table no 39. Showing chronicity of Cestasanga in patients

Chestasanga	Total	Percentage
Acute	11	55
Chronic	08	40
Not found	01	05

In this study 11 (55%) patients were suffering from Chestasanga of acute onset and 08 (40%) patients were of chronic onset and 01 (5%) patient was not having Chestasanga symptom.

Figure No 36. Showing chronicity of Cestasanga in patients

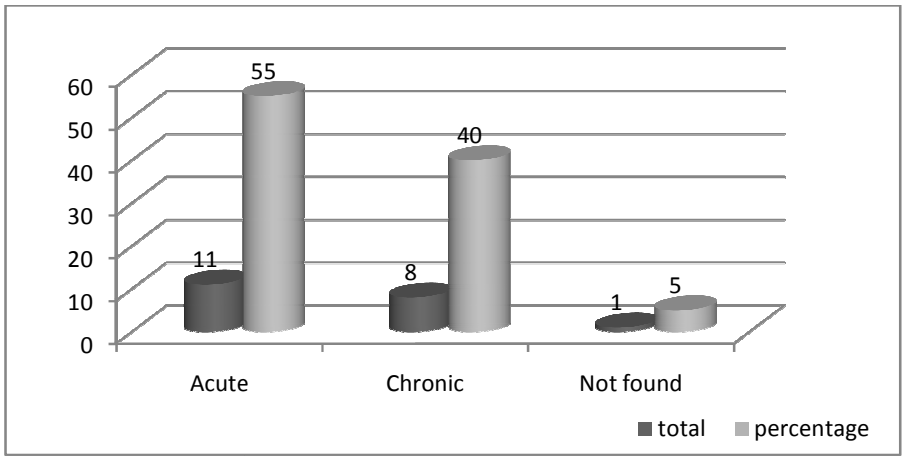


Table no 40. Showing chronicity of Avanamana in patients

Avanamana	Total	Percentage
Acute	01	5
Chronic	05	25
Not found	14	70

In this study 1 (5%) patients were suffering from Avanamana of acute onset and 05 (25%) patients were of chronic onset and14 (70%) patient was not having Chestasanga symptom.

Figure No 37. Showing chronicity of Avanamana in patients

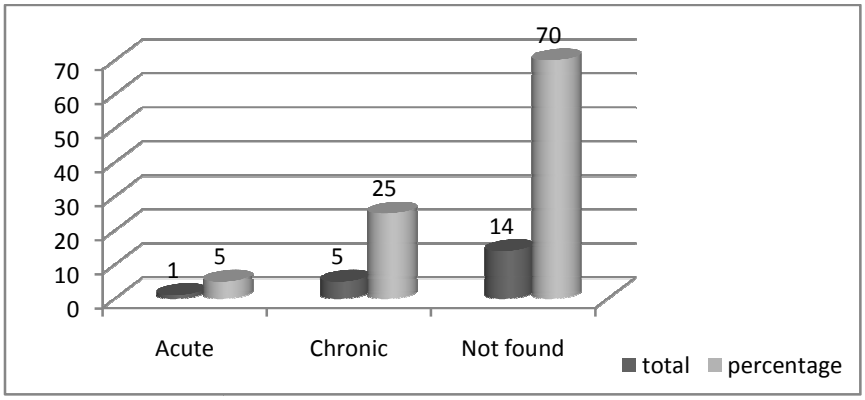


Table no 41. Showing kampa in different part of body wise distribution of patients

Kampa of	Total	Percentage
Head	02	10
Tongue	02	10
Lips	00	00
Chin	00	00
Right UE	10	50
Left UE	15	75
Right LE	02	10
Left LE	03	15
One side of body	03	15
Complete body	01	05

In this study as mentioned in above table, 02 (10%) patients had kampa (tremor) of head, 02 (10%) patients had kampa of Tongue, no patient was found with kampa of lips and chin, 10 (50%) patients had kampa(tremors) in right upper extremity, 15 (75%) patients had kampa (tremor) in left upper extremity, 02 (10%) patients had kampa(tremor) in right lower extremity, 03 (15%) patients had kampa (tremor) in left lower extremity, 03 (15%) patients had kampa (tremors) on one side of body, and 01 (5%) patient had kampa (tremor) in complete body.

Figure No38 Showing kampa in different part of body wise distribution of patients

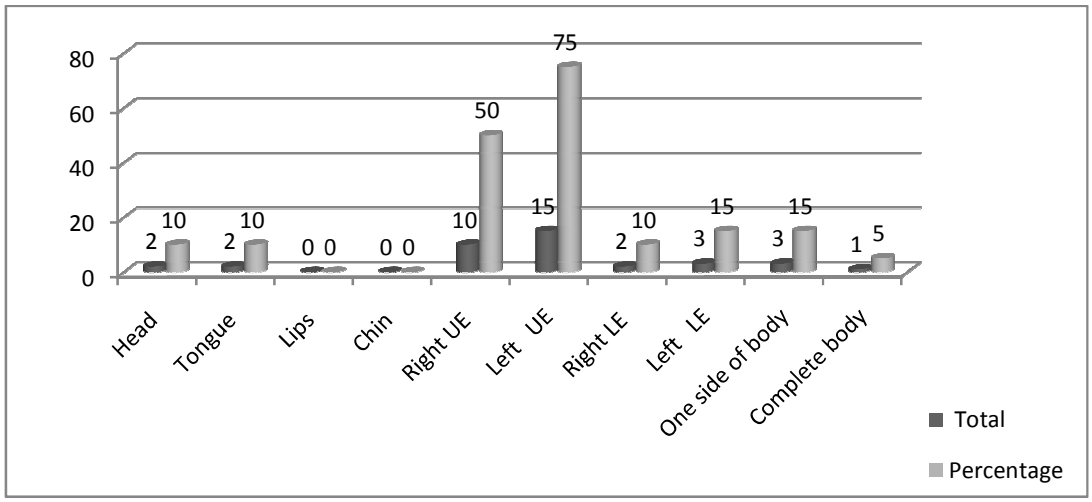


Table no 42. Showing Chestasanga pradhana vedana wise distribution of patients

Chestasanga	Frequency	Percentage
Slow movement	13	65
Stooped	05	25
Difficulty to begin walk	08	40
Small hand writing	07	35
Decreased facial expression	06	30
Difficulty in voluntary movements	08	40
Soft speech	08	40

In this study 13 (45%) patients were suffering from slow movement,05 (25%) patients had stooped body, 08 (40%) patients had difficulty to begin in walking, 07 (35%) patients had small hand writing, 06 (30%) patients had decrease in their facial expression, 08 (40%) patients had difficulty in carrying out voluntary movements, and 08(40%) had soft speech.

Figure No 39 Showing Chestasanga pradhana vedana wise distribution of patients

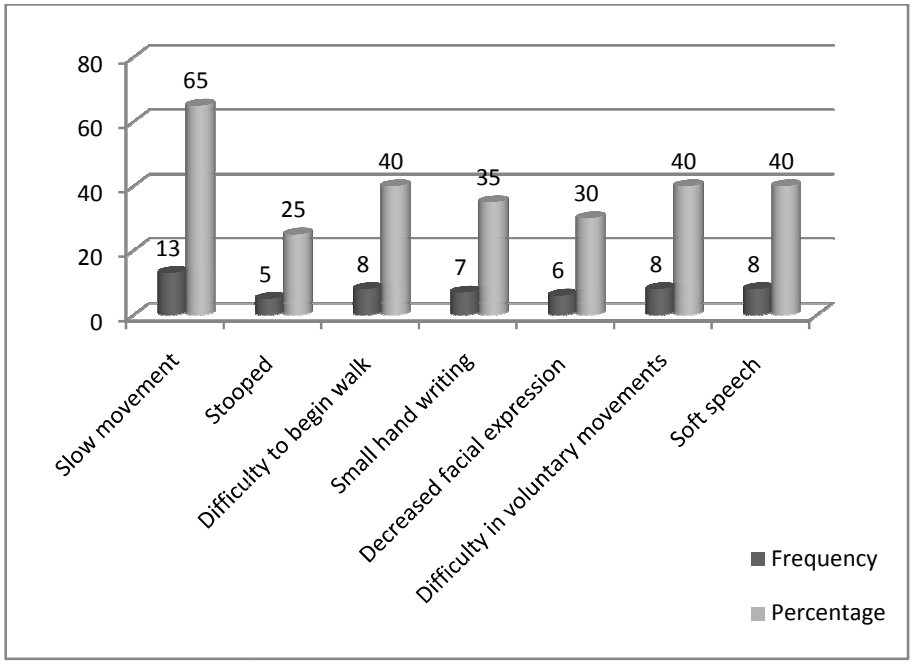


Table no 43. Showing distribution of Stamba in patients

Sthamba	Total	Percentage
Neck	00	00
Right UE	09	45
Left UE	14	70
Right LE	01	05
Left LE	01	05
Avanamna	05	25

Stambha as on e of pradhan vedana explained as no patients were found with stiffness of neck, 09 (45%) patients were suffering from stiffness (stambha) of right upper extremity, 14 (60%) patients had stiffness (stambha) of left upper extremity,01 (5%) patient had stiffness (stambha) of right lower extremity, 01 (5%) patient had stiffness (stambha) of left lower extremity and 05 (25%) patients were having avanamana (flexed posture).

Figure No 40. Showing stamba pradhana vedana wise distribution of patients

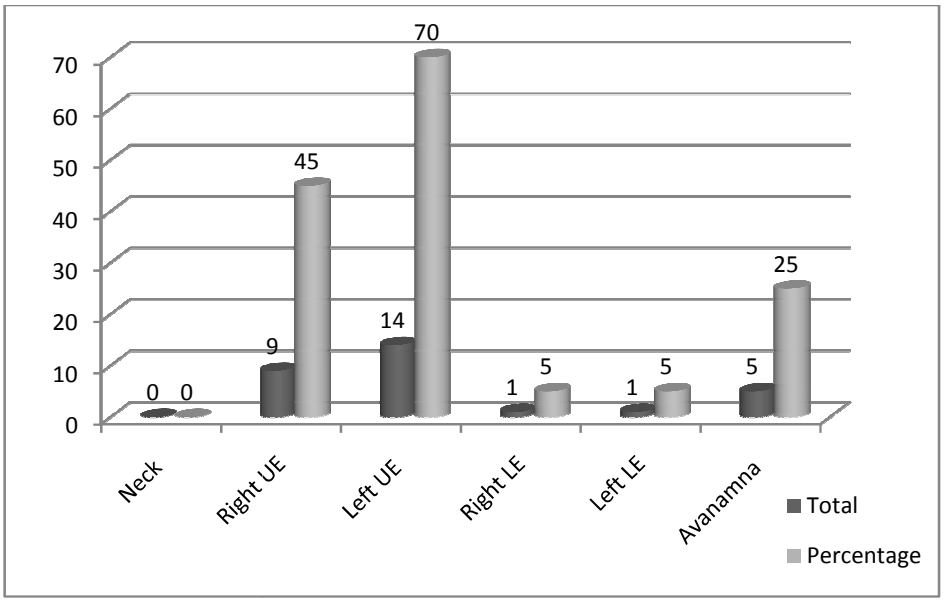
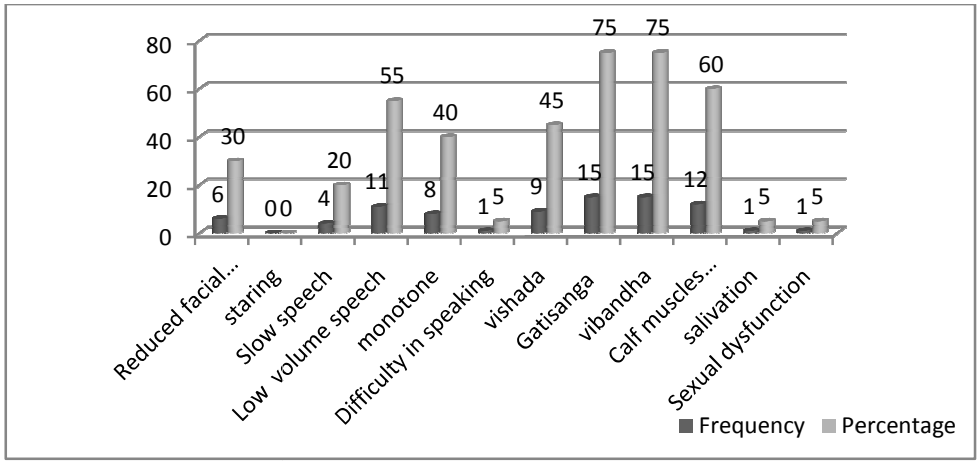


Table no 44. Showing Anubhandha vedana wise distribution of patients

	Frequency	Percentage
Reduced facial expression	06	30
staring	00	00
Slow speech	04	20
Low volume speech	11	55
monotone	08	40
Difficulty in speaking	01	05
vishada	09	45
Gatisanga	15	75
vibandha	15	75
Calf muscles tightness	12	60
salivation	01	05
Sexual dysfunction	01	05

In the present study 06 (30%) patient had reduced facial expression, no patients were suffering from symptom of staring, 04 (20%) patients had slow speech, 11 (55%) patients had low volume speech, 08 (40%) patients had monotone voice, 01 (05%) patient had difficulty in speaking, and 09 (45%) patients were suffering from vishada,15 (75%) patients had gatisanga, 15 (75%) patients were suffering from vibhanda, 12 (60 %) patients had tightness of calf muscle, 01 (05%) patients had excess salivation, 01 (05%) patient had sexual dysfunction.

Figure No 41. Showing Anubhandha vedana wise distribution of patients



Observations on Subjective parameters

Table no. 45 Showing Subjective parameters wise distribution of patients

Subjective parameters	Total	Percentage
Kampa	20	100
Gatisanga	19	95
Vakvikruti	18	90
Sthamba	17	85
Avanamana	05	25
Chestasanga	20	100

In the present study Kampa was seen in all 20 (100%) patients, gatisanga was seen in 19 (95%) patients, 18 (90%) patients had vakvikruti, 17 (85%) patients had sthamba, all 20 (100%) patients had Chestasanga and only 5 (25%) patients had Avnamana.

Figure No 42.Showing Subjective parameters wise distribution of patients

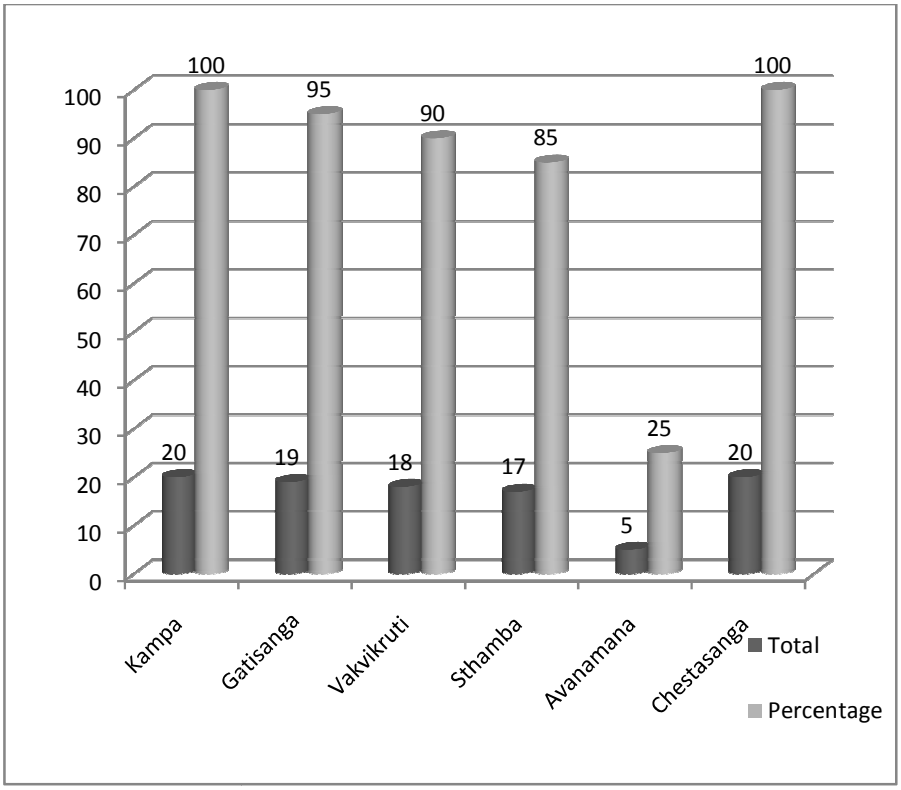
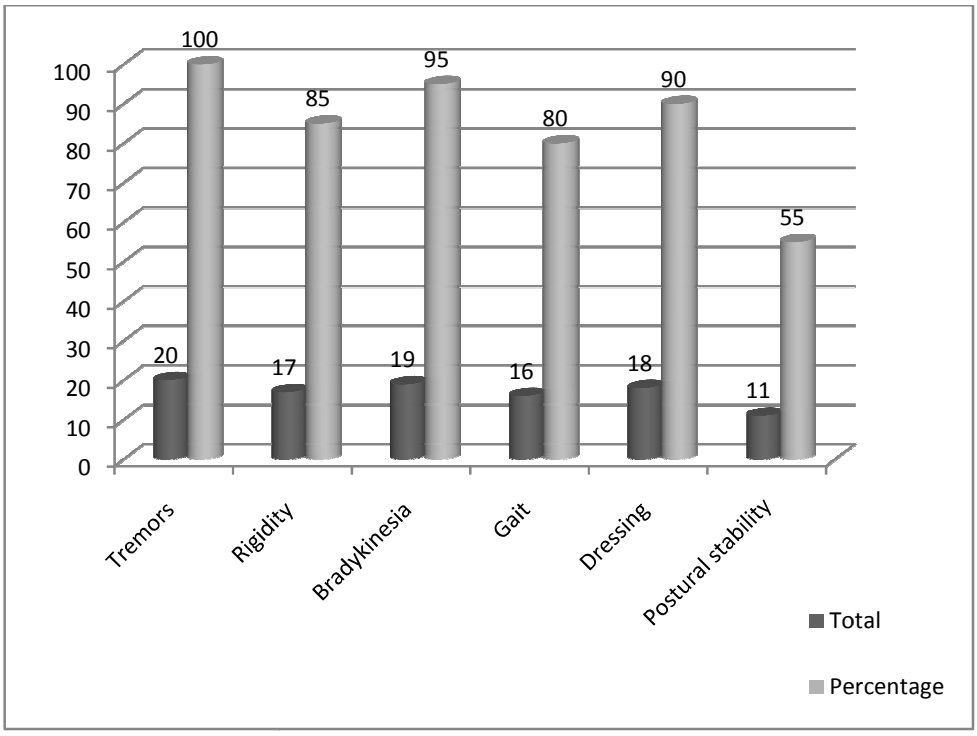


Table no.46 Showing Objective parameters wise distribution of patients

Objective parameters	Total	Percentage
Tremors	20	100
Rigidity	17	85
Bradykinesia	19	95
Gait	16	80
Dressing	18	90
Postural stability	11	55

In the present study Tremor was seen in all 20 (100%) patients, Rigidity was seen in 17 (85%) patients, 19 (95%) patients had Bradykinesia, 16 (80%) patients had Gait impairment, 18 (90%) patients had Dressing difficulties and 11 (55%) patients had problems with postural stability.

Figure No 43. Showing Objective parameters wise distribution of patients



Master charts Data of trial

Table No 47 -Showing demographic data in patients

Sr.	O.P.D	Age	Sex		Religion		Economic statu			Occupation			Diet			Marital status	
			M	F	H	Ms	P	M	H	Se	L	A	V	M	St	Md	Um
01	35969	50	+	-	+	-	-	+	-	-	+	-	+	-	-	+	-
02	37765	50	-	+	+	-	+	-	-	-	+	-	+	-	-	+	-
03	40927	70	+	-	+	-	-	+	-	+	-	-	-	+	-	+	-
04	2237	67	+	-	+	-	-	+	-	+	-	-	-	+	-	+	-
05	13950	50	+	-	+	-	-	+	-	-	+	-	+	-	-	+	-
06	17011	60	+	-	+	-	-	-	+	+	-	-	+	-	-	+	-
07	17608	60	-	+	+	-	-	+	-	+	-	-	+	-	-	+	-
08	22814	60	+	-	+	-	-	+	-	-	+	-	+	-	-	+	-
09	24501	56	-	+	+	-	-	+	-	+	-	-	+	-	-	+	-
10	25076	50	+		-	+	-	+	-	-	-	+	-	+	-	+	-
11	25097	56	+	-	+	-	-	-	+	-	-	+	+	-	-	+	-
12	25118	60	+	-	+	-	-	+	-	-	-	+	+	-	-	+	-
13	27545	50	-	+	+	-	+	-	-	-	+	-	+	-	-	+	-
14	29351	62	+	-	+	-	-	+	-	+	-	-	+	-	-	+	-
15	29823	70	+	-	+	-	-	+	-	+	-	-	+	-	-	+	-
16	31235	74	+	-	+	-	+	-	-	+	-	-	+	-	-	+	-
17	33122	57	+	-	+	-	-	+	-	-	+	-	+	-	-	+	-
18	33709	58	+	-	+	-	-	+	-	-	-	+	-	+	-	+	-
19	34211	62	+	-	+	-	-	-	+	-	-	+	+	-	-	+	-
20	34674	65	-	+	+	-	-	+	-	+	-	-	+	-	-	+	-

NOTE- Sex: M-Male, **F**- Female, **Religion: H**-Hindu, **M**- Muslim, E**conomic statuP**-poor, **M**- Middle, **H**- Higher class, **Occupation: S**- Sedentary, **A**-Active, **L**-Labor **Diet: V**-Vegetarian, **M**-Mixed **St**-Stored,Md-married,Um- unmarried.

Master chart vyaktika vrittanta

Table No 48-Showing Vayktika vrittanta wise data in patient

S l no	O.P.D no	Nidra		Kosta	Agni	Work involving stress	Difficulty		Frequent fall	Urine	
		Normal	Disturbed				Writing	Eating		Frequency	Urgency
1	35969	+	-	K	T	-	-	-	-	-	-
2	37765	-	+	Mr	Md	-	-	+	+	+	+
3	40927	+	-	Mr	Md	-	-	-	-	-	+
4	02237	-	+	M	Md	-	-	-	-	-	-
5	13950	-	+	M	V	-	+	-	-	-	-
6	17011	-	+	M	S	-	+	-	+	-	+
7	17068	+	-	M	Md	-	-	-	-	-	-
8	22814	+	-	M	Md	-	-	-	+	-	+
9	24501	+	-	M	S	-	-	-	-	-	+
10	25076		+	M	S	+	+	-	-	-	-
11	25097	+	-	M	S	-	+	-	+	-	-
12	25118	-	+	M	V	-	+	+	-	-	-
13	27545	+	-	Mr	Md	-	-	-	-	-	-
14	29351	-	+	Mr	S	-	-	-	-	-	-
15	29823	-	+	Mr	V	-	+	+	-	-	-
16	31235	-	+	Mr	Md	-	-	+	+	-	+
17	33122	+	-	M	S	-	-	-	-	+	-
18	33709	-	+	M	S	-	+	+	-	-	-
19	34211	-	+	M	S	-	+	-	-	-	+
20	34674	+	-	Mr	Md	-	-	-	-	+	-

Agni = V-vishama, Md - Mandagni, T- Tikshna , S-Samagni **Koshtha=** K- krura, M-Madhyama, Mr- Mrudu

Table No 49 -Showing demographic data in patients

Sr. No	O.P.D. No	Prakriti	Sara	Samhanana	Satmya	Satwa	Vyayama Shakti	Vaya	Pramana	Ahara shakti	
										Aby	Jr
1.	35969	VP	At	Ss	P	P	P	M	Su	M	M
2.	37765	VP	At	M	M	M	M	M	Su	M	A
3.	40927	V	At	M	A	A	A	Vr	H	M	M
4.	02237	VP	At	M	M	M	M	M	Su	A	M
5.	13950	VK	At	M	M	M	M	M	Su	M	M
6.	17011	VP	Mm	M	M	M	M	M	Su	M	M
7.	17068	VP	At	M	M	M	M	M	Su	M	M
8.	22814	VP	At	M	M	P	P	M	Su	M	M
9.	24501	VP	At	M	P	P	M	M	Su	P	M
10.	25076	VP	At	M	P	P	M	M	Su	M	M
11.	25097	VP	Mm	M	M	M	M	M	Su	M	M
12.	25118	VP	At	M	P	M	M	Vr	Su	M	M
13.	27545	VK	Me	M	P	M	M	M	Su	M	M
14.	29351	VK	At	M	M	P	M	M	Su	M	M
15.	29823	VP	At	H	A	A	A	Vr	H	M	M
16.	31235	VP	At	M	M	M	M	Vr	Su	A	M
17.	33122	VK	At	M	P	M	M	M	Su	M	M
18.	33709	VP	Me	M	M	M	M	M	Su	M	M
19.	34211	VP	At	M	M	M	M	Vr	Su	M	M
20.	34674	VP	At	S	M	M	M	M	Su	M	M

V-vata, P-pitta, VP-Vatapitta, VK- vatakapha, PK- pittakapha, A-Avara, M-Madhyama,P-Pravara H-Heena, B-Balya, Vr-Vrudha, Su-Suparimanita Aharashakti = Aby – Abyavarana , Jr- Jarana,Ss – Susamhita At – Asthi ,Mm-Mamsa,Me-Medha

Table No 50-Showing Pradhana vedana in patients

Sl no	OPD no	Kampa									
		Head	Tongue	lips	Chin	UE		LE		One side of body	Complete body
						right	left	right	left		
1	35969	-	-	-	-	+	-	-	-	-	-
2	37765	-	+	-	-	+	-	+	-	+	-
3	40927	-	-	-	-	-	+	-	-	-	-
4	02237	-	-	-	-	-	+	-	-	-	-
5	13950	-	-	-	-	-	+	-	-	-	-
6	17011	-	-	-	-	+	-	-	-	-	-
7	17068	-	-	-	-	-	+	-	-	-	-
8	22814	-	-	-	-	+	+	-	-	-	-
9	24501	-	-	-	-	-	+	-	-	-	-
10	25076	-	-	-	-	+	+	-	-	-	-
11	25097	-	-	-	-	-	+	-	-	-	-
12	25118	-	-	-	-	+	-	-	-	-	-
13	27545	-	-	-	-	-	+	-	+	+	-
14	29351	+	-	-	-	+	+	-	-	-	-
15	29823	-	+	-	-	+	+	+	+	-	+
16	31235	+	-	-	-	-	+	-	+	+	-
17	33122	-	-	-	-	-	+	-	-	-	-
18	33709	-	-	-	-	+	-	-	-	-	-
19	34211	-	-	-	-	+	+	-	-	-	-
20	34674	-	-	-	-	-	+	-	-	-	-

Table No 51 -Showing Pradhana vedana in patients

Sl no	OPD no	Chesta sanga						
		Slow movement	stooped	Difficulty to begin walk	Small hand writing	Decreased facial expression	Difficulty in voluntary movements	Soft speech
01	35969	-	+	+	-	-	+	+
02	37765	+	+	+	-	+	+	-
03	40927	+	-	+	-	+	-	-
04	02237	+	-	-	-	-	-	-
05	13950	+	-	-	+	-	-	-
06	17011	-	-	+	+	-	-	-
07	17068	-	-	-	-	-	+	-
08	22814	-	-	+	-	-	+	-
09	24501	+	-	-	-	-	-	-
10	25076	+	-	-	+	-	+	+
11	25097	+	-	+	+	-	-	-
12	25118	+	-	-	+	-	-	-
13	27545	+	+	-	-	-	-	+
14	29351	-	-	-	-	-	+	-
15	29823	+	+	+	+	+	+	+
16	31235	+	+	-	-	+	-	+
17	33122	+	-	-	-	-	-	+
18	33709	-	-	-	-	-	-	+
19	34211	-	-	+	+	-	+	-
20	34674	+	-	-	-	-	-	+

Table No52-Showing Pradhana vedana in patients

Sl no	OPD no	**Sthamba**					**Avanamana**
		Neck	UE		LE		
			right	left	right	left	
1.	35969	-	+	-	-	-	-
2.	37765	-	+	-	+	-	+
3.	40927	-	-	+	-	-	-
4.	02237	-	-	-	-	-	-
5.	13950	-	-	+	-	-	-
6.	17011	-	+	-	-	-	-
7.	17068	-	-	+	-	-	-
8.	22814	-	+	+	-	-	+
9.	24501	-	-	+	-	-	-
10.	25076	-	+	+	-	-	-
11.	25097	-	-	+	-	-	-
12.	25118	-	+	-	-	-	-
13.	27545	-	-	+	-	-	-
14.	29351	-	+	+	-	-	-
15.	29823	-	+	+	-	-	+
16.	31235	-	-	+	-	+	+
17.	33122	-	-	+	-	-	-
18.	33709	-	-	-	-	-	-
19.	34211	-	+	+	-	-	-
20.	34674	-	-	+	-	-	-

UE-upper extremity. LE- lower extremity.

Table No 53 -Showing Anubandha Vedhana wise data in patients

Sl no	OPD no	Bhavarahita mukha		Swara bheda									
		Reduced facial expression	staring	Slow speech	Low volume speech	monotone	Difficulty in speaking	vishada	gatisanga	vibandha	Calf muscles tightness	salivation	Sexual dysfunction
	35969	-	-	-	-	+	-	+		+	-	-	-
	37765	+	-	-	-	-	-	+	-	-	+	-	-
	40927	+	-	-	+	-	-	-	-	+	+	-	-
	02237	-	-	-	-	-	-	-	-	-	+	-	-
	13950	-	-	-	-	+	-	-	-	+	-	-	-
	17011	-	-	-	+	-	-	-	-	+	+	-	-
	17068	-	-	-	+	-	-	-	-	+	-	-	-
	22814	-	-	-	-	+	-	-	-	+	+	-	-
	24501	-	-	-	+	-	-	-	-	+	+	-	-
	25076	-	-	+	+	-	-	+	+	-	-	-	-
	25097	-	-	+	-	-	-	-	-	+	-	-	-
	25118	+	-	-	-	-	-	-	-	+	+	-	-
	27545	+	-	-	+	-	-	-	-	+	+	-	+
	29351	-	-	-	-	-	-	+	-	-	-	-	-
	29823	+	-	+	+	+	+	+	+	+	+	+	-
	31235	+	-	-	+	-	-	+	-	+	+	-	-
	33122	-	-	-	+	-	-	+		+	-	-	-
	33709	-	-	+	+	-	-	+	+	+	-	-	-
	34211	-	-	-	-	-	-	-	-	-	+	-	-
	34674	-	-	-	+	-	-	+		+	+	-	-

Table No 54-Showing subjective parameters data in patients

sl no	Opd no	Kampa		Gatisanga		Vakvikruti		Stamba		Avanamana		Chestasanga	
		BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1	35969	01	00	01	00	02	01	01	00	00	00	02	02
2	37765	01	00	02	01	02	02	02	01	02	02	02	02
3	40927	01	01	01	01	02	02	01	01	00	00	01	01
4	02237	01	00	01	01	02	01	00	00	00	00	01	00
5	13950	01	00	01	00	01	00	02	01	00	00	01	00
6	17011	01	00	02	01	02	01	01	00	00	00	01	00
7	17068	01	01	01	00	02	01	01	00	00	00	01	00
8	22814	02	01	01	00	01	01	02	00	02	01	02	00
9	24501	01	00	01	01	02	01	01	00	00	00	01	00
10	25076	02	01	01	00	01	01	01	00	00	00	02	00
11	25097	01	00	01	00	01	01	01	00	00	00	02	01
12	25118	01	00	01	00	01	01	02	01	00	00	02	00
13	27545	01	00	01	01	01	01	01	00	02	02	01	01
14	29351	02	00	01	00	02	01	00	00	00	00	01	00
15	29823	03	02	01	01	02	02	01	01	02	02	03	02
16	31235	01	01	01	01	02	01	02	01	01	01	02	02
17	33122	01	00	01	00	01	01	01	00	00	00	02	01
18	33709	01	00	02	01	00	00	00	00	00	00	01	01
19	34211	02	01	00	00	00	00	02	00	00	00	01	00
20	34674	01	00	01	00	02	01	01	00	00	00	01	01

Table No 55 -Showing Objective parameters data in patients

sl no	Opd no	Tremors		Rigidity		Bradykinesia		Gait		Dressing		Postural stability	
		BT	AT	BT	AT	BT	AT	BT	AT	BT	AT	BT	AT
1	35969	02	00	02	00	02	02	01	00	02	01	00	00
2	37765	03	01	02	01	03	03	02	02	02	02	01	01
3	40927	03	01	02	02	02	02	01	01	02	01	01	01
4	02237	02	00	00	00	01	00	01	01	00	00	00	00
5	13950	02	00	02	01	02	00	00	00	02	00	00	00
6	17011	02	00	02	00	02	00	01	00	02	01	02	01
7	17068	03	03	02	00	02	00	01	00	01	00	01	01
8	22814	03	02	02	00	03	00	01	01	02	01	00	00
9	24501	02	00	02	00	02	00	02	01	01	01	00	00
10	25076	03	02	02	00	03	00	02	01	01	01	01	00
11	25097	02	00	02	00	02	01	02	01	01	00	02	01
12	25118	03	00	02	01	02	00	01	01	01	00	01	01
13	27545	03	00	02	00	01	01	01	01	01	01	01	01
14	29351	02	00	00	00	00	00	00	00	00	00	00	00
15	29823	04	03	04	04	03	02	02	02	02	02	01	01
16	31235	03	03	02	01	01	01	03	02	02	01	01	01
17	33122	02	00	01	00	01	01	02	00	01	00	00	00
18	33709	02	00	00	00	01	01	00	00	01	00	01	01
19	34211	02	01	02	00	01	00	00	00	01	00	00	00
20	34674	02	00	01	00	01	01	01	00	01	01	00	00

Figure No 44. Showing Mean relief of all parameters

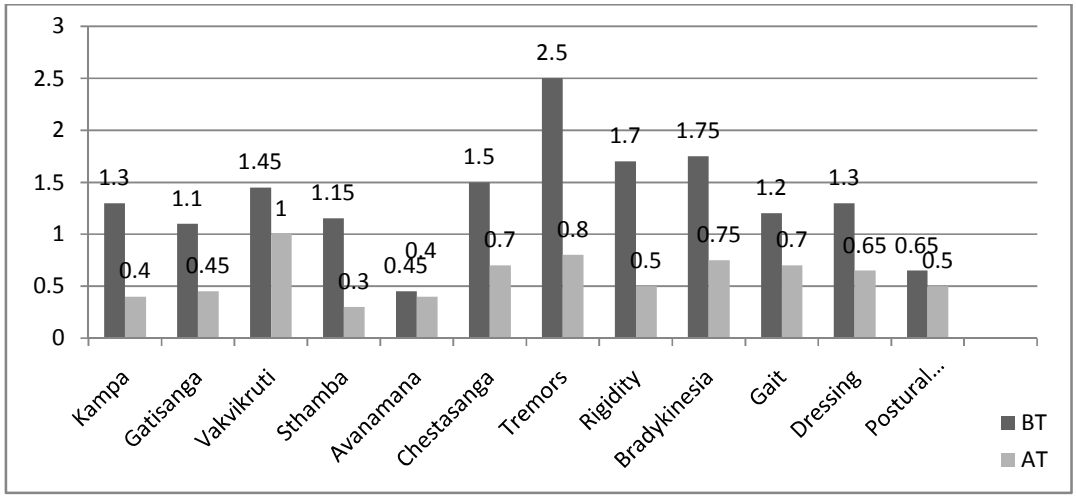


Table No. 56 Kampa Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Kampa Before Treatment	1.30	20	0.571	0.128
	Kampa After Treatment	0.40	20	0.598	0.134

Table No. 57 Kampa Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	Kampa Before Treatment & Kampa After Treatment	20	0.708	0.000

Table No. 58 Kampa Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	**Kampa** Before Treatment **Kampa** After Treatment	0.900	0.447	0.100	0.691	1.109	9.000	19	0.000

Conclusion: The Statistical analyses done by using paired t-test by using SPSS Software-15 version.

From table 56, mean of parameter Kampa, Before Treatment was 1.30 with S.D. 0.571 is reduced to 0.40 with S.D. 0.598 after treatment. The correlation from table 57 between before and after treatment is 0.708 which is moderately positively correlated and significant at 0.00. From table 58, the treatment shows more highly significant before and after treatment at0.000.

Table No. 59 Gatisanga Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Gatisanga** Before Treatment	1.10	20	0.447	0.100
	Gatisanga After Treatment	0.45	20	0.510	0.114

Table No. 60 Gatisanga Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Gatisanga** Before Treatment & **Gatisanga** After Treatment	20	0.484	0.031

Table No. 61 Gatisanga Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	**Gatisanga** Before Treatment **Gatisanga** After Treatment	0.650	0.489	0.109	0.421	0.879	5.940	19	0.000

From table 59, mean of parameter *Gatisanga* Before Treatment was 1.10 with S.D. 0.447 is reduced to 0.45 with S.D. 0.510 after treatment. The correlation from table 60 between before and after treatment is 0.484 which is low degree positively correlated and significant at 0.031. From table 61, the treatment shows more highly significant before and after treatment at 0.000

Table No. 62 Vakvikruti Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Vakvikruti** Before Treatment	1.45	20	0.686	0.153
	Vakvikruti After Treatment	1.00	20	0.562	0.126

Table No. 63 Vakvikruti Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Vakvikruti** Before Treatment & **Vakvikruti** After Treatment	20	0.682	0.001

Table No. 64 Vakvikruti Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Vakvikruti Before Treatment - Vakvikruti After Treatment	0.450	0.510	0.114	0.211	0.689	3.943	19	0.001

From table 62, mean of parameter *Vakvikruti* Before Treatment was 1.45 with S.D. 0.686 is reduced to 1.00 with S.D. 0.562 after treatment. The correlation from table 63 between before and after treatment is 0.682 which is moderately positively correlated

and significant at 0.031. From table 64 the treatment shows more highly significant before and after treatment at 0.001

Table No. 65 Stamba Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Stamba** Before Treatment	1.15	20	0.671	0.150
	Stamba After Treatment	0.30	20	0.470	0.105

Table No. 66 Stamba Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Stamba** Before Treatment & **Stamba** After Treatment	20	0.517	0.019

Table No. 67 Stamba Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Stamba** Before Treatment **Stamba** After Treatment	0.850	0.587	0.131	0.575	1.125	6.474	19	0.000

From table 65, mean of parameter *Stamba* Before Treatment was 1.15 with S.D. 0.671 is reduced to 0.30 with S.D. 0.470 after treatment. The correlation from table 66 between before and after treatment is 0.571 which is moderately positively correlated and significant at 0.091. From table 67, the treatment shows more highly significant before and after treatment at 0.000

Table No. 68 Avanamana Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Avanamana** Before Treatment	0.45	20	0.826	0.185
	Avanamana After Treatment	0.40	20	0.754	0.169

Table No. 69 Avanamana Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Avanamana** Before Treatment & **Avanamana** After Treatment	20	0.964	0.000

Table No. 70 Avanamana Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Avanamana** Before Treatment **Avanamana** After Treatment	0.050	0.224	0.050	-0.055	0.155	1.000	19	0.330

From table 68, mean of parameter Avanamana before Treatment was 0.826 with S.D. 0.45 is reduced to 0.40 with S.D. 0.754 after treatment. The correlation from table 69 between before and after treatment is 0.964 which is high degree positively correlated and significant at 0.000.

From from table 70, the treatment shows not significant before and after treatment as 0.330

Table No.71 Chestasanga Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Chestasanga** Before Treatment	1.50	20	0.607	0.136
	Chestasanga After Treatment	0.70	20	0.801	0.179

Table No. 72 Chestasanga Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Chestasanga** Before Treatment & **Chestasanga** After Treatment	20	0.541	0.014

Table No.73 Chestasanga Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Chestasanga** Before Treatment - **Chestasanga** After Treatment	0.800	0.696	0.156	0.474	1.126	5.141	19	0.000

From table 71, mean of parameter Chestasanga, Before Treatment was 1.50 with S.D. 0.607 is reduced to 0.70 with S.D. 0.801 after treatment. The correlation from table 72 between before and after treatment is 0.541 which is moderately positively correlated and significant at 0.014.

From table 73, the treatment shows highly significant before and after treatment at 0.000

Table No. 74 Tremors Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Tremors** Before Treatment	2.50	20	0.607	0.136
	Tremors After Treatment	0.80	20	1.152	0.258

Table No.75 Tremors Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Tremors** Before Treatment & **Tremors** After Treatment	20	0.753	0.000

Table No. 76 Tremors Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Tremors** Before Treatment - **Tremors** After Treatment	1.700	0.801	0.179	1.325	2.075	9.488	19	0.000

From table 74, mean of parameter Tremors before treatment was 2.50 with S.D. 0.607 is reduced to 0.80 with S.D. 0.80 after treatment. The correlation from table 75 between before and after treatment is 0.753 which is moderately positively correlated and significant at 0.000.

From table 76 the treatment shows highly significant before and after treatment at 0.000

Table No. 77 Rigidity Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Rigidity** Before Treatment	1.70	20	0.923	0.206
	Rigidity After Treatment	0.50	20	1.000	0.224

Table No. 78 Rigidity Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Rigidity** Before Treatment & **Rigidity** After Treatment	20	0.627	0.003

Table No. 79 Rigidity Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Rigidity** Before Treatment - **Rigidity** After Treatment	1.200	0.8340	0.186	0.810	1.590	6.439	19	0.000

From table 77, mean of parameter Rigidity Before Treatment was 1.70 with S.D. 0.923 is reduced to 0.50 with S.D. 1.00 after treatment. The correlation from table 78 between before and after treatment is 0.627 which is moderately positively correlated and significant at 0.003.

From table 79, the treatment shows highly significant before and after treatment at 0.000

Table No. 80 Bradykinesia Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Bradykinesia** Before Treatment	1.75	20	0.851	0.190
	Bradykinesia After Treatment	0.75	20	0.910	0.204

Table No. 81 Bradykinesia Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Bradykinesia** Before Treatment & **Bradykinesia** After Treatment	20	0.255	0.278

Table No. 82 Bradykinesia Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Bradykinesia** Before Treatment - **Bradykinesia** After Treatment	1.000	1.076	0.241	0.496	1.504	4.156	19	0.001

From table 80, mean of parameter Bradykinesia Before Treatment was 1.75 with S.D. 0.851 is reduced to 0.750 with S.D. 0.910 after treatment. The correlation from table 81 between before and after treatment is 0.255 which is low degree positively correlated and significant at 0.278.

From table 82, the treatment shows highly significant before and after treatment at 0.001

Table No. 83 Gait Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Gait** Before Treatment	1.20	20	0.834	0.186
	Gait After Treatment	0.70	20	0.733	0.164

Table No. 84 Gait Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Gait** Before Treatment & **Gait** After Treatment	20	0.707	0.000

Table No. 85 Gait Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Gait** Before Treatment - **Gait** After Treatment	0.500	0.607	0.136	0.216	0.784	3.684	19	0.002

From table 83, mean of parameter Gait, Before Treatment was 1.20 with S.D. 0.834 is reduced to 0.70 with S.D. 0.733 after treatment. The correlation from table 84 between before and after treatment is 0.707 which is high degree positively correlated and significant at 0.000.

From table 85, the treatment shows highly significant before and after treatment at 0.00

Table No. 86 Dressing Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Dressing** Before Treatment	1.30	20	0.657	0.147
	Dressing After Treatment	0.65	20	0.671	0.150

Table No. 87 Dressing Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Dressing** Before Treatment & **Dressing** After Treatment	20	0.609	0.004

Table No. 88 Dressing Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	**Dressing** Before Treatment -**Dressing** After Treatment	0.650	0.587	0.131	0.375	0.925	4.951	19	0.000

From table 86, mean of parameter Dressing, Before Treatment was 1.30 with S.D. 0.657 is reduced to 0.65 with S.D. 0.671 after treatment. The correlation from table 87 between before and after treatment is 0.609 which is moderately positively correlated and significant at 0.004.

From table 88, the treatment shows highly significant before and after treatment at 0.000

Table No. 89 Postural stability Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	**Postural stability** Before Treatment	0.65	20	0.671	0.150
	Postural stability After Treatment	0.50	20	0.513	0.115

Table No. 90 Postural stability Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	**Postural stability** Before Treatment & **Postural stability** After Treatment	20	0.841	0.000

Table No. 91 Postural stability Paired Samples Test

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95%ConfidenceInterval of the Difference Lower	Upper			
Pair 1	**Postural stability** Before Treatment - **Postural stability** After Treatment	0.150	0.366	0.082	-0.021	0.321	1.831	19	0.083

From table 89, mean of parameter dressing before Treatment was 0.65 with S.D. 0.671 is reduced to 0.50 with S.D. 0.115 after treatment. The correlation from table 90 between before and after treatment is 0.841 which is high degree positively correlated and significant at 0.000.

From table 91, the treatment shows not significant before and after treatment as P>0.05

Based on the analysis the conclusion can be drawn as, the treatment is more highly significant in the parameters*, Kampa, Gatisanga, Stamba*, Chestasanga, *Tremors and Rigidity*. In the parameters *Stamba* and *Chestasanga* have same effects with positive correlation between before and after treatment. In the parameters *Gait, Vakvikruti* and *Bradykinesia* treatment has less significant. In the parameters *Avanamana and Postural stability* treatment not significant even they positive correlation before and after treatment. This may be because of the involvement of whole vertebral column which cannot be corrected with medical management.

Healthy discussion paves a way to the generation of thoughts that guides us in a new dimension for the better evaluation of the problem. At this juncture some of the aspects of the present disease are discussed. Discussion is the most important part of any research where the observations are discussed and given reasons by the researcher. The significant results and insignificant results will be discussed in the same section with reasons. Hence it becomes important to discuss the clinical study in detail.

Discussions on this study are made under the following headings

I. Discussion on Disease.

II. Discussions on Drug

III. Discussions on the observation of patients

IV. Discussions on Results of clinical trial

I. Discussion on Disease Review

The present study has been entitled as "A clinical study on Kampavata (Parkinson's disease) and its management with Triguna ras."[183] The 'Vata' which is the motivator and controller of other two doshas, is responsible for the manifestation of almost all diseases. Major neurological problems come under vata vyadhis. Kampavata as one among them manifests with Dehabhramana (postural instability), "Karapada tale kampa" (tremors in hands and legs), Matiksheena (dementia), and Nidrabhanga (sleeplessness). [184] There are many vata vyadhis commonly seen but Kampavata is one of the rare mentioned under vata vyadhis because of its crippling nature and non availability of curative treatment this disease has remained a great problem in the ageing society which usually affects after the age of 50 years. The disease is increasing in its frequency with the world population showing an incidence of 1-2 per 1000 population and has an equal sex distribution.

According to Ayurveda, Kampavata is a Vata Nanatmaja vikara. During the period of Charaka and Sushruta clinical manifestations of kampavata like kampa, sthamba, chestasanag, vakvikriti etc was not explained as one disease instead explained under various contexts majority of the symptoms of kampavata were found in kaphavrita udana and kaphavrita vyana but no single avarana process completely covers the symptoms of kampavata. It is therefore suggested that in view of classical reference, a complete clinical entity having symptoms, signs etc. the term Kampvata the most appropriate term, for the first time explained by Basavarajiyam with clinical features similar to that of Parkinson's disease.

Direct reference to the Parkinson's disease in the ancient Ayurvedic literature is sparse and refers only to related symptoms including tremors. Thus, the condition is referred to the modern ayurvedic literature by various names for kampavata (tremors due to vata), vepthu (shaking), prevepana (excessive shaking), shirakampa (head tremor), spandin (quivering) and kampana (tremors). The basic pathologic change is degeneration of a group of nerve cells deep within the centre of the brain in an area called substantia nigra. These cells use Dopamine as their neurotransmitter to signal other nerve cells. As these cells degenerate and, stop functioning, dopamine fails to reach the areas of the brain that affect motor functions. Parkinson's disease remains the only neurodegenerative disorder that has demonstrated significant responsiveness to therapeutic intervention. However the treatments which are present now have a little evidence that this treatment changes the course of disease.[185] Parkinson's disease has remained as a great problem though in modern medical science lot of research works have been done. Some medications like Carbidopa, Levodopa, some neurosurgeries like thalamotomy, subthalamotomy, some brain stimulation technique like thalamic stimulation, subthalamic stimulation are used for Parkinson's disease but, no therapy is

present which stops the progress of this disease. The goal of treatment is to provide the best possible function and quality of life over the long term. In this regard the study is undertaken for management of kampavata with Triguna rasa. The management of Kampavata can be done by the oral administration of Triguna Rasa which is explained in sahasrayoga[186] as the karma of Haritaki over nadivaha samsthan is balya and medhya indicated in conditions like mastishkya dourbalya, nadidourbalya and best in all vata vyadhis,[187] kajjali (parada and gandhaka) mitigates all types of roga and even tridoshas.[188]

NIDANA PANCHAKA

The Nidana Panchaka is nothing but a full horoscope of a disease, right from the indulgence in the causative factor up to the complete manifestation of the disease including prognosis of the disease. The perfect knowledge of which is very much essential for a proper diagnosis and a line of treatment.

NIDANA:

According to Ayurveda, consideration of etiological factors is important for the diagnosis, prognosis and line of treatment. Nidanas refers to all the causative factors which are responsible for the initiation and progress of the disease process. The general etiological factors which provoke Vata[189] need contemplation in this context. However, a careful Search of Samhita furnishes specific factors responsible for the symptoms i.e. Stambha, Vepathu and Vinamana which are included in Kampavata. Kampavata as a Vatika disorder, so the causative factors which provoke Vata, can be considered as etiological factors of Kampavata. Use of alpa laghu ,rooksha, sheeta, Katu, Kashaya, Tikta, Masoora,Mudga, Raktashali, Rajamasha, Shyama, Yavagu, Vishamashana, Atibhukta, Abhojana, Langhana, Adhovata Mutra Pureesha rodha, Ratri jagarana and manasika nidanas like Chinta, Bhaya, Dukha Krodha Shoka.

PURVARUPA

Kampavata as one of vata vyadhi which doesn't have any purvarupa as Acharya Charaka says avyakta lakshana is the purvaupa of vata vyadhi.[190] Kampavata as one among vata vyadhi doesn't have any purvarupa but following may be considered as Purvarupa of Kampavata which are Klama, Shrama, Angamarda, Smritihani, Anavasthita, Chittatva, Gatraruka and Vishada.

RUPA

The signs and symptoms produced in an individual as a result of sequential changes in the disease process can be studied under the heading "Roopa". Basavarajeeyam has explained the symptoms of Kampavata [191]as Karapadatale Kampa (i.e. tremor in hands and feet), Dehabhramana (Rombergism), Nidrabhanga (disturbed sleep) and Matiksheena (dementia). Certain symptoms like Stambha (Rigidity), Cestahani (Slowness of the movement), Vinaman (Flexed posture), Vakvikriti (Speech disorders). Other than these above mentioned symptoms they have described as rigidity, bradykinesia, monotonous speech, postural instability, nocturia and constipation.

Tremor is of the main symptoms associated with Parkinson's disease. However, contrary to popular belief, it is not universal and approximately one-quarter of patients do not have tremor. The involuntary rhythmical shaking normally occurs at rest and tends to reduce or stop when the affected part is used for some activity. The tremor of Parkinson's disease is quite coarse with a frequency usually between 4 and 6 Hz. Although the hands are often affected, some patients experience tremor of the jaw or foot. The tremor affecting the thumb and first finger produces the commonly called 'pill rolling' effect.

Rigidity is an important feature of Parkinson's disease. Rigidity is actually hyper tonicity of muscles. In rigidity muscles become continuously or intermittently firm and

tense. Rigidity is detected clinically by resistance to passive manipulation of the limbs, neck or trunk, the rigidity, or muscular stiffness occurring with Parkinson's disease exacerbates the problems with movement resulting from hypokinesia and bradykinesia. All muscle groups can become affected the increase in muscle resistance occurs when there is passive movement; the resistance to passive movement is constant throughout the range of movement, unlike spasticity where sudden relaxation can occur after movement has begun. If the patient also suffers with tremor, the so-called cog wheeling effect can be seen. This jerky movement results from the tremor superimposed on top of the rigidity. The rigidity associated with Parkinson's disease is also often asymmetrical at onset.

Bradykinesia is defined as slowness or poverty of movement with loss of automatic stereotyped movements. It is perceived by the patient as a slowing of his ability to perform the usual activities of daily living such as bathing, dressing, rising from chair, turning over in bed, making buttoning, shaving etc. Bradykinesia accounts for many of the characteristic features of Parkinson's disease such as the expression less face (mask face), a decrease and subsequent loss of blinking, reduced arm swing while walking, small cramped hand writing (micrographia), monotonous speech, and loss of expressive gestures of hand.

A steady *flexed posture* is normally maintained by the nervous system making continuous reflex adjustments. Impairment of these mechanisms leads to a reduced ability to maintain balance, making the patient less steady when walking and particularly when turning.

Gait symptoms are a common feature of Parkinson's disease, but usually occur around five years after initial diagnosis. Parkinson's disease patient generally takes small shuffling steps, difficulty in beginning to walk and to stop walking once started

leads to the so called "festinating gait" or "hurrying gait" i.e. invincible propensity to run, when wishing only to walk.

Decrease in muscle movement of the larynx can reduce the volume and articulation of *speech* making it difficult for others to understand what is being said. This is compounded by the tendency for phrases to be said in a rush, the patient being unable to control the speed of delivery. Sometimes, long silences occur as a patient has difficulty starting the beginning of a sentence or new phrase.

Loss of sleep is another symptom nearly all patients with Parkinson's disease report. In the majority of cases, problems are the result of limb movements, myoclonic jerks or leg cramps. Serious cognitive impairment, adverse effects on judgment, reasoning, visual hallucinations reduced short-term memory and confusion occurs in about one-fifth of patients with advanced Parkinson's disease which are key features of *dementia*.

Constipation as the result of reduced stool transit in the colon, Pelvic floor muscle dystonia may affect the rectum and anus, which, instead of relaxing when trying to pass a stool, goes into spasm, but it is usually attributed to the combined effects of sedentary life and diminished food and fluid intake.

Dysphagia (difficulty in swallowing) is a common problem in up to one half of patients, especially those in the more advanced stages of Parkinson's disease. *Micrographia,* writing that is very small sometimes to the point of being unreadable, writing becomes smaller and smaller the longer the patient writes which are other features of the disease. [192]

SAMPPRAPTI

Samprapti of vatavyadhi is complex process to understand. Acharya Madhava while stating the 'vatavyadhi' has explained "*vikruta vatajanito asadharano vyadhihi*

vatavyadhi,"[193] vatavyadhi is manifested due to vikruta vata and is asadharana in nature. Though Kampa is mentioned under the heading of vataja nanatmaja vikaras, samprapti for Kampavata is not explained separately, so the general samprapti of vatavyadhi can be considered.

Due to the etiological factors, as mentioned the Vata gets aggravated by its chala, ruksha and sheeta properties. Prana, Udana and Vyana are most affected among the five types of vata, which in turn vitiate the Mastulunga Majja in the Shiras, because of Srotovaigunya present at Shiras. As Mastishka is Sneha pradhana, vitiated Vayu impairs this Sneha by its ruksha, sheeta and laghu guna. These vitiated doshas selectively affect the Vata vaha srotas in the Mastishka leading to impairment in the motor functions of the body leading to Kampa, Dehabhramana, Matiksheena etc.

The Pathology of Parkinson's disease is Degeneration of Substantia nigra, loss of at least 60% of dopaminergic neurons and presence of Lewy bodies in surviving neurons of the Substantia nigra.

Parkinson's disease results from degeneration of the dopaminergic pathway from the substantia nigra to the corpus striatum. The essential lesion is one of an idiopathic degeneration of the pigmented neurons of the brain stem seen most convincingly in the substantia nigra resulting in depigmentation of these brain stem nuclei. Thus the projection of the nigral neurons to the corpus striatum is destroyed so that their modifying influence on this corpus striatum resulting in the clinical picture of rigidity and tremor

UPASHAYA AND ANUPASHAYA

The factors that aggravate vata can be considered as anupashaya and that which pacifies vata can be considered as upashaya.

SADHYASADHYATA

Most of our acharyas consider shuddha vataja vyadhi to be asadhya or krichrasadhya. So Kampavata being one of the shuddha vata vyadhi, is also krichrasadhya / asadhya for chikitsa. Parkinson's disease is a progressive disorder but its rate of progression is variable. The exact prognosis for an individual patient is difficult to predict precisely.

II. DISCUSSION ON DRUG REVIEW:

Indication of Triguna rasa in arohana and avarohana krama orally is said for Kampavata in gutika parkarana of sahasrayoga of which the study is intended.[194] Triguna rasa has three ingredients they are Parada, Gandhaka and Haritaki in 1 :8 :9 ratio respectively. Kajjali given with different herbal formulations and with different anupanas is sarvamayahara Brimhankaraka, Veerya-vardhaka and Tridoshahara.[195] Haritaki it purifies Dhatus and alleviates doshas on them acts as Rasayana and Vayasthapana as the karma of Haritaki over nadivaha samsthan is balya and medhya indicated in conditions like mastishkya dourbalya, nadidourbalya and best in all vata vyadhisi[196].

III DISCUSSION ON GENERAL OBSERVATIONS

AGE

Symptoms of Parkinson's disease can appear at any age, but the average age of onset is 60. Parkinson's is rare in people younger than 30 years of age, and risk for the disease increases with age. Parkinson's disease is common in the elderly and affects one person in 20 over the age of 80. In this study, maximum patients were in between the age group of 50 - 70. The data supports that this disease is seen after 50 years of age. Dalhana has considered Kampa as Kala Bala Pravritta disease and Vagbhata has

mentioned tremors and flexion postures of the body under the symptomatology of ageing [197]

Though the incidence in this study is seen in early of age 50, because of life style modification, improper diet, exposure to triggering factors and stress full life may contribute the early manifestation of disease.

GENDER

Both the sex found to be affected with Parkinson's disease. But in this study male patients were more (75%) than female (25%) patients. Usually males are more prone to disease as their disease course seems to be similar, including age at death, which one would not expect given women's longer life expectancy. As sample is small it may be the reason why female ratio is less.

RELIGION

Maximum number of patients belongs to Hindu community (95%) due to majority of Hindus in Gadag. Disease prevalence is not having any significance to race and religion.

SOCIOECONOMIC STATUS

In the present study more number of patients i.e. 70% was belonging to middle class and 15% from Poor. It can be inferred from the study that social status of the patient does not have any significance in this disease. This fact doesn't hold any contributory factor for disease prevalence. The study was carried in remote area where more people are of middle class.

OCCUPATION

In the present study 30% patients were labor, 45% were sedentary life style, and 25% people were of active and this does not carry any significance in the production of the disease. In the study people of labor were more among the agriculture

field who were exposed to Rural living, farming, gardening, pesticide use, or well-water drinking have been associated with PD and sedentary lifestyle were most among the urban area who had irregular diets and much mental stress and even exposed to harmful gases like smoke of vehicles etc

DIET

Most of the patients were vegetarian, (80%) and 20 % were mixed. It may be because in this region of Gadag most of the people are vegetarian. 85 % patients were using katu rasa pradhana ahara rasa and (30%) of Kashaya rasa and 04 (20%) patients consumed tikta rasa so the data which has highest consumption of katu rasa signifies it causes vata vriddi. Diet is a very difficult exposure to measure because of its complexity and the fact that most individuals have diets that are qualitatively relatively similar. Despite these challenges, several dietary factors have been associated with PD. A diet that is high in antioxidants has been proposed to lower the risk of PD, but to date such a diet has not been consistently associated with this lower risk. On the other hand, positive associations of PD with animal fat consumption and with a diet that is high in iron have been reported. Excess intake of dairy products has been associated with increased risk of PD in two large prospective. It could not be determined whether the effect was due to calcium or milk. Moreover, the risk was most marked in men and not clearly observed in women.

MARITAL STATUS

Majority of the patients were married, this may be due to increased prevalence of the disease in adolescence and middle age.

DIAGNOSIS

In this study 13 (65%) patients were newly diagnosed and 07 (35%) patients were previously diagnosed cases.

ADDICTION

In this study 04 (20%) patients had habit of consuming alcohol and 04 (20%) patients had habit of smoking. Observation of addiction in present series revealed that very less patients were addicted to alcohol and smoking. 20% of the patients were addicted to smoke bidi or cigarette and 20 % people addicted to alcohol. Smoking is associated inversely Parkinson's disease. Smoking has a dose-response relationship with Parkinson's disease. Current heavy smokers have a lower risk than current light smokers and former heavy smokers who had recently quit. For smoking the cause effect relationship between the Parkinson's disease has not yet established.[198] Alcohol use has been found by some to be inversely associated with Parkinson's disease even after controlling for possible confounding by smoking. A biological explanation for this observation has not been articulated. One study found that fewer cases with Parkinson's disease had a diagnosis of alcoholism than controls. The variability across studies is great and, overall, the current evidence for an association between alcohol intake and risk of Parkinson's disease is weak.

NIDRA

In the present study 55% were suffering from disturbed sleep, Provoked Vata may be the reason of disturbed sleep. Basavarajiyam mentioned Nidranasha as one of the symptom of Kampavata. In old age most of people suffer with insomnia. In the present study nidranash observed may be the age related. In one patient it was found that because of repeated attack of involuntary tremor made patient get disturbed sleep.

PATTERN OF WORK

Stress at work was seen very less in the present study only 03 (15%) patients had stress at work. This may be due to most of patients were not working. There are reports indicating that people experiencing the extreme emotional and hardships at work

have been shown to have an increased risk for developing Parkinson's disease. These observations reflect an accelerated nigral injury as the result of stress-related increase in dopamine turnover with resultant increased oxidative injury, leading to manifestation of disease.

DIFFICULTIES IN CARRYING ROUTINE WORK

In the study 08(40%) people had difficulty in writing, 05(20%) people had difficulty in eating and 10(50%) patients had difficulty in carrying small movements, Parkinson's disease (PD) is a progressive neurological disorder characterised by a large number of motor and non-motor features that can impact on function to a variable degree as in above said data 10 patients were finding difficulties in carrying out their routine activities which suggest the involvement of the motor functions impairment. Micrographia is seen as early non specific feature of the disease, it suggests rigidity and bradykinesia. Slower and more effortful handwriting, with smaller letters (micrographia), writing that is very small sometimes to the point of being unreadable, writing becomes smaller and smaller the longer the patient writes.

HABITAT

Most of the patients i.e. 55 % were belonged to urban area. It may be possible due to constant exposure to pollution, certain gases like carbon monoxide; hydrocarbon which is environmental neurotoxins more affects the urban area than rural area. Another factor is stressful lifestyle of cities comparatively to rural area may also play a key role, and 45% were from rural among them most of them were agriculturists who were exposed for pesticides.

PRAKRUTI

Most of the patients were having Dwandaja Doshika constitution with majority of the patients i.e. 75% of Vatapitta Prakriti, 20% were of Vata-kaphaja and 5% vtaja

Prakriti. Vata Dosha was present in dominancy of deha prakruti which is the main causative factor for the disease. In classics it has been stated that Dwandva prakruti persons are more prone to disease. That's why Dwandva prakruti is said as Heena prakruti.

SATWA, SAMHANANA & SATMYA

The distribution of the patient's shows that majority of the patients were having Madhyama Satwa (65%) and 25% patients had pravara satwa and 10% patients had heena satwa. As most of the patients had early symptoms of the disease most of them had moderate mental faculties, predominant of rajo guna and even tolerated pain or odd situations of disease moderately. In this study patients were having moderately demarcated bones, knit joints and were moderately built and had moderate strength which accounted for Madhyama Samhanana (85%). Most of the patients in this surrounding area were accustomed to katu, amla, lavana, madhura, sushka, ruksha and ushna dravyas in excess which justifies Madhyma Satmya (60%).

VYAMASHAKTI

The distribution of the patient's shows that majority of the patients were having Madhyama Vyama Shakti 16 (90 %). As the patients were of older group most of them had moderate strength which was determined by their slow working process due to Bradykinesia.

AHARASHAKTI

In the Study maximum patients ie 17(85%) were having Madhyama ahara shakti. Patients were comfortable with normal regular diet and when consumed large amount of food had difficulties to digest the food properly which indicate the moderate strength of digestive fire.

JARANA SHAKTI

In this study 19 (95 %) patients had madhyama jarana shakti and were able to digest laghu ahara.

MANASIKA VRITTANTA

In the present study 04 (20%) patients had emotional stress, 12 (60%) patients had anxiety, 05 (25%) patients had depression, 01 (5%) patient had unusual laugh, 12 (60%) patients had (aggressiveness) in their nature, 02 (10%) patients had mada and 01 (5%) patient was suffering with panic. Charaka [199]has mentioned these manasika bhavas incite vata and even the concept has been propounded that stress triggers for initiation of disease.

CHRONICITY

Chronicity of Kampa in this study 10 (50%) patients were of acute onset of disease and another 10 (50%) were of chronic origin. Acute onset of sthamba was seen in 12 (60%) patients were as 07 (35%) patients were of chronic onset and 01 (5%) patient dint have sthamba symptom. 11 (55%) patients were suffering from Chestasanga of acute onset and 08 (40%) patients were of chronic onset and 01 (5%) patient was not having Chestasanga symptom. The above data signifies acute onset of symptoms was seen predominatly as the disease was in its early progression of the affected individuals. Though the equal distribution of acute and chronic onset doesn't clearly signifies the real onset. Parkinson's disease is one of chronic neuro degenerative disorder seen in old people. Because of small sample size in the present study, this fact not justified.

DISCUSSION ON PRADHANA VEDANA

History of onset of disease

In history of onset 10 patients were of acute onset and 10 of chronic onset, as the typical age at onset of Parkinson's disease is between 40 to 60 years but may vary

widely. The clinical course is chronic and Progressive, with severe disability attained after approximately 10 years. A smaller proportion of patients have a more rapidly progressive disease and yet a smaller group has slowly progressive disorder in which deterioration plateaus remain minimal for two to three decades. So the data supports the onset and progression.

TREMOR- KAMPA

In the present study maximum number of patients were having moderate tremor. There were no patients without tremor. It again proves that tremor is the principle symptom in Parkinson's disease. Tremor seen only in tongue 02 (10%) patients, head 02(10%) patients, right upper limb 10(50%) patients, most of the tremors were seen in left upper limb 15 patients, which accounts for 75 %, right lower limb 02(10%) patients, left lower limb 03(15%) patients, 05 (25%) patients had tremors in both upper limbs and 01 (5%) patient had tremor at both lower limbs, one side of the body 03(15%) patients and complete body tremor seen in 01(5%) patients.

CHESTASANGA BRADYKINESIA

Among the 20 patients slow movement was seen in 13(65 %)patients, difficulty to begin walk seen in 08(40%), small hand writing seen in 07(35%) patients, decreased facial expression seen in 04(20%) as hypokinesia together with rigidity results in a reduction of facial expression., difficulty in voluntary movements seen in 08, soft speech noticed in 08 (40%) patients, as decrease in muscle movement of the larynx can reduce the volume and articulation of speech making it difficult for others to understand what is being said.

STHAMBA - RIGIDITY

Rigidity, occurring with Parkinson's disease exacerbates the problems with movement resulting from hypokinesia and bradykinesia. Rigidity in left upper extremity

was seen in 14 patients (60 %), 09 patients (45%) had rigidity in right upper extremity, 01 patient (5%) had rigidity in right lower extremity, 01 patient (5%) had rigidity in left lower extremity, flexed posture seen in 01 patient (5%).

ANUBANDHA VEDANA

Slow speech was seen in 04 (20%) patients, low volume speech in 11 patients (55%) of patients, monotone in 4(20%) patients, difficulty in speaking was found in 01(5%) patients, Vak (speech) and Swara (phonation) are both functions of Udana Vayu. Vak Sanga is a vata nanatmaja vikara where [200]ruksha guna of Vata is responsible for Kshama (low), Jarjara (broken), Ruksha (dry), Sakta (obstructed) and Sanna (hoarse) voice.[201]Gatisanga wsa seen in 3(15%) patients. Vibhanda was seen in most of patients in the study 15 (75%), constipation is very common in Parkinsonism, particularly in more severe cases. It is major symptom of disease and cause of great distress to many patients. Over half the patients of Parkinsonism fail to defecate once daily this is the result of reduced stool transit in the colon, Pelvic floor muscle dystonia may affect the rectum and anus, which, instead of relaxing when trying to pass a stool, goes into spasm. But it is usually attributed to the combined effects of sedentary life and diminished food and fluid intake. Vishada is seen in 09(45%), calf muscle tightness seen in 12 (60%), salivation seen 1 patient (5%), sexual dysfunction in 1 patients (5%).

DISCUSSION ON SUBJECTIVE PARAMETERS

In the present study Kampa was seen in all 20 (100%) patients, gatisanga was seen in 19 (95%) patients, 18 (90%) patients had vakvikruti, 17 (85%) patients had sthamba, all 20 (100%) patients had Chestasanga and only 5 (25%) patients had Avnamana. There are four cardinal features of PD that can be grouped under the acronym TRAP: Tremor at rest (Kampa), Rigidity (sthamba), Akinesia (or bradykinesia) (chestasanga) and Postural instability (avanamana). Postural instability

due to loss of postural reflexes is generally a manifestation of the late stages of PD and usually occurs after the onset of other clinical features.

DISCUSSION ON OBJECTIVE PARAMETERS

In the present study Tremor was seen in all 20 (100%) patients, Rigidity was seen in 17 (85%) patients, 19 (95%) patients had Bradykinesia, 16 (80%) patients had Gait impairment, 18 (90%) patients had Dressing difficulties and 11 (55%) patients had problems with postural stability. T*remor* is the most common presenting sign of early PD. Approximately 70% of patients notice tremor as the first symptom [202] as per the textual reference but due to less sample in this study every patient ie 100 % of patients had tremor as common symptom. Rigidity is also cardinal feature of Parkinson's disease, which is is usually asymmetric in early PD. Rigidity is characterised by increased resistance, usually accompanied by the ''cogwheel'' phenomenon, particularly when associated with an underlying tremor, present throughout the range of passive movement of a limb (flexion, extension or rotation) about a joint. *Rigidity* presents in all muscle groups, both flexor and extensor, but it tends to be more prominent in those which maintain a flexed posture i.e. flexor muscles of trunk and limbs. It appears to be somewhat greater in large muscle groups, but this may be merely, a matter of muscle mass. *Bradykinesia* refers to slowness of movement and is the most characteristic clinical feature of Parkinson's disease; bradykinesia is one of the most easily recognisable symptoms of PD. It is hypothesised that bradykinesia is the result of a disruption in normal motor cortex activity mediated by reduced dopaminergic function. Postural instability (along with freezing of gait) is the most common cause of falls and contributes significantly to the risk of hip fractures. Postural instability due to loss of postural reflexes is generally a manifestation of the late stages of PD and usually occurs after the onset of other clinical features.

IV. DISCUSSION ON RESULTS

Results were interpreted after statistically analyzing the grading of the symptoms mentioned in assessment criteria before, during and after the treatment in all the 20 cases.

KAMPA

In statistical analyses, Kampa was compared before and after treatment showed, mean of parameter Kampa before treatment was 1.30 with S.D. 0.571 is reduced to 0.40 with S.D. 0.598 after treatment. The correlation between before and after treatment is 0.708 which is moderately positively correlated and significant at 0.00. The treatment shows more highly significant before and after treatment at0.000. This supports that the medicine has highly significant effect over kampa as the medicine having haritaki acts as best nervine tonic and kajjali as a rasayan and sarva roga hara. Though the result statistically very high significant, relief subjectively is less. The trial drug shows encouraging result in controlling tremor.

GATISANGA

In statistical analyses mean of parameter Gatisanga before Treatment was 1.10 with S.D. 0.447 is reduced to 0.45 with S.D. 0.510 after treatment. The correlation between before and after treatment is 0.484 which is low degree positively correlated and significant at 0.031. The treatment shows more highly significant before and after treatment at 0.000. This parameter also statistically very high significant, but net relief in this parameter is moderate.

VAKVIKRUTI

In statistical analyses mean of parameter Vakvikruti before Treatment was 1.45 with S.D. 0.686 is reduced to 1.00 with S.D. 0.562 after treatment. The correlation between before and after treatment is 0.682 which is moderately positively correlated

and significant at 0.031. From, the treatment shows more highly significant before and after treatment at 0.001. Highly significant result is seen statistically on Vakvikruti.

STHAMBA

In the study the sthamba parameter had a mean of 1.15 with S.D of .671 which is reduced to 0.30 with S.D of 0.0470 after the treatment. The treatment shows more highly significant before and after treatment at 0.000.

AVANAMANA

In this study mean parameter of Avanamana before Treatment was 0.826 with S.D. 0.45 is reduced to 0.40 with S.D. 0.754 after treatment. The correlation between before and after treatment is 0.964 which is high degree positively correlated and significant at 0.000. The treatment shows not significant before and after treatment as 0.330.

CHESTASANGA

In statistical analyses Mean parameter Chestasanga, Before Treatment was 1.50 with S.D. 0.607 is reduced to 0.70 with S.D. 0.801 after treatment. The correlation between before and after treatment is 0.541 which is moderately positively correlated and significant at 0.014. The treatment shows highly significant before and after treatment at 0.000.

TREMORS

In statistical analyses mean parameter Tremors before Treatment was 2.50 with S.D. 0.607 is reduced to 0.80 with S.D. 0.80 after treatment. The correlation between before and after treatment is 0.753 which is moderately positively correlated and significant at 0.000. The treatment shows highly significant before and after treatment at 0.000.

RIGIDITY

In this study mean parameter Rigidity Before Treatment was 1.70 with S.D. 0.923 is reduced to 0.50 with S.D. 1.00 after treatment. The correlation between before and after treatment is 0.627 which is moderately positively correlated and significant at 0.003. The treatment shows highly significant before and after treatment at 0.000

BRADYKINESIA

Mean parameter Bradykinesia Before Treatment was 1.75 with S.D. 0.851 is reduced to 0.750 with S.D. 0.910 after treatment. The correlation between before and after treatment is 0.255 which is low degree positively correlated and significant at 0.278. The treatment shows highly significant before and after treatment at 0.001.

GAIT

In the study of mean parameter Gait Before Treatment was 1.20 with S.D. 0.834 is reduced to 0.70 with S.D. 0.733 after treatment. The correlation between before and after treatment is 0.707 which is high degree positively correlated and significant at 0.000. The treatment shows highly significant before and after treatment at 0.002.

DRESSING

Mean of parameter Dressing before Treatment was 1.30 with S.D. 0.657 is reduced to 0.65 with S.D. 0.671 after treatment. The correlation between before and after treatment is 0.609 which is moderately positively correlated and significant at 0.004. the treatment shows highly significant before and after treatment at 0.000.

POSTURAL STABILITY

In statistical analyses mean parameter Dressing before Treatment was 0.65 with S.D. 0.671 is reduced to 0.50 with S.D. 0.115 after treatment. The correlation between before and after treatment is 0.841 which is high degree positively correlated

and significant at 0.000. The treatment shows not significant before and after treatment as P>0.05.

The disease Kampavata is a Swabhavaja Vyadhi associated with old age which is a Vata predominant period, which makes the disease Yapya (incurable). However, it was the success of the therapy that improvement was noticed in almost all the patients and none was deteriorated. Parkinson's disease is a chronic, progressive, incurable type of Vata disorder. Triguna rasa being Rasayana acts superior treatment for Vata disorder.

Based on the literature and observations made in this clinical study, following conclusions can be drawn.

- The present study has been entitled as “A clinical study on Kampavata (Parkinson’s disease) and its management with Triguna ras”.
- Parkinson's disease, known in Ayurveda as "Kampa Vata," is a neurological disorder affecting 1% of the population over age 65 and is the fourth most common neurological degenerative disorder found in the elderly.
- Scattered references of Kampa were found and mentioning of kampavata was not found in classics, except Acharaya Basavaraja mentioned the symptoms. To appear at proper diagnosis symptoms explained for Parkinson’s disease in allied science can be considered.
- Parkinson’s disease, also known as Paralysis agitans or shaking palsy, is a serious chronic disease of nervous system.
- The brain changes that lead to Parkinsonism are not fully understood.
- Specific etiology was not found in any of the patients considered for the present study. All these cases can be included under the variety of idiopathic Parkinson’s disease.
- Tremor, Rigidity, Bradykinesia, Gait abnormalities and Postural abnormalities, vak vikruti, being cardinal features of the disease, were noticed in majority of the patients.
- Smoking and alcohol are associated inversely to Parkinson's disease but 04 patients were having habit of smoking and 04 patients were having habit of alcohol.

- Even kajjali has been said as sarvraamayahara but its yogavahi property carries the properties of Haritaki.
- Haritaki forms the 50% of formulation of the drug which is selected for the study, Haritaki purifies Dhatus, acts as Rasayana and Vayasthapana as the karma of Haritaki over nadivaha samsthan is balya and medhya indicated in conditions like mastishkya dourbalya, nadidourbalya and best in all vata vyadhis
- Haritaki is the drug of choice for rejuvenation for all the seasons. This is called as the "Ritu Haritaki".
- The treatment is more highly significant in the parameters, Kampa, Gatisanga, Stamba, Chestasanga Tremors and Rigidity .
- Compared to other symptoms, moderate improvement was observed in Gait, Vakvikruti and Bradykinesia.
- The therapy had no effect on the Avanamana and Postural stability. But in large sample size its efficacy on this parameter can be established.
- This particular yoga is widely practiced in Kerala and we get only the reference in Sahasrayoga. No other texts have such reference.
- More appropriate result can be drawn in large sample study.

Chapter 8 Summary

The present study has been entitled as ***"Clinical study on kampavata (Parkinson's disease) and its management with Triguna rasa"*** comprises following parts.

Introduction:

This part includes importance of the disease entity Kampavata comparison with Parkinson's disease with its prevalence and about the importance of Triguna rasa prayoga and its effects along with response of disease to the therapeutic intervention.

Objectives of the study:

- To evaluate the efficacy of Triguna Rasa in Kampavata
- To evaluate the efficacy of Triguna Rasa in Minimizing effects of Parkinson's disease.
- To study the literary work in Kampavata.
- To study the literary work in Parkinson's disease.

Review of literature

This part includes historical review, vyutpatti and paribasha of kampavata, paryaya, disease review, anatomy and physiology of Basal ganglia, concept of vata vyadhi, and description regarding nidana, lakshanas, nidana, poorvaroopa, roopa, upashaya-anupashaya, samprapti, samprapti ghataka, sapeksha nidana, vyadhi vinischiya, chikitsa, pathya-apathya, upadrava chikitsa etc. laboratory investigations, treatments etc. In the drug review description concerning about the properties and preparation of Yoga.

Methodology:

This includes the selection criteria, study design, plan of the study, subjective and objective parameters, posology, sample size, criteria of diagnosis, inclusion and exclusion criteria and, investigations, grading for variables etc.

Observation and results:

It includes observation on all demographic data with their percentage and graphical representation, regarding the observation nidanas, poorvaroopas, lakshanas and results of individual symptoms followed overall response of the treatment.

Discussion:

Discussion on disease review, Discussion on drug review, Discussion on clinical study, Discussion on results, Discussion on the patients who underwent the trial has been highlighted here.

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Department of Post Graduate studies in Kayachikitsa
D.G.M Ayurvedic Medical College and Hospital GADAG
Special case sheet for evaluation ofs
Triguna Rasa in Kampavata (Parkinson's disease) management

Guide: Dr. R.V. Shettar, M.D (Ayu), (Asst. Professor, P.G. Dept of Kayachikitsa.)										**Scholar: Dr. vijaymahantesh**			
1) Name of the Patient													
2)Father's / husband's name											Sl.No		
3) Sex	Male	Female		D.O.A		D.O.D					OPD No		
4) Age (in years)				Birth place							IPD No		
5) Religion	Hindu			Muslim		Christian		Other					
6) Occupation	Sedentary			Active		Labor							
7) Marital status	Married			Unmarried									
8) Economical status	Poor		Middle		Higher middle		Higher class						
9) Address													
	Contact No:									Pin			
10) Selection	Included			Excluded									
11) Schedule	Initiation Date			Completion Date									
12) Residence	urban			Rural									
13) Result	Well responded		Moderately responded										
	Responded		Not responded		Discontinued								

INFORMED CONSENT

I Son/Daughter/Wife of am exercising my free will, to participate in above study as a subject. I have been informed to my satisfaction, by the attending physician the purpose of the clinical evaluation and nature of the drug treatment. I am also aware of my right to opt out of the treatment schedule, at any time during the course of the treatment.

ಇದು ನಾನು ಶ್ರೀ/ಶ್ರೀಮತಿ _____ ನನ್ನ ಸ್ವಇಚ್ಛೆಯಿಂದ ಕೊಡುವ ಚಿಕಿತ್ಸಾ ಸಮ್ಮತಿ. ಪ್ರಸ್ತುತ ನಡೆದಿರುವ ಚಿಕಿತ್ಸಾ ಪದ್ಧತಿಯ ಬಗ್ಗೆ ನನಗೆ ಚಿಕಿತ್ಸಕರಿಂದ ಸಂಪೂರ್ಣ ಮಾಹಿತಿ ದೊರೆತಿದ್ದು ಮತ್ತು ಯಾವಾಗಾದರು ಚಿಕಿತ್ಸೆಯಿಂದ ಹಿಂತಿರುಗಲು ಸ್ವಾತಂತ್ರ್ಯವಿದೆ ಎಂದು ತಿಳಿದಿರುತ್ತೆನೆ.

ರೋಗಿಯ ರುಜು/Patient's Signatue

14) Pradhana vedana:

Sl no	Complaints		Present	Occur at rest	Occur at any time	Increase with activties	absent	Duration		
								Fresh	<5yrs	>5yrs
1	**Kampa**	**Head**								
		Tongue								
		Lips								
		Chin								
		Upper extremities								
		Lower extremities								
		One side of body								
		Complete body								

			Duration				
			Absent	Present	Fresh	<5yrs	>5yrs
2	**chestasanga**	**Slow movement**					
		stooped					
		Difficulty to begin walk					
		Small handwriting					
		Decreased facial expression					
		Difficult in voluntary movement					
		Soft speech					
3	**sthamba**	**Neck**					
		Upper extremities					
		Lower extremities					
4	**avanamana**						

15) Anubandha vedana:

Sl.no.	Cmplaints		Present	Absent	Duration		
					Fresh	<5 Yrs	>5 Yrs
1	**Bahavarahita mukha**	**Reduced facial expression**					
		staring					
		Unable to close mouth					
2	**Swara bheda**	**Slow speech**					
		Low volume speech					
		Monotone					
		Difficulty in speaking					
3	**vishada**						
4	**gatisanga**						
5	**vibandha**						
6	**calf muscles tightness**						
7	**Salivation**						
8	**Sexual dysfunction**						

16) Adhyatana vyadhi vrittanta:

Mode of onset	Sudden		Gradual	
Duration	Acute (Less than 6week)		Chronic (more than 6weeks)	

17) Poorva vyadhi vrittanta:

History of previous illness –

Treatment History

18) Kula vrittanta:

Heart Disease		Cancer	
Hypertension		Thyroid disorders	
Diabetes		Obesity	
Hemiplegics		Any other	

19) Chikitsa vrittanta

Newly Diagnosed				Previously Diagnosed				
Previous Medication		Ayurvedic		Allopathic		Discontinued		
Drug used	1			2				
	Dose		duration		dose		duration	
Response		controlled		not controlled				
Oral Contraceptives	Yes		No		Duration		Dose	
Anti depressant	Yes		No		Duration		dose	

20) Vyasana-

Smoking	Duration			
	Daily			
	Occasionally			
	Frequency of smoking			
Alcohol	Duration			
	Daily		Quantity	
	Occasionally		Present condition	

21) Vyaktika vrittanta

Nidra	Night sleep	Hours		Day sleep	Hours						
	Natureof sleep	Normal		Disturbed							
	Dreams	Yes		No							
Ahara	Vegetarian		Rasa predominance	Madhura		Amala					
	Mixed food			Lavana		Katu					
	Stored food			Tikta		Kshaya					
Kosta	Krura		Madhyama		Mrudu						
Jatharagni bala	Manda		Teekshna		Vishama		Sama				
Occupational history	Type of employment										
	Use of pesticides	Yes		No							
Student		Labour		Executive		Sedentary					
Work involving any mental stress		Yes		No							
Diffiulty in writing		Yes		No							
Difficulty in eating		Yes		No							
Difficulty with any activities that requires small movement		Yes		No							
Frequent fall		Yes		No							
Drenching of sweets		Yes		No							
Urine frequency		Yes		No							
Urine urgency		Yes		No							
Nausea		Yes		No							

Use of Drugs like	Neuroleptics	Yes		No	
	Antihypertensives	Yes		No	
	Antiemetics	Yes		No	

22)Manasika vrittanta:

1	Emotional stress		6	Samprahara (Aggressiveness)	
2	Udvega (Anxiety		7	Mada (Delirium)	
3	Depression		8	Dementia	
4	hallucinations		9	panic	
5	Unusal laugh		10	Unusual cry	

23) Samanya Pareeksha

Pulse	/min	Temp	°F	Respiration rate	/min
Weight	/kgs	Height	cms	Heart rate	/min

24) Vishesha pariksha

Activities of daily living	dressing		normal		slow		unable	
	walking		normal		slow		unable	
Higher mental functions	memory	past			present			
	depression	present			absent			
Motor functions	facial expression	Normal		Expressionless				
	Tremor	Tremor at rest	face	RT UE	LF UE	RT LE	LF LE	
		Postural Tremor	face	RT UE	LF UE	RT LE	LF LE	
Posture	Normal			stooped				
Gait	Normal		Short steps		Festinant			
Bradykinesia	Mild		Moderate		Marked			

25) Systemic examination:-

Respiratory System:

Shape	Normal		Abnormal										
Respiratory Rate													
Rhythm													
Character	Abdominal		Thoracic		Thoraco abdominal								
Chest expansion	Measurement		Expiration		Inspiration								
Intensity	Normal		Reduced		Increased								
Type of breathing	Vesicular		Bronchial		Bronchovasicular								

Abdomen:

Inspection	
Palpation	
Percussion	
Auscultation	

Other systems (If any)**:**

26) Aturabala pareeksha:

a)Ashta vidha pareeksha :-

1.Nadi	Rate		Type		4. Jihwa	
2.Mala	Varna		Times/day		5. Shabda	
	Consistency				6. Sparsha	
3.Mootra	Varna				7. Druk	
	Times/day				8. Akruti	

b) Dashavidha pareeksha:-

Prakruti	Shareerika	V		P		K		VP		VK		PK		Sama					
Sara	Twak	Rakta		Mamsa		Meda		Asthi		Shukra		Majja		Satwa					
Samhanana	Susamhita			Madhyama samhita								HeenaSamhita							
Satmya	Pravara			Madhyama								Avara							
Satwa	Pravara			Madhyama								Avara							
Vyama shakti	Pravara			Madhyama								Avara							
Vaya	Balya			Madhya								Vruddha							
Pramana	Supramanita			Adhika								Heena							
Ahara shakti	Abhyvarana			Jarana															

27) Laboratory investigations:

No	Investigations:	Before	After	Changes observed
1	Hb%			
2	Total WBC count			

3	Differential count			
4	Erythrocyte sedimentation rate			
5	Random blood glucose			
6	Blood urea			
7	Sr creatinine			
8	Sr copper			
9	Complete urine examination			

28) Chikitsa

Yoga: Triguna rasa

Posology

Abhyantara : Triguna rasa starting with 7 ratti on first day and increasing one ratti each day upto 21 days and from 22nd day decreasing one ratti till to seven ratti remains.

Anupana: Ghrita followed by rice milk.

29) Subjective parametrers:

	BT	**After 21 days**	**After 41 days**	**Follow up**
Kampa				
Gatisanga:-				
Vakvikriti :				
Stambha				
Avanamna				
Chestasanga				

30) Objective parameters

	BT	**After 21 days**	**After 41 days**	**Follow up**
Tremors				
Rigidity				
Bradykinesia				
Gait				
Dressing				
Postural stability				

31) ASSESSMENT CRITERIA FOR OVER ALL EFFECT OF TREATMENT

- Good Response : >75% improvement in clinical parameters
- Moderate Response : 50-75% improvement in clinical parameters
- Poor Response : up to 50% improvement in clinical parameters
- No Response : 0 % or No improvement in clinical parameters

Investigator notes:

Signature of the guide.	signatutre of the scholar.

30)Subjective parametrers:

Kampa (Tremor)	**Score**
Bilateral violent tremor along with tremor in tongue and / or in eyelids lips and not suppressed or diminished by willed movement.	-3-
Bilateral tremor	-2-
Unilateral slight tremor present at rest decreased by action, increases by emotion and stress	-1-
No tremor	-0-
Gatisanga:-	
Unable to raise from bed and walk without assistance	-3-
Can walk slowly but need substantially help, shuffling with retropulsion/ propulsion lack of associated movement	-2-
Can walk without assistance slowly but with shuffling gait	-1-
Can walk brisk without aid	-0-
Vakvikriti :-	
Incomprehensive words, monotonous voice, echoing, speaks only on insistence of examiner	-3-
Monotonous voice, spilt consonance but understandable speaks feels with examiner	-2-
Variable tone of voice.	-1-
Normal speech	-0-
Stambha (rigidity)	
Marked rigidity in major joints of limbs, patients maintain abnormal sitting postures, stared eyes	-3-
Rigidity demonstrable on one of major joints	-2-
Cog-wheel rigidity feebly present and on continuos examination vanishes	-1-
No rigidity	-0-
Avanamna	
Complete bend down of body	3
Head bent forward with legs bent at knees	2
Only arm bent at elbows	1
No bending or flexion	0
Chestasanga	
Unable to carry routine activities of daily life	3
Able to perform daily activities with moderate difficulties	2
Able to perform daily activities with less difficulties	1
No difficulties in carrying out activities	0

31) Objective parameters:

Grading for Variables

Sl.No	Objectives	Gradations
1	Tremors	**Gr 0 – Absent**
		Gr 1 - Slight and infrequent
		Gr 2 – moderate
		Gr 3 – Marked
		Gr 4 - Marked with all activities
2	Rigidity	**Gr 0 – Absent**
		Gr 1 - Slight and infrequent
		Gr 2 – moderate
		Gr 3 - Severe, interferes with many activities
		Gr 4 - Marked with all activities
3	Bradykinesia	**Gr 0 – None**
		Gr 1 - Minimal slowness
		Gr 2 – Mild slowness and poverty of movement
		Gr3 – Moderate slowness poverty or small amplitude
		Gr 4 - Marked slowness ,poverty,or amplitude
4	Gait	**Gr 0 – Normal**
		Gr 1 - Walks slowly , may shuffle with worst steps no propulsion
		Gr 2 - Walks with difficulty or little assistance or no assistance
		Gr 3 - Severe disturbance no assistance
		Gr 4 - Cannot walk
5	Dressing	**Gr 0 - – Normal**
		Gr 1 - Slow no help needed
		Gr 2 Occasional help with buttons
		Gr 3 - Considerable help required
		Gr 4 – helpless
6	Postural stability	**Gr 0 - – Normal**
		Gr 1 - Recovers unaided
		Gr 2 Would fall if not caught
		Gr 3 - Falls spontaneously
		Gr 4 - Unable to stand