Journal of Pharmaceutical and Scientific Innovation

www.jpsionline.com (ISSN :2277 -4572)

Review Article

A COMPREHENSIVE REVIEW ON VAMANAUSHADHA KARMUKATA

Sangeeta S [1]*, Lohith B A [2], Amritha Rajan [3]

[1] PG Scholar, Department of Panchakarma, Sri Dharmasthala Manjunatheshwara College of Ayurveda and Hospital, Hassan, Karnataka, India

[2] Professor, Department of Panchakarma, Sri Dharmasthala Manjunatheshwara College of Ayurveda and Hospital, Hassan, Karnataka, India

[3] Assistant Professor, Department of Panchakarma, Sri Dharmasthala Manjunatheshwara College of Ayurveda & Hospital, Hassan, Karnataka, India

*Corresponding Author Email: drsangeetasatish@gmail.com

DOI: 10.7897/2277-4572.091161

Received on: 04/10/19 Revised on: 28/11/19 Accepted on: 01/12/19

ABSTRACT

Vamana, included among the Panchakarma is a procedure in which vitiated doshas are expelled through the process of emesis i.e. expelling contents through the mouth. Vamana drugs act on the basis of its Ushna, Tiksna, Suksma, Vyavayi, Vikasiguna and by virtue of its bhautika composition. Drugs of Vamanopaga category support the easy execution, by aiding the whole procedure. From the modern point of view the drugs act via many of the pathways, ultimately stimulating the vomiting centre in the brain which initiates and controls the whole process. Through this article, an attempt has been made to understand the process of vomiting induced by the ingestion of Vamana aushadha; its properties and how these effects the corresponding pathways to trigger vomiting.

Keywords: *Vamana Karmukata*, Vomiting reflex, Vamana, Mode of action.

INTRODUCTION

Vamana is a process in which vitiated doshas are expelled through the process of emesis i.e. expelling contents through the mouth. Chakrapani opines that that the term urdhvabhaga can be attributed to urdhvamukha meaning through the upper orifice, in this case the mouth. The process in which the callow (apakwa) pitta or shleshma is expelled out through the urdhwabhaga can be termed as Vamana.[1] The word doshaharana in the context of Vamana does not always denote elimination of Kaphadosha, it can also easily bring out other mala from the body. Here, mala can be understood as any substance which obstructs or adheres to the srotas, which is formed separately, or as a metabolic end product.[2]

Vamana can be prescribed in a plethora of situations, namely

1. Increase of Kapha in swasthana: As in ritushodhana, Tamakashwasa
2. Pitta mixed with kapha: Amlapitta
3. Pitta in kaphasthana: Unmada, Jwara
4. Pitta-vata in kaphasthana: Mano-vikaras, madatyaya, dhatu-kshaya janya rajayakshma
5. Kapha as mala: Nanatmajavyadhis

Some of the common Vamaka dravyas which can be categorised according to the part used are

1. Moolini[3]: Haimavathi (*Acorus calamus*), Shanapushpi (*Calotropis verrucosa*), Bimbi (*Coccinia indica*)
2. Phalini[4]: Dhamargava (*Luffa aegyptiaca*), Jeemutaka (*Luffa echinata*), Madanaphala (*Randia dumetorum*)
3. Vamaka lavana[5]: Saindhava (Himalayan pink salt), Bida (Ammonium salt), Souvarchala (Unaqua Sodium chloride)
4. Kshira[6]: Ashmantaka (*Ficus rumphii*), Arka (*Calotropis procera*)

Properties of vamanaushadha

The properties of the Vamana drug have been mentioned as ushna, tikshna, sukshma, vyavayi and vikashi. These characters bring about the much needed pachana and spreading of the drug at deeper cytological level by virtue of its sukshmaguna. A quicker response after ingestion of the medicine, shodhana, chedana and sravana of doshas from where they are situated (vichchandana of dosha) due to teekshnaguna. The sukshmaguna also helps in deeper penetration into the minute channels of the body and mobilisation of it back into the koshta. Vyavayi guna helps in getting the medicine absorbed faster before being metabolised completely; hence spreading the effect of medicine in its most potent form for a better action. Vikashiguna induces sandhi shaithilyata, which can be understood as the property of the medicine which helps separate the doshas from the morbid matter. The urdhwabhagaprabhava of the medicine helps in expelling the contents of the stomach through the mouth rather than causing increased bowel movements and thereby defecation/ virechana (loose stools). Saratvaguna is the property which helps the process of emesis to be continued uninterrupted, till the flushing out of all the metabolised phytochemicals of the administered medicine.

Most of the vamanaushadha have laghu, rukshaguna, and katu,kashaya rasa which are Vayu and Agni mahabhuta predominant. This explains the reason for causing Vamana, as these mahabhutas further substantiate the urdhwabhagaprabhava due to the laghuguna it possesses. All drugs which possess an emetic effect cannot be used for Vamana. This is due to the property of anupravana bhava. This property helps the drugs not

to get accumulated in the cells, thereby lowering the chances of causing toxicity or any complication. An ideal vamana drug penetrates into the minute channels and returns quickly after exercising their effect wherever it is intended.[7]

Vamana karmukata

Once the medicine is ingested, in normal cases after a certain amount of time (one muhurtha) it exhibits a specific set of signs like Swedapravartana (sweating is induced), Romaharsha (horripilations), Kukshiadhmapana (bloating of abdomen/stomach), Hrillasa (nausea). The reason behind experiencing these symptoms are explained as pravilayana (dilution/liquefaction due to the action of drug) of doshas, dislodgement of accumulated doshas from its place, displacement from shakha to koshtha and ultimately the feeling of nausea just before the actual vomiting reflex takes place.

The occurrence of these symptoms can also be understood in the light of the gunas of the vamana medicine, as in, Swedapravartana can be due to its ushna, teekshnaguna which acts on the sympathetic innervations of the body thereby producing sweating. Kukshiadhmana can be related to the anupravana bhava and urdhwabhagaharaprabhava which is more of a parasympathetic origin. Romaharsha and Nausea are sensations just before the act of vomiting due to neurological involvement, which is guided predominantly by sympathetic activity.

According to the classics the mode of action has been explained as the Vamana aushadhi possessing the properties discussed previously due to their swavirya (own potency) moves tohridaya**,** through the dhamanees into the Sthoola and sookshma srotas and liquefies the aggregated metabolic complexes and reaches the Amashaya causing kukshiaadhmana, from where it further moves upward in gut propelled by udanavayu,andagni and vayu dominancy and is expelled out of the body as Vamana.

The vomiting reflex

Vomiting can be understood in the light of contemporary understanding of sciences as true vomiting i.e. the active expulsion of gastric contents through the mouth, also known as Emesis, or a passive regurgitation of the gastric contents, merely due to reverse peristalsis. Immediately preceding the act of vomiting are tachypnoea and widespread autonomic discharge like copious salivation, dilatation of the pupils, sweating and pallor. Vomiting is usually experienced as the finale in a series of three events:

a. Nausea
b. Retching (dry heaves)
c. Emesis

An unpleasant and difficult to describe psychic experience just before vomiting in humans and probably animals can be termed as nausea. Physiologically, nausea is typically associated with decreased gastric motility and increased tone in the small intestine. Gastric relaxation is observed in this period preceding the evacuation of the stomach contents. This relaxation is brought about by stimuli descending in that part of the vagus nerve which innervates the intrinsic inhibitory enteric neurons.

Additionally, there is often reverse peristalsis in the proximal small intestine. During the gastric relaxation that precedes vomiting, the electrical spike activity that is normally present, when superimposed on the basic electrical rhythm (BER), indicates, contractile behaviour disappears in the stomach and duodenum and the BER itself slows down or is suppressed. The electrical silence is followed rapidly, before vomiting occurs, by an intense spike activity in the duodenum and jejunum. This intense spike activity either occurs simultaneously over the whole duodenum or clearly starts distally from the duodenum or the jejunum to travel back to the pylorus; it is the marker of a generalized muscular contraction which can behave as an anti-peristaltic wave. So we can conclude that the electrical silence of the stomach musculature and the intense spike in activity of muscles of intestine help in the reverse peristalsis which often aides the process of vomiting. Retching refers to spasmodic respiratory movements conducted with a closed glottis. While this is occurring, the antrum of the stomach contracts and the fundus and cardia relaxes. Emesis occurs when gastric and often small intestinal contents are propelled up to and out of the mouth. It results from a highly coordinated series of events.[8]

Vomiting: The anatomical structures involved

In describing the anatomy of vomiting these can be broken down more specifically into a description of:

A. The vomiting centre : Chemoreceptor Trigger Zone, Nucleus tractus Solitarius
B. The afferent pathways: GI Tract, Higher brainstem, Cortical centre
C. The efferent pathways and mechanics of vomiting: Phrenic nerve for diaphragm, Spinal nerve for abdominal musculature, Visceral efferent fibres in vagus- larynx, oesophagus and stomach.
D. The gut: Three sphincters i.e. the gastro-oesophageal, the laryngeal or the nasopharyngeal sphincter, the pyloric sphincter and musculature of stomach along with its plexuses.
E. The structure and nerve supply of the diaphragm.

A. Within the brainstem are two anatomically and functionally distinct units that control vomiting. Bilateral vomiting centers in the reticular formation of the medulla integrate signals from a large number of external sources and their excitement is ultimately what triggers vomiting. Electric stimulation of these centres induces vomiting. The vomiting centre of the brain refers to the groups of loosely organized neurons in the medulla that include the CTZ within the area postrema and the nucleus tractus solitarius (NTS). Chemo receptors in the CTZ relay information about there being emetic agents in the blood to the adjacent NTS. The vomiting centre is activated only by afferent impulses which arise in many parts of the body; it is generally not stimulated directly by emetic substances.
B. The effective stimuli exciting afferent fibres are, tactile stimulation to the back of the throat; distension of the stomach or duodenum to a pressure of about 20 mm Hg; distension or injury of the uterus, renal pelvis or bladder; a rise in intracranial pressure; rotation or unequal stimulation of the labyrinths; acceleration of the head in any direction. The vomiting centers receives afferent signals from at least four major sources:

Afferents (Visceral) from the gastrointestinal tract:

Comprises of vagus or sympathetic nerves, these signals inform the brain of such conditions as gastrointestinal distention and mucosal irritation.

Afferents (Visceral) from outside the gastrointestinal tract: this includes signals from bile ducts, peritoneum, heart and a variety of other organs.

Afferents from extra medullary centres in the brain: Certain specific psychic stimuli (odours, fear), vestibular disturbances (motion sickness) and cerebral trauma can result in vomiting.

The chemoreceptor trigger zone

A bilateral set of centres in the brainstem is lying under the floor of the fourth ventricle. Electrical stimulation of these centres does not induce vomiting directly, but helps trigger the vomiting reflex.

C. The gastrointestinal system is an important source of sensory stimuli. In the gastrointestinal system several neural plexuses exist which are essentially similar in pattern throughout the tubular digestive system. A subserous plexus containing only a few ganglia is found under the serosa. Between the longitudinal and circular smooth muscle layer lies the myenteric or Auerbach's plexus containing prominent ganglia and nerve strands; a muscular plexus containing a few ganglia is situated deep in the circular muscle layer; finally the sub mucous or Meissner's plexus, sometimes consisting of two interconnected plexuses, is found under the mucosa.
D. The crus of diaphragm (pl. crura), refers to one of two tendinous structures that extends below the diaphragm to the vertebral column. There is a right crus and a left crus, which together form a tether for muscular contraction. It prevents rapid expulsion of air in the lungs during the act of vomiting and also allows the stomach to be lowered by lowering physiological barrier. During vomiting, both shortening and EMG activity significantly increase compared to the resting state in the costal segment; lateral crural shortening does not increase in spite of a significant increase in EMG activity; the medial crural lengthens without any increase in EMG activity; the central crural diaphragm shows a significant biphasic length change, with initial shortening followed by lengthening.

Table 1: The vomiting reflex

↓	Target Toxins OR drugs
↓	Activation of CTZ
↓	Stimulates the vomiting centre
↓	Contractions in the gut
↓	Stomach contents forced out of mouth.
↓	VOMITING

Vomiting: The process

Vomiting begins with deep inspiration. The glottis is closed and the nasopharynx partly or completely shut off. Inspiration is converted to an expiratory effort with simultaneous contraction of the abdominal muscles. Because the glottis is closed, the increase in intrathoracic and intra-abdominal pressure is transmitted to the stomach and oesophagus. The body of the stomach and the muscle of the oesophagus relax. At the same time, a strong annular contraction at the angular notch of the stomach nearly divides the body from the antrum. While the body of the stomach remains flaccid, peristaltic waves sweep aboral over the antrum. Due to the positive intrathoracic and intra-abdominal pressure, the gastric contents are expelled out of the mouth. The oesophagus is then emptied partly by the elevated intrathoracic pressure and partly by peristaltic waves stimulated by vomitus in the gut and the mouth. Finally, the voluntary muscles relax and respiration resumes. After a deep breathe, the glottis is closed and the larynx is raised which opens the upper esophageal sphincter. Also, the soft palate is elevated to close off the posterior nares of the nose. The diaphragm is contracted sharply downward to create negative pressure in the thorax, which facilitates opening of the esophagus and distal esophageal sphincter. Simultaneously with downward movement of the diaphragm, the muscles of the abdominal walls are vigorously contracted, squeezing the stomach and thus elevating intragastric pressure. With the pylorus closed and the oesophagus relatively open, the route of exit is clear and hence the vomiting occurs.

DISCUSSION

Sukshma property of medicine allows penetration into minute channels. The pressure gradient developed by anu- Pravanabhava of medicine aided downward movement of doshas from suksma to mahasrotas, thereby accumulating into amashaya and helps to maintain a flow within the channels[9]. Due to the speciality of Vamana drugs these accumulated doshas and malas in the stomach move in the upward direction and gets expelled out, resulting in Vamana karma.

Vayudosha is responsible for the movement of muscles involved in Vamana. Initiation of muscular contraction by Agni mahabhuta and Usnaguna, followed by properties possessed by Vayu mahabhuta makes a forcible contraction of diaphragmatic and inter-costal muscles. Vyavayi and Vikasiguna make the process of Vamana faster.

After collecting all the available information about vamana from the classical texts and segregating the modern knowledge about the vomiting reflex we can draw out certain co- relations in order to understand the process of Vamana in a better way.

Some of the probable correlations for understanding of the mode of action of the vamana aushadha are enlisted as under, classified according the correlations drawn by similarities,

1. According to guna
2. According to prabhava
3. According to lakshanas
4. According to action of drug

According to the guna

Vyavayiguna helps in getting the medicine absorbed faster before getting metabolised completely is what has been explained in the classics, we may interpret it as, a vamana drug possessing this property of vyavayiguna quickly acts on the CTZ which happens to lie outside the blood-brain barrier thereby bringing about the required action. Muscular involvement of the diaphragm and abdomen can be attributed to the vyavayi, teekshna and vikashiguna of the drug as these properties help penetrate deep and work on the innervations of the concerned muscles. Spreading of the drug at deeper cytological level (vishyandana of dosha) by virtue of its sukshmaguna can be attributed to the action of the drug on the gastric plexuses and help in providing sensory stimuli to the vomiting reflex. The ushna, teekshna and saraguna may also help in emptying of gut contents effectively.

Table 2: Guna and its effect

Guna	Effect
Vyavayi guna	Since the CTZ lies outside the blood-brain barrier, the drug has the potency to reach up to the CTZ
Vyavayi, tikshna, Vikashiguna	Stimulates movement of muscles of diaphragm
Sukshmaguna	Stimulates the gastric plexus
Ushna, teekshna, saraguna	Emptying of gut contents effectively

According to Prabhava

Activation of Stretch receptors due to aakanthapana (making the patient drink milk/or any liquid which aids the process of vomiting) through afferent fibres which stimulate vomiting reflex by stimulating the Sympathetic or Vagus nerve can also be correlated to the action of Udaanavayu. As this vayu has been held responsible for Prayatna, urja and, therefore in this case the whole process of initiation of emesis.

Further, the Vamana aushadhi due to their own potency moves tohridaya and then into the sthoola and sookshma srotas, the whole process can be understood as being carried out under the influence of vyanavayu, as it has been explained earlier that the chemicals or alkaloids present in the vamanaushadhis and vamanopagadravyas stimulate the CTZ causing vomiting, so correlation can be drawn on the metabolism of these alkaloids in the body and the action of Vyana in the body. The action of vomiting centre over the whole Vamana process, involvement of higher cortical centres and the effect of ghreyavamaka aushadhi can be compared to the action of Prana vayu because of the role it plays in our body. CTZ is surrounded by the respiratory centre, the vasomotor centre, the salivatory nuclei, vestibular nuclei, and therefore an effect can be seen in the body due to indirect stimulation of these centres merely due to its proximity to the CTZ, thereby also explaining the reason behind the lakshanas mentioned in the classics like Hrullasa (salivatory nuclei), Increase in breathing rate due Prana involvement (the respiratory centre), Indriyashudhi (vestibular nuclei) etc.

Table 3: Vayu

Controlled by Udana Vayu	Activation of Stretch receptors by Akanthapana (afferent fibres which stimulate vomiting reflex) Sympathetic and vagal stimulation
Controlled by Vyana Vayu	Stimulation of CTZ due to alkaloids in vamanaaushadhis and vamanopagadravya are metabolised effectively due to this vayu.
Controlled by Prana Vayu	Action of vomiting centre over the whole Vamana process, Higher cortical centre and ghreyavamaka effect

According to lakshanas

Lately, many studies have been published stating that the gastric plexus help in aiding the process of digestion and assimilation of food by influencing factors like gastric emptying, gut motility etc. It is thought that the myenteric plexus stimulates the muscles to contract in peristaltic waves and that it helps keep muscle tone throughout the intestine walls, promotes secretions of intestinal juices, and allows muscular constrictions (sphincters) to open, thus permitting food to pass from one part of the digestive system to another. It has been earlier explained that the sukshmaguna can be understood as the ability of the vamana drug to act on the level of these gastric plexuses. Correlating these two information we can conclude that the lakshanas like agnivardhana, and dhatu sthirata after samyakavamana are achieved due to the stimulation of these gastric plexuses either due to the effect of the emetic drug or during the process of vomiting.

After the process of vomiting a state of sympathetic and parasympathetic equilibrium is achieved wherein the normalcy of the gut and the body, in general is achieved. Similarly according to the ancient perspective also Manahbuddhiprasadanam and Indriyashudhi is achieved.

Trushna (intracellular water demand), moha, murcha, anilakopa are some of the symptoms which have been included in some of the complications of vamana can be attributed to the increased sympathetic activity post vomiting. Various researches suggest that there is increased histamine release post vomiting reflex, this may be the reason behind certain subjects experiencing sphota, kandu and kotha after vamana as an atiyogalakshana.

Table 4: Lakshanas and site of action of drug

Lakshanas	Site of action of drug
Indriyashudhi , Hridayaprapti, Hrullasa Increase in breathing rate, Prana involvement	CTZ is surrounded by the respiratory centres the vasomotor centre; the salivatory nuclei; vestibular nuclei
Agni vardhana thereby dhatu sthirata	Gastric plexus
ManahbuddhiprasadanamIndriyashudhi	Sympathetic & Para Sympathetic equilibrium
Occurrence of Sphota / kotha/ kandu	Histamine release
Trushna (intracellular water demand), moha, murcha, anilakopa	Increased sympathetic activity

According to action of drug

Discussing about the various routes of carrying the signal for vomiting in the body many tracks have been summarised earlier. For better understanding the mode of action of vamana through these circuits we can sum up that, visceral afferents from the gastrointestinal tract are stimulated by the process of akanthapana by which activation of Stretch receptors takes place. Certain vamanopagadravyas also cause distension in abdomen thereby stimulating the stretch receptors.

Snehapana as poorvakarma, Snighdhayavagu administered just before vamana increases bile secretion (due to high fat content) and gastric stimulation thereby relaying signals to visceral afferents from outside the gastrointestinal tract to induce vomiting. Ghreyavamanaushadhi (emetics which induce vomiting through smell), anabhishtadrushya/ gandha (unpleasant sight/smell) stimulates the afferents from extra medullary centres in the brain thereby causing vomiting. Other than these, there are certain specific alkaloids which act as toxins to directly stimulate the CTZ to induce vomiting.

Table 5: Steps involved in Vamana and Effect on vomiting centre

Steps involved in Vamana	Effect on vomiting centre
Akanthapana	Stretch receptors, Visceral afferents from the gastrointestinal tract
Snehapana as poorvakarma, Snighdhayavagu administered before vamana	Visceral afferents from outside the gastrointestinal tract, Increased bile secretion, gastric stimulation
Adhmana	Visceral afferents from the gastrointestinal tract
Ghreyavamanaushadhi, anabhishtadrushya/ gandha	Afferents from extra medullary centres in the brain
Vamanopagadravyas due to stimulation of gastric mucosa	The chemoreceptor trigger zone

CONCLUSION

Modern pharmacological concepts depend on the data obtained through various *in vitro* and *in vivo* studies, while in Ayurveda, concepts have been explained on the basis of various philosophies or on the basis of effects seen in subjects after many years of trials. Hitherto, these concepts/philosophies have not been scientifically proved but it is seen and documented widely that

these principles do help in managing a disease condition effectively. In order to have a better understanding of the mode of action of these dravyas, in depth studies and experiments should be carried out to gather substantial scientific evidence for the reason behind the effects of these medicines

REFERENCES

1. Murthy KR. Srikantha. Sharanghdharasamhita. Purvakhanda, chapter 4, verses, 7-8 Reprint. Vol. 1. Varanasi: chaukhambha orientalia; 2012.
2. Pulak Kantikar. Comprehensive theory and practice of panchakarma. vol. 1, ed. Delhi: chaukhambha Sanskrit prathishthan; the chaukhambha ayurvigyan studies; 2013. p. 328.
3. Khushwaha HC, Charakasamhita, Ayurvedadeepika. vol.2, 8th ed. Sutrasthana, chapter 1, verse 79, Varanasi: chaukhambha Orientalia, Jaya krishnadasayurveda series; 2018.
4. Khushwaha HC, Charakasamhita, Ayurvedadeepika. Sutrasthana, vol. 2. chapter 1, verse 84, Varanasi: chaukhambha orientalia; 2018.
5. Khushwaha HC, Charakasamhita, Ayurvedadeepika. Sutrasthana, vol. 2, chapter 1, verse 88-9, Varanasi: chaukhambha orientalia; 2018.
6. Khushwaha HC, Charakasamhita, Ayurvedadeepika. vol. 2. Sutrasthana, chapter 1, verse 115, Varanasi: chaukhambha orientalia; 2018.
7. Khushwaha HC, Charakasamhita, Ayurvedadeepika. Kalpasthana, vol. 2, chapter 1, verse 5, Varanasi: chaukhambha orientalia; 2018.
8. Brown HG. The applied anatomy of vomiting. Br Janaesth. 1963; 35(3): 136–45.
9. Nitin UA review on pharmacodynamics of vamana karma and vamanopagadasemāni 2012; 1(11): 6.

How to cite this article:

Sangeeta S. *et al*. A comprehensive review on vamanaushadha karmukata. J Pharm Sci Innov. 2020;9(1):5-9.
http://dx.doi.org/10.7897/2277-4572.091161

Source of support: Nil, Conflict of interest: None Declared