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A REVIEW ARTICLE ON *PAKSHAGHAT* WITH SPECIAL REFERENCE TO STROKE

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ABSTRACT

Stroke or Hemiplegia can be defined as a clinical feature of one side weakness or paralysis in upper & lower extremities and with same part of face. Hemiplegia is the commonest manifestation of a 'Stroke' with neurological shortage affecting the face, limbs and trunk on one side or either side of the body. Impulses for voluntary movements are conveyed by the pyramidal tracts or upper motor neurons. Destruction to these pyramidal tracts due to any lesion, trauma, ischemia or hemorrhage produces paralysis. In Ayurveda, it can be interrelated with 'Pakshaghata'. In the term 'Pakshaghata', Paksha indicates an individual's flank or side and Ghata indicates destruction, killing, and paralysis. When Vata Dosha gets aggravated, it paralyzes one side of

body, either right or left and causes immobility of that side in association with Pain and loss of speech. Aggravated *Vata Dosha* dries up the *Snayu* and *Strotas* of that side of the body, resulting in loss of sensation and incapability of functioning of organs or parts of that side. *Acharya Charaka* has mentioned *Pakshaghata* in *Vata Nanatmaja Vyadhi* and *Acharya Sushruta* has described in *Mahavata Vyadhi*.

KEYWORDS: Stroke, Hemiplegia, Pakshaghata, Vata Vyadhi.

INTRODUCTION

In the term '*Pakshaghata*', *Paksha* indicates an individual's flank or side^[1] and *Ghata* indicates destruction, killing, and paralysis.^[2] *Acharya Charaka* has mentioned *Pakshaghata* in *Vata Nanatmaja* Vyadhi^[3] and *Acharya Sushruta* has described in *Mahavata Vyadhi*.^[4] In modern science, *Pakshaghata* can be correlated with 'Stroke' or 'Hemiplegia'. When *Vata*

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Dosha gets aggravated, it dries up the *Snayu* and *Strotas* of one side of the body, resulting in loss of sensation and incapability of functioning of organs or parts of that side.^[5]

Stroke is clinically characterized by unilateral loss of voluntary power in the affected side i.e. arm, leg and in the lower face. Tongue is protruded towards the paralyzed side (in facial palsy). Clasp-knife type spasticity. Upper limb flexed at the elbow and wrist forearm slightly pronated. Movement of the fingers and hand are more affected than those of upper arm.

Hemiplegia is the commonest manifestation of a 'Stroke' with neurological deficit affecting the face, limbs and trunk on one side or either side of the body. Impulses for voluntary movement are transmitted by the pyramidal tracts or upper motor neurons. Damage to these pyramidal tracts due to any lesion, trauma, ischemia or hemorrhage produces paralysis. In Ayurveda, it can be correlated with '*Pakshaghata*'. When *Vata Dosha* gets aggravated, it dries up the *Strotas* and *Snayu* (tendons) of one side (half) of the body, makes the organ/ parts of that side incapable of functioning and loss of sensation.^[6]

Stroke is damage to the brain tissue due to either cerebral infarction or cerebral hemorrhage. It is third commonest cause of death in developing countries. Stroke is common in older patients and affects males 1.5% more than females. Younger people occasionally suffer from the Stroke because of trauma to cerebral vessels, inflammatory disorders of arteries or congenital vascular anomalies. Stroke is a prominent cause of disability. Pathological studies indicate that 80-85% Strokes are due to cerebral infarction, 15-20% are caused due to hemorrhage. The most common vascular disorder underlying Stroke is Atherosclerosis affecting intracranial and extra cranial arteries, and other mechanisms are arteriolar atherosclerosis embolisms, arteritis, dissection, vasospasm.^[7]

Patients with other cardiovascular diseases (particularly ischemic heart diseases) and peripheral vascular diseases are more prone to Stroke.

The major risk factors of Stroke are arterial hypertension, cigarette smoking, diabetes mellitus, hyperlipidemia, polycythemia, thrombocythaemia.

MATERIAL AND METHODS

Material is collected from classical text books, Articles, journals and other issued works. This is a review article i.e. based on a review of *Ayurvedic* texts and Modern texts. Main *Ayurvedic Samhitas* used in this article are *Charak Samhita, Sushrut Samhita, Ashtang*

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Hridaya, *Yogaratnakara*, *Bhavaprakasha*, *Vangasena* and all relevant book which gives idea to complete this article.

REVIEW OF LITERATURE

Pakshaghata Nirukti

The word '*Pakshaghata*' derived from two *Sanskrit* words *paksha* + *Aaghata*. *Paksha* = *Dehanga* here one side of body.^[8] *Aaghata* = *Hanana* i.e. destruction means *dama*ge or injury.^[9]

Definition

According to shabdakalpadruma Vachaspatyam 'pakshaghata' has been defined as "Pakshaghatama Dehangsya Ghatam Avinasham Yasmat Yatra Va."^[10]

Therefore, hemiplegia can be defined as a clinical feature of one side weakness or paralysis in upper and lower extremities and with same part of face.

Types of Pakshaghata

According to Acharya Sushruta, there are 3 types of Pakshaghata as;^[11]

- 1) Shuddha Vataja:- where Vata is aggravated on account of its own Nidana.
- 2) Anya Doshanubandha:- where Vata is associated with Anya Dosha to manifest the disease.
- 3) Kshya Hetuja Pakshaghata:- where Vata is aggravated as consequence of Dhatu Kshya.

Hetu

The importance of *Hetu* in the management of any disease can be well understood from texts. To understand *Hetu*, it can be classified in four categories.

Aharaja Hetu, Viharaja Hetu, manasaja Hetu and Anyahetu.

According to charaka

There is no any specific *Hetu* of *Pakshaghata* is described in *Charak Samhita, Samanya Hetu* of *Vatavyadhi* also considered for *Pakshaghataja Hetu*. General *Hetu* of *Vatavyadhi* described in Charaka are,^[12]

Sr. No	Type of Hetu	Examples	
1	Aaharaja Hetuexcessive intake of Ruksha, Laghu, Shita Gunatmaka Aahara Dravya, Alpa Aahara Sevana		
2	Viharaja Hetu	araja Hetu Atishrama Ativyavaya, Atijagrana ,Atiplavan , Dukhashaiyya- Aasana, Divaswapa, Vegavidharana, Abhighataati-	

		Shighrayanayam.
3	Manasa Hetu	Atichinta, Shoka, Bhaya, Krodha.

Poorva Rupa

There is no any specific *Poorva Rupa of Pakshaghata* mentioned in any *Samhita*. *Pakshaghata* is described under *Vatavyaddi*.^[13] The general *Poorvarupa* of *Vatavyadhi* are not mentioned, *Acharya Charka* said as '*Avyakta Lakshana Tesham Poorvarupam Etismrutama*.^{/[14]}

Rupa

Various symptoms of *Pakshaghata* described in *Ayurvedic* literatures are.

Sr. no.	Lakshana	Charak [15]	Sushrut [16]	Ashtang Samgraha ^[17]	Ashtang Hridaya ^[18]	Madhava Nidana ^[19]	Yogarat nakara ^[20]	Bhava praka sha ^[21]	Vang asena [22]
1	Anyatara Paksha Chesta Nivritti	+	+	+	+	+	+	+	+
2	Anyatara Pakshahanan	+	+	+	+	+	+	+	+
3	Achetana		+	+	+	+	+	+	+
4	Akarmanyata		+	+	+	+	+	+	+
5	Hasta Pada Sankocha	+					+	+	
6	Sira Snayu Vishosha	+		+	+	+	+	+	
7	Vak Stambha	+							
8	Ruja	+							
9	Toda	+							
10	Shoola	+						+	
11	Sandhibandha Vimoksha		+	+	+	+	+	+	+
12	Patatya		+						+
13	Daha, Santap, Moorcha		+			+	+	+	+
14	Shaitya, Shopha, Gurutva		+			+	+	+	

Vangasena – same as like Sushruta and Vagabhata.^[22]

Samprapti Ghataka of Pakshaghata:

Samprapti Ghataka chiefly constitutes Dosha, Dushya, Strotasa, Aama and Agni. Detailed description of each in context of Pakshaghata is as follows;

Samprapti Ghatakas of Pakshaghata are

• Dosha - Pradhana Dosha- Vata Dosha

Prakara - Prana, Udana, Vyana,

- Anubandhi Pitta, Kapha
- Dushya (Dhatu)- Rasa, Rakta
- Upadhatu Sira, Snayu, kandara
- Agni May be affected
- *Aama* May be present
- Strotasa Rasa Vaha, Rakta Vaha
- Stroto Dushti Sanga
- Udhbhava Sthana Pakwashaya
- Sanchara Sthana Dakshina/ Vama Sira, Dhamani, Snayu
- Sthana Samshraya Shiras
- Vyaktsthana Ardha Sharira
- Dosha: Vata dosha is mainly involved in Pakshaghata. Vata Dosha Prakar i.e. Prana, Udana and Vyana Vayu are chiefly involved. Thereafter Pitta and Kapha Doshas also play a vital role in manifestation of Pakshaghata to specify its types as said by Acharyas; Pittanubandhi Pakshaghata and Kaphanubandhi Pakshaghata. Ancient scholars were well aware of the neural connection and various disorders created by provoked Vata Dosha.^[23]
- Dushya: In Pakshaghata Rasa, Rakta, Mamsa, Meda, Majja Dhatus are involved. Sira, Snayu, Dhamani and Mala are involved.
- Strotasa: In Pakshaghata Rasavaha, Raktavaha, Mamsavaha and Medovaha Strotasa are involved.
- Strotodushti: Atipravritti, Sanga, Sira granthi and Vimargagamana^[24] are four types of Strotodusti. All these types are observed in Pakshaghata. Paralysis occurs due to obstruction in cerebral vessels leading to ischemia. This can be reflected as Sanga. Various types of aneurysm can be measured as Siragranthi. Cerebral hemorrhage can be well-thought-out as Atipravritti and Vimargagaman.
- Agni: as stated by Acharya "Roga Sarve Api Mandagane", Mandagni is root cause of all diseases. In Vatavyadhi Agni remains Vishama. So, in Pakshaghata also Agni remains Vishama.
- > Aama: Mandagani causes production of 'Aama' means undigested or semi digested

material. It causes obstruction in various *Strotas* by *Guna Vishesh* i.e *Snigdha, Picchila Guna.* This *Strotorodha* further causes vitiation of *Dosha* and *Dhatukshaya* due to obstruction of nutrition of various *Dhatus*.

- Udbhava Sthana: Pakvashaya
- > Adhisthana: Ardhsharira or Sarvanga^[25]

STROKE

• Definition

Stroke or cerebrovascular accident is defined as an immediate onset of a neurologic deficit that is attributable to focal vascular cause. Thus, the clinical definition of stroke and laboratory studies including brain imagining are used to support the diagnosis.^[26]

• Etio-pathogenesis

The clinical manifestation of stroke is highly variable because of the complex anatomy of the brain and its vasculature. Cerebral ischemia is caused by a reduction in blood flow that lasts longer than several seconds. Neurologic symptoms are manifest within seconds because neurons got lack glycogen, so energy failure is rapid.

A generalized reduction in cerebral blood flow due to systemic hypotension i.e. cardiac arrhythmia, myocardial infarction, or hemorrhagic shock usually produce syncope. If low cerebral blood flow persist for longer duration, then infarction in the border zones between the major cerebral arteries distributions may develops. In more severe instances, global hypoxia ischemia causes widely spread brain injury.

• Ischemic stroke

Focal ischemia or infarction is usually caused by thrombosis of the cerebral vessels themselves or by emboli from a proximal arterial source or the heart.

• ETIOLOGY OF ISCHEMIC STROKE

Establishing a cause of Stroke is essential to reduce the risk of recurrence. Particular focus should be on atrial fibrillation and carotid atherosclerosis, because these etiologies have proven secondary prevention strategies. The clinical presentation and examination findings often establish the cause of Stroke or narrow the possibilities to a few. Still, nearly 30% of strokes remain unexplained despite extensive evaluation.

• Hemorrhage stroke

Intracranial hemorrhage is caused by bleed directly into or around the brain. It produces neurologic symptoms by producing a mass effect on neural structures, from the toxic effect of blood itself, or by increasing intracranial pressure.

• Transient ischemic attack (TIA)

When the cessation of blood flow last for more than a few minutes, it results into infarction or death of brain tissue. Hampered blood flow is quickly restored brain tissue can recover fully, and patient symptoms are only transient, this is called transient ischemic attack (TIA).

In the condition of TIA, all the neurogenic signs and symptoms resolve within 24 hrs. without evidence of brain infarction on brain imagining. Stroke has occurred if the neurogenic signs and symptoms lasts for more than 24 hrs. or brain infarction is demonstrated.

Causes of Stroke^[27]

The primary pathophysiology of Stroke is an underlying heart or blood vessel disease. The secondary manifestations in the brain are the result of one or more of these underlying diseases or risk factors. The primary pathologies include hypertension, atherosclerosis leading to coronary artery disease, dyslipidemia, heart disease and hyperlipidemia. The two types of stroke that result from these disease states are ischemic and hemorrhagic Strokes.

Non-reducible Risk Factors

- 1. The possibilities of a stroke occurrence increases with age. For every decade (10 years) over the age of 55, the possibility of a stroke occurrence doubles. A patient that is 75 years of age has four times the risk of having a stroke compared to someone who is 55 years old. Of all Strokes that occur in people, approximately 65% occur in those who are over the age of 65.
- Those who have had a Stroke or TIA are more likely to have another Stroke or Transient Ischemic Attack. Approximately 60% of strokes occur in patients who have had a previous TIA.
- 3. Generally, Stroke occur more often in males than females, till the age of 55; after age 55 the risk is the same for both men and women.
- 4. The occurrence of Stroke is higher in the African-American, Hispanic, and Asian-Pacific Islander population than in other ethnical backgrounds.

- 5. Patients who have immediate family members (mother, father, or sibling) that have had a Stroke or TIA are at greater risk for having a stroke or TIA than those who do not have a family history with these events.
- 6. People who have diabetes are also at greater risk of stroke that those without diabetes.

Reducible Risk Factors

- Lower your high blood pressure. Hypertension (high blood pressure) is the number one most treatable risk factor for Stroke. You can help prevent a TIA or Stroke considerably by working to lower your blood pressure.
- 2. Lowering cholesterol levels may decrease the risk of Stroke. By working to lower your cholesterol, you can help prevent a TIA or Stroke.
- 3. Stop smoking, by this you can decrease your risk for Stroke to that of a non-smoker within two to five years.
- 4. Management of heart disease and diabetes may also help to decrease your risk of Stroke.

• Pathophysiology of Stroke

Acute occlusion in an intracranial vessels causes reduction of blood flow to the brain region it supplies. The magnitude of flow reduction is a function of collateral blood flow, and this depends on individual vascular anatomy the site of occlusion and systemic blood pressure.

Sr.no	Duration of decreased cerebral blood flow	Result of decreased cerebral blood flow
1	Complete cessation of blood flow for 4-10 min.	Death of brain tissue.
2	Value decreased less than 16-18 ml/100 g tissue/min. for an hour.	Result in ischemia within 1 hr.
3	Value of decreased blood flow less than 20 ml/100 g tissue/ min.	Ischemia without infarction.
4	If blood flow restored to ischemic tissues before significant infarction develops, the patient may only experiences transient symptoms.	Trans Ischemic Attack (TIA).

• Mechanism of Stroke

A) Cerebral Infarction

Occlusion of a major cerebral artery usually leads to infarction unless as in some young people, a collateral circulation is developed. Thrombosis at atheromatus degeneration in a major cerebral vessels is probably the commonest mechanism, but embolism of thrombotic or atheromatus material from heart or extra cranial artery is also frequent. Cardiac emboli tend

to be a large and cause occlusion of one of the principal cerebral artery or a major branch, thereby causing usually major stroke.

Once deprived of blood supply cerebral tissue undergoes infarction within a few minutes. Released excitatory amino acids may exacerbate the neural damage by promoting calcium influx. The damaged neurons and glia become edematous after some hours. The resultant cerebral edema causing more damage by further impairing cerebral blood flow.

B) Cerebral Hemorrhage

About half of Strokes are cause by cerebral hemorrhage due to subarachnoid bleeding from rupture of an aneurysm at the circle of willis and less commonly from an artiovenous malformation. In other patients, hemorrhage is mainly into the cerebral substance and is due to rupture of small perforation arteries or arterioles weakened by hypertension or artheromatus degeneration. Intracerebral hemorrhage of this type tends to occur at three distinct sites.

- 1. The internal capsule- from lenticulo striate arteries
- 2. The pons- from perforating branches of the basilar artery
- 3. The cerebellum.

Subarachnoid hemorrhage may be induce secondary arterial spasm and thereby cerebral infarction. Although a large cerebral infarction cause severe disability, small bleeds in deep cerebral white matter may cause only mild and transient defects. Cerebral hemorrhage can be fatal if secondary compression of the brain stem occur.

Clinical features of Stroke

Classification of clinical features of stroke as.

- A) Complete Stroke
- Complete major Stroke
- Complete minor Stroke
- B) Evolving Stroke
- C) Trans Ischemic Stroke

A) Complete stroke

This is an episode of focal cerebral dysfunction, due to either cerebral infarction or hemorrhage.

The symptoms are lasting longer than 24 hrs. The Stroke evolved rapidly over a few minutes and reach maximum disability within two hours. Sometimes a slower course occurs, the disability advancing gradually over several hours or days. This is known as Stroke in evolution.

Headache is a common accompaniment to acute Stroke and does not help to distinguish infarction from hemorrhage. Epileptic seizures, vomiting and depressed consciousness may also occur and later usually indicating a severe lesion. The precise features of Stroke are depend on vascular territory involved, as lesion.

Minor Stroke

Some patients with a complete Stroke improves rapidly, with recovery from disability over the course of the first or two weeks. Rather arbitrary definition of minor Strokes is useful because of minor Stroke is useful because such patients can be managed in much the same way as a case of Transient Ischemia.

B) Evolving Stroke

This type of Stroke can be due to cerebral tumor or subdural hematoma, but is more often due to slow occlusion of a major cerebral vessel such as internal carotid or middle cerebral artery.

DISCUSSION

When considerable *Agni* is decreased, it causes production of undigested or semi digested material called as '*Aama*'. This *Aama* has *Snigdha*, *Picchila Guna* and it causes obstruction in various *Strotas* i.e. *Strotorodha*. This *Strotorodha* further causes vitiation of *Dosha* and also obstructs nutrition of various *Dhatus* leading to *Dhatukshaya*. In *Pakshaghata Rasagata* and *Raktagata Aama* (Thrombus embolism) are produced, which obstructs cerebral arteries causing ischemia in brain tissue and leads to its necrosis (*Dhatukshaya*). All these causes *Vata Prakopa* i.e. increase in *Ruksha*, *Shita Guna* of *Vayu* and decrease in its *Chala Guna* (loss of voluntary movements).

CONCLUSION

• Various functions of *Vata Dosha* are; it encourages all the types of actions, restrains and induces mental activities, coordinates all the sense faculties etc. These functions of *Vata Dosha* might be correlated with the functions of brain. Hence, here we can say that there is presence of neural connection in various disorders manifested by provoked *Vata Dosha*.

• The diseases like *Pakshwadha* or *Ardita* are having the involvement of brain from modern studies. Various signs and symptoms seen in *Pakshaghat* are related to brain and nervous system which are also present in Stroke or Hemiplegia. *Pakshaghat* has been linked to Hemiplegia, which is a type of Paralysis caused by a cerebrovascular accident or Stroke.

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