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Case Study

A CONCEPT OF *VIRECHANA KARMA* IN AYURVEDA WITH SPECIAL REFERENCE TO AUTOPHAGY

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ABSTRACT
Virechana Karma is one of the five *Shodhana* therapy. It is specifically designed for the therapeutic cleansing of the body by inducing controlled purgation. It is one of the ten *Langhana* therapy which produces *Laghuta* in body by eradication accumulated of *Doshas* through *Rechana* (purgation). It is indicated in *Bahudoshaavastha* like *Avipaka*, *Aruchi*, *Sthaulya*, *Panduta* etc. Autophagy is body cleansing mechanism found to have therapeutic value in certain diseases like, some metabolic disorders, infection, cancer, neurological diseases. It is a self-degradative process having significant role in important balancing sources of energy at critical times in development and in response to nutrient stress. Researches have shown that fasting helps to induce autophagy, thus latest research also approve the effectiveness of therapeutic measure developed by ancient ayurvedic scholars. Hence *Virechana* is consider as one of the ten *Langhana* therapy which literally means the act which induced lightness, so that we can consider *Virechana* as kind of fasting therapy hence it produced *Laguta* into body by eliminating *Prakupita Dosha*. Also, *Virechana Karma* and autophagy are both cleansing processes aimed at removing accumulated waste and restoring balance in the body. Both are activated under metabolic stress and promote detoxification, homeostasis and improve physiological function. **Objective:** 1. To study the concept of *Virechana Karma* in Ayurveda. 2. To study the corelation of *Virechana Karma* with autophagy.

INTRODUCTION

The concept of Autophagy

Autophagy is a conserved catabolic process that is involved in cellular homeostasis and is required to maintain normal cellular physiology under stressful conditions. The primary function of autophagy (macroautophagy) is to recycle and re-uptake nutrient from the cytoplasm during condition of metabolic stress and to degrade specific cytosolic components, such as damaged mitochondria through mitophagy or intracellular bacteria through xenophagy. It overcomes carcinogenic, infectious, degenerative, and deleterious agents to maintain the homeostasis of bodily systems and regulate healthy life processes; thus, its dysregulation is known to cause multiple human diseases. Autophagy can be a selective or non-selective lysosomal degradative process and is activated by stresses such as starvation or rapamycin via regulatory signalling complexes. There are three types of autophagy: macroautophagy, microautophagy, and chaperone-mediated autophagy (CMA). the roles of autophagy have been explored in fields such as health, disease, infection, degeneration, and genetic or lifestyle-acquired diseases. Autophagy might play both physiological and pathological roles since it is involved in overcoming cell stresses. Cellular stress such as nutrient deprivation or low energy status activates AMPK, the cell's primary energy sensor, which simultaneously inhibits mTORC1, a key growth regulator that normally suppresses autophagy under nutrient-rich conditions. The inhibition of mTORC1 and activation of AMPK together stimulate the ULK1 complex (ULK1–ATG13–FIP200), which serves as the

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initiation complex of autophagy. Activated ULK1 then promotes the PI3K complex consisting of VPS34 and Beclin-1, leading to the generation of PI(3)P and the formation of the phagophore, the initial isolation membrane. Membrane elongation and expansion are mediated by the ATG5–ATG12–ATG16L1 complex, while LC3 is first cleaved by ATG4 to form LC3-I and then conjugated with phosphatidylethanolamine to form LC3-II, which anchors to the autophagosomal membrane and facilitates its maturation. LC3 and GABARAP proteins help shape the developing autophagosome, and cargo receptors such as p62, NDP52, optineurin, and NBR1 recognize ubiquitinated cellular components and link them to LC3-II for selective sequestration. The mature autophagosome then fuses with the lysosome through the action of Rab7, SNARE proteins, and lysosomal membrane proteins such as LAMP, forming an autolysosome. Within the autolysosome, lysosomal hydrolases degrade the enclosed material into basic biomolecules such as amino acids and fatty acids, which are recycled back into the cytoplasm to restore energy balance and maintain cellular homeostasis.[2,3,4]

The question arises here that whether, this concept is new for Ayurveda science? Our ancient science is said to be *Anadi, Anant, Swayam-Sansiddha*. This concept is hidden in our science, we need to explore it.

The concept of *Virechana karma*

Virechana Karma is the process of expelling *Doshas* through *Adhobhaga (Guda).*[5] *Virechana* implies the use of *Aushadha* that induced purgation. It is indicted in multiple conditions viz, *Kushta, Prameha*, *Swasa-Kasa, Apasmara, Pakshaghata, Amavastha (Amavata, Avipaka, Aruchi), Pandu, Kamla* etc.[6] In of proper purgation symptoms *like Laghuta, Daurbalya, Glani, Hridasudhhi, Kale Kshudha Pravritti, Kale Trishna Pravritti, Kale Vega Pravritti, Vatanulomana, Buddhi* & *Mana suddhi*, *Srotasa Visuddhi* and *Agni Vriddhi* are induced in body. [7] But in case of excessive purgation symptoms like *Suptata, Angamarda, Klama, Vedana, Nindra-Bala nasha, Tamapravesha* and other symptoms of *Vata Vriddhi* and *Kapha-Pitta Kashaya* are induced in body.[8] The end goal of the *Virechana Karam* is to get *Samyaka Virechana Lakshana* to achieve main aim of Ayurveda which is *Dhatusamyata* by removing *Utkeshita Dosha*. While understanding mode of action of *Virechana Karma* according to modern science, it understands that *Virechana Aushadha* stimulate hypothalamus and neuclius tractus solitary which stimulated ANS and then ANS increases parasympathetic activity to promote purgation. Hence purgation decreases oxidative stress by removing gut derived pro-oxidant, improved bile-mediated antioxidant signalling, reducing inflammatory cytokines, rebalancing autonomic activity and restoring mitochondrial and redox homeostasis.[9] Which is compared with elimination of *Dushita Pitta Dosha* and *Ama Dosha*, improvement of *Agnibala, Dhatu Samyata, Urja Vriddhi (*or *Ojasa Rakshana).*

In the context of Ayurveda, the classical process of *Virechana Karma* is systematically organized into *Trividha Karma* viz., *Purva Karma, Pradhana Karma* and *Paschata Karma.*

***Purva Karma*:** The essential preparatory phase before any main procedure is called *Purva Karma.* The *Purva Karma* administered prior to any *Shodhana Karma* involves a sequence of three essential procedure viz., *Deepana-Pachana, Snehana and Swedana*.

***Deepana-Pachana*:** Drugs that quantitatively and qualitatively increase *Agni* are termed as *Deepana-Pachana Dravyas*.[10] Medicines that bringing state of *Nirama*, which is essential for an effective *Snehapana* and proper conduction of *Shodhana* therapy. If *Shodhana* therapy is administered in an *Ama* condition, it will destroy the body in same way that extracting juice from unripe fruit, and there is a chance of working the therapy in the opposite direction.[11]

Deepana–Pachana is administered before *Shodhana* to correct *Mandagni,* digest *Ama*, and remove *Srotorodha*. When *Agni* becomes weak, improperly digested food forms *Ama*, which is heavy, sticky, and obstructive in nature, blocking the channels and preventing proper elimination of *Doṣhas*. *Deepana* kindles *Jatharagni* and *Dhatvagni*, thereby improving digestive capacity, while *Pachana* digests the already formed *Ama* and transforms it into a non-toxic and movable state. The action of *Deepana–Pachana* depends mainly on *Deepana-Pachaniya Gravya's Guṇas* such as *Laghu* (light), *Rukṣa* (dry), *Tikṣhṇa* (sharp), and *Uṣhṇa* (hot). *Laghu Guṇa* reduces heaviness and makes digestion easier; *Rukṣa Guṇa* absorbs excess moisture and decreases the stickiness of *Ama; Tikṣhṇa Guṇa* penetrates deeply into the *Srotasa* and breaks obstruction; and *Uṣhṇa Guṇa* stimulates *Agni* and liquefies accumulated *Doshas*. Due to these combined qualities, Ama becomes lighter, less viscous, and suitable for movement toward the *Koṣṭha*. Thus, the channels are cleared, *Agni* is normalized, and vitiated *Dosha*s are properly mobilized, ensuring effective and safe elimination during *Shodhana Karma*.

***Shodhanartha Snehapana*:** Large dose of therapeutic *Sneha Dravya* is used for internal administration in increasing manner to fulfil the purpose of *Dosha Utkleshana* is called *Shodhanartha Snehapana. Shodhanartha Snehapana* is administer orally when previously ingested food gets digested to avoid digestion of *Dosha* which are present in *Srotasa* and to achieve *Dosha Utkleshana* by intaking *Madhyama*

Matra of *Sneha*.[12] Chemically, fat contains triglycerides rich in saturated and short-chain fatty acids like butyric acid, along with fat-soluble vitamins. After oral intake, fat undergoes digestion in the small intestine through the action of pancreatic lipase and bile salts. The triglycerides are emulsified, broken into free fatty acids and monoglycerides, absorbed by enterocytes, and packed into chylomicrons, which enter the lymphatic circulation and then systemic circulation. From a modern perspective, this high lipid load temporarily increases circulating lipids and stimulates bile secretion from the liver. At the cellular level, repeated administration of increasing doses creates a metabolic state similar to short-term lipid adaptation. Fatty acids activate nuclear receptors such as PPAR-α (Peroxisome Proliferator Activated Receptors), which regulate genes related to lipid metabolism, detoxification enzymes, and anti-inflammatory pathways. This enhances β-oxidation and mobilization of stored lipids from adipose tissue. Since many environmental toxins, inflammatory mediators, and oxidized lipids are lipophilic and stored in adipose tissue membranes, increased lipid turnover may mobilize these compounds into circulation. At the hepatic level, increased fat intake stimulates bile production and flow. Bile acids act not only as digestive agents but also as signaling molecules via FXR (Farnesoid X receptor) pathways, influencing lipid metabolism and intestinal barrier function. Enhanced bile flow helps carry cholesterol, bilirubin metabolites, and lipid-soluble waste toward the intestine, which correlates with the Ayurvedic concept of "*Utklesha*" (mobilization of *Dosha* toward *Koṣṭha*). In other hand while understanding of mode of action of high therapeutic dose of fat ingestion due to high dose nutrient flux is generated in endoplasmic reticulum which temporary increases cellular stress. [13]

***Bahya Abhyanga* & *Swedana*:** The combination of both *Abhyanga* and *Swedana* works synergistically to melt toxins deeply embedded within the body's subtle channels and to liquefies the *Leena Dosha*.[14] When *Abhyanga* and *Swedana* are done after internal oleation: Internal *Snehapana* mobilizes lipophilic substances from cells and adipose tissue. *Abhyanga* enhances peripheral circulation and lymphatic flow. *Swedana* liquefies interstitial contents via vasodilation and heat. Mobilized metabolites move toward central circulation. In simple modern terms, *Bahya* abhayanga and Swedana convert"stable stored metabolic waste" into a "circulating eliminable form". *Swedana Karma* causes vasodilation, Heat shock protein activation and generate ROS. [15,16,17]

***Pradhana Karma*:** *Virechana Karma* is a specialized Ayurvedic *Panchakarma* procedure define as controlled therapeutic purgation achieved through the administration of specific, calibrated dose of *Virechaka Aushadha* which is designed to eliminated accumulated toxins and morbid *Doshas*, primarily *Pitta* and *Pitta Samsrishta Tridosha* to clear obstruction of *Srotasa* and ensure unobstructed flow of *Ahara Rasa (Poshakatatva)* and *Prana (Urja). Virechana Karma*, as described in classical Ayurvedic texts like *Charaka Samhita*, can be explained in modern physiology as a controlled therapeutic purgation that acts through coordinated gastrointestinal, hepatobiliary, neuro-enteric, and autonomic mechanisms. After proper *Snehapana* (internal oleation) and *Swedana* (sudation), lipid-soluble metabolites, inflammatory mediators, and fat-stored toxins are mobilized into circulation and shifted toward the gastrointestinal tract via bile secretion. When *Virechana* drugs (commonly containing stimulant or osmotic purgative principles) are administered, they primarily act on the small intestine and colon. At the intestinal level, these drugs stimulate enterochromaffin cells in the mucosa, leading to release of serotonin (5-HT). Serotonin activates intrinsic primary afferent neurons of the enteric nervous system and also stimulates vagal afferent fibers. The vagus nerve (cranial nerve X) carries sensory signals from the gut to the nucleus tractus solitarius in the brainstem. This gut-brain signaling enhances parasympathetic outflow back to the gastrointestinal tract, increasing coordinated peristalsis and intestinal secretion. Thus, *Virechana* involves a vasovagal reflex mechanism- afferent signaling from gut to brain and efferent parasympathetic stimulation back to the gut. Simultaneously, purgative agents increase chloride ion secretion into the intestinal lumen via activation of secretomotor neurons in the submucosal plexus. Sodium and water follow osmotically, leading to liquefaction of stool. Prostaglandin release and mild mucosal irritation enhance smooth muscle contraction through myenteric plexus activation, producing strong propulsive peristaltic waves (*Vega*). The vagus nerve modulates this activity by enhancing coordinated motility and promoting relaxation of sphincters (especially pyloric regulation during earlier phases and small intestinal transit) to stimulate smooth and control purgation.[18,19,20]

Paschata Karma: *Paschat Karma* refers to post-procedural care taken after *Pradhanakarma* to maintain *Agni Bala* and *Sharira Bala*. After *Virechana Karma Paschatakarma* generally involved two core components viz, *Samsarjana Krama* to maintain *Agni Bala*[21] and *Pariharya Vishaya* to maintain *Sharira Bala*. Following *Virechana*, the gastrointestinal mucosa remains in a temporarily sensitive and hypo-functional state due to repeated peristalsis, fluid loss, electrolyte shifts, and reduced digestive enzyme activity.

Saṃsarjana Karma begins with easily digestible, low-residue, semi-liquid foods (such as rice gruels), which are low in fat and protein but adequate in simple carbohydrates. These foods require minimal gastric acid, pancreatic enzymes, and bile for digestion, thereby preventing overloading of the recovering digestive system. Gradual progression from liquid to semi-solid and then solid diet allows stepwise reactivation of gastric secretion, pancreatic enzyme release, bile flow, and intestinal motility. At the mucosal level, mild carbohydrate-rich diets promote rapid glucose absorption, which supplies energy to enterocytes (intestinal epithelial cells). This supports regeneration of villi, restoration of tight junction proteins, and normalization of intestinal permeability. The progressive diet also helps stabilize electrolyte balance (especially sodium and potassium), preventing post-purgation fatigue and hypotension. [22,23,24]

MATERIAL AND METHODS

This study is a conceptual review and theoretical analysis based on classical Ayurvedic texts and modern biomedical literature available on internet in form of research article.

Classical descriptions of *Virechana Karma* including its procedural stages *Purva Karma, Pradhana Karma, and Paschat Karma* were studied from *Ayurvedic* literature. Modern physiological mechanisms related to gastrointestinal motility, liver–gut axis, bile acid metabolism, oxidative stress, and cellular signaling pathways involved in autophagy were reviewed from contemporary scientific sources. The theoretical correlation was established by comparing the physiological changes occurring during each stage of *Virechana* therapy with known molecular triggers and pathways involved in autophagy, such as AMPK activation, mTOR inhibition, ROS signaling, and lysosomal degradation pathways.

DISCUSSION

The three *Doshas (Vata, Pitta & Kapha), Saptadhatu (Rasa, Rakta, Mamsa, Meda, Asthi, Majja & Shukra)* and *Mala (Purisha, Mutra & Sweda)* are the basic component of body constitution. *Doshas* are fundamental bio-energies that govern all physical, mental and emotional processes. While mentioning *Dosha Acharya* says '*Dushyanti Iti Doshah*, means which have tendency to vitiate both *Dhatu* and *Mala*. To maintain *Swathya* its necessary to *Dhatu Samyata* which is achieved through balancing all three *Doshas* through two-fold of therapies viz, *Langhana* and *Brumhana*. Hence *Virechana* is one of the ten *Langhana* therapy which induce purgation to eradication of vitiated *Doshas* to maintain *Dhatu Samyata*. In other hand Autophagy is a cellular catabolic activity which maintain homeostasis. *Virechana* helps in maintaining *Doshas* equilibrium, induces lightness in the body, maintain oxidative stress balance and generates energy in other hand Autophagy helps in maintaining homeostasis of body, induces lightness in the body, manage oxidative stress and generate ATP during catabolism process.

We can hypothesize that the tree-steps protocol of *Virechana Karma* function as a biological catalyst for autophagy as follow:

Deepana-Pachana:- *Aushadha* use for *Deepana-Pachana* has contains alkaloids as an active pharmaceutical ingredients for example, *Pippali* (*piper longum*) and *Maricha* (*piper nigrum*) contain piperine which act as deactivation of mTORC1 protein and induce ULK1 and BECNI gene expression to inhibit proliferation which is the initiate autophagy to induce apoptosis and this whole process is happens at cellular level.

Shodhanartha Snehapana: Therapeutic large quantity of *Sneha Dravya* leads to a temporary spike in free fatty acids, triggering endoplasmic reticulum stress and a paradoxical activation of AMPK due to nutrient overload, which ultimately destabilize mTOR activity through transient metabolic stress. Hance activation of AMPK and destabilization of mTOR is initiate autophagy at systemic level.[25]

Bahya Abhyanga and Swedana: The application of heat causes dilation of superficial blood vessels, which increases peripheral circulation and enhances tissue perfusion. This rise in temperature activates heat shock proteins and promotes cellular protective mechanisms, including Nrf2 pathway activation. Mild thermal stress leads to controlled production of reactive oxygen species (ROS), which act as signaling molecules rather than causing damage. As metabolic activity increases due to heat exposure, oxygen consumption also rises, resulting in enhanced electron flow through the mitochondrial electron transport chain (ETC). The increased ATP demand during this process can cause slight electron leakage from the ETC, leading to limited superoxide formation. This mild oxidative stimulus further triggers adaptive cellular responses, improves microcirculation, facilitates removal of metabolic wastes, and supports the therapeutic effects of *Swedana Karma* such as reduction of stiffness, heaviness, and obstruction in channels (*srotoshodhana*). [26,27]

***Virechana Karma*:** During *Virechana Karma*, the Liver–Gut axis functions as an integrated pathway of detoxification and elimination. The liver continuously produces bile containing bile acids, bilirubin, cholesterol, metabolic by-products, and various endogenous and exogenous toxins, which are secreted into the small intestine. Normally, a large portion of

bile acids is reabsorbed through enterohepatic circulation and returned to the liver. However, during *Virechana*, purgative drugs stimulate intestinal motility and secretion, accelerating the downward movement of intestinal contents and reducing the time for reabsorption. This temporary interruption of enterohepatic circulation promotes increased fecal excretion of bile acids and bile-bound toxins. The rapid evacuation creates a functional gradient that encourages continued bile secretion (choleresis), thereby enhancing hepatic clearance of lipid-soluble waste products, inflammatory mediators, and excess metabolites. Simultaneously, intestinal cleansing reduces microbial load and endotoxin absorption into the portal circulation, thereby decreasing hepatic inflammatory stress and improving metabolic efficiency. In Ayurvedic terms, this coordinated Liver–Gut interaction reflects the process of *Pitta Shodhana*, as *Pitta* is closely associated with bile and hepatic function; thus, *Virechana* facilitates elimination of vitiated *Pitta* from its principal seat, restoring systemic metabolic balance. In hepatic autophagy deficiency, there is not only an increase in total bile acid levels but also a significant alteration in their composition, particularly an increase in unconjugated bile acids. These altered bile acids activate intestinal FXR receptors and stimulate FGF15 production, forming a compensatory liver–gut feedback mechanism. However, persistent elevation and qualitative modification of bile acids can produce hepatic stress, inflammation, and metabolic imbalance. In the context of *Virechana Karma*, this condition can be correlated with *Pitta Vriddhi* and *Pitta Dushti*, especially involving the *Yakrita* (liver) and *Grahani* (small intestine). Bile acids functionally resemble *Pitta* because both are responsible for digestion, transformation, and metabolic regulation. When bile acids become excessive or qualitatively irritative, it parallels the state of *Prakupita Pitta* circulating in the *Amashaya–Pakvashaya* region. *Virechana,* being a specific therapy for elimination of aggravated *Pitta* through the lower route, may help by promoting excretion of excess bile acids, reducing their enterohepatic recirculation, decreasing hepatic overload, and modulating gut microbiota composition. By eliminating the excessive and altered bile acid pool, *Virechana* may assist in restoring bile acid homeostasis, normalizing Liver–Gut axis signaling, and re-establishing systemic metabolic balance. Thus, the presence of high and compositionally altered bile acids provides a modern physiological justification for the therapeutic role of *Virechana* in *Pitta*-dominant liver disorders. [28]

Samsarjana Krama: After *Virechana,* the body enters a transient catabolic and low-nutrient state in which glycogen stores are depleted, insulin levels are reduced, and cellular energy stress increases. This activates AMPK and suppresses mTOR, the key nutrient-sensing pathway, thereby naturally upregulating autophagy. In this phase, the ULK-1 complex is activated and autophagosomes begin forming to sequester damaged proteins, dysfunctional mitochondria, and accumulated metabolic waste. When *Samsarjana Krama* is initiated with *Peya*, the extremely light and low-protein intake does not abruptly stimulate mTOR, allowing autophagy to continue at a mild but effective level while *Agni* gradually rekindles. As the diet progresses to *Vilepi,* caloric intake increases slightly, causing a gentle rise in insulin without suddenly shutting down autophagic activity; autophagic flux continues efficiently, promoting degradation of cellular debris and supporting intestinal epithelial repair. With the introduction of *Akruta Yusha* and subsequently more substantial foods, mTOR activity gradually resumes, shifting metabolism from a predominantly catabolic, autophagy-driven state toward controlled anabolism and tissue rebuilding. By this stage, major cellular cleansing is largely complete, and the body transitions smoothly to regenerative processes such as protein synthesis, mitochondrial biogenesis, and *Dhatu Poshana*.

CONCLISION

Based on the aforementioned theories, it can be hypothesized that *Virechana Karma* indices a systemic autophagic effect, facilitating cellular rejuvenation throughout the body through eliminating excessive *Pitta Dosha*, decreasing inflammatory cytokine, enhancing gut epithelial turnover and promoting mitochondrial biogenesis. We can understand that *Virechana Karma* works at systemic level by eliminating *Doshas* and *Ama* through purgation, while autophagy functions at cellular level by degrading damaged components. Thus, *Virechana Karma* can be viewed as a macroscopic equivalent of autophagy.

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