Published online in http://ijam.co.in ISSN No: 0976-5921

International Journal of Ayurvedic Medicine, Vol 13 (4), 2022; 828-835

A Narrative Review on the etiopathogenesis of Primary Dysmenorrhea with reference to *Kastartava* in Ayurveda literature

Review Article

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Abstract

Objectives: Different dimensions of Primary Dysmenorrhea in Ayurveda literature has been explored and conceptualised it with the available physiological and anatomical understanding. Content: The concept of Primary Dysmenorrhea is better understood upon the basic understanding of *Kastartava* dating back to the phase of adolescence. 75% of adolescent girls' experiences problems related to menstruation including delayed, irregular, painful, and heavy menstrual bleeding. Among these, painful menstruation or dysmenorrhea is the most common gynecological problem seen in reproductive age group. "Primary dysmenorrhea, one among its two types, is a cyclical lower abdominal or pelvic pain of sufficient magnitude so as to incapacitate day to day activities without any identifiable or evident pelvic pathology". Ayurveda classics were reviewed to analyse the various aspects of *Kastartava* and correlated with research papers in terms of evidence-based physiological and anatomical advancements. Databases like PubMed, Scopus were searched using keywords like *Shoola, Kastartava, Udavartini, Vatiki Yonivyapat*, Dysmenorrhea, Primary Dysmenorrhea AND Ayurveda, Pain measurement, Quality of life, Inflammatory Markers, Prostaglandin theory. Summary: Various aspects of *Shoola* and *Kastartava* in Ayurveda from evidence-based advancements in Primary Dysmenorrhea based on recent researches have been put forward for basic evidence in relation with Pathophysiology of Primary Dysmenorrhea. Outlook: Understanding the pathophysiology of pain in Primary Dysmenorrhea with respect to the clinical presentation of *Kastartava* will give new insights to manage the clinical condition.

Key Words: *P*rimary Dysmenorrhea, *Kastartava, Shoola, Ayurveda,* Prostaglandins, Inflammatory Markers, *Udavartini Yonivyapat.*

Introduction

The menstrual function in a female is deemed to be one of the main factors reflecting the functional potentiality of women which will be disturbed by some sort of discomfort in more than 50% of women (1). Among them, dysmenorrhea is one of the most common gynecological conditions causing several problems in the personal as well as social life of a woman as the cyclical pain associated with menstrual cycle is so as to incapacitate day to day activities (2). Epidemiological survey studies suggest that even though prevalence rates vary by geographical area, symptoms of dysmenorrhea are widespread in varied population, in a range between 28% and 71% (3, 4). According to 2006 health report of WHO, the prevalence rate of dysmenorrhea was found as 1.7-97 % (5). Among the disease spectrum dysmenorrhea, Primary dysmenorrhea (PD) is the most common cause of pelvic pain in women particularly in the adolescent age group. The studies have shown that approximately 90 % of the female population suffers from this condition and among them, 10% are affected with severe forms for 1–3 days monthly during each menstrual cycle (6). The resulting sickness absenteeism causes severe economic loss each month due to its psychological and economic impact (7). Though our country has amazing achievements in the field of adolescent and women health, the overall rating in the prevention and cure of primary dysmenorrhea is not good. Recently the researcher pioneers in the field, George and Bhaduri, concluded that there is a tremendous increase in the incidence of the disease rate from 34% to 87% in India (8). Even though dysmenorrhea is not a life-threatening condition (9), health-related Quality of life is seen to be disturbed among adolescent population, due to its negative effects on the psychological status (10). Despite the negative effect on quality of life and general health, few women with dysmenorrhea will not seek treatment as they believe it would not help. On the other hand, a study has shown excessive use of over-the counter analgesics (53%) and NSAID'S (42%) (11). Although the certain cause of primary dysmenorrhea is unknown, one accepted causative factor is the excessive production of

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endometrial prostaglandins therefore, reducing the level of prostaglandin are thought to be effective. Although there have been various methods recommended to cure dysmenorrhea till date, due to the possible NSAID resistance over a period of time, many patients are now moving towards alternatives in herbal medicine (12). The studies have shown evidence that bodily metabolism plays an important role in the cause and treatment of menstrual disorders. By understanding the importance of bodily metabolism by counteracting the inflammatory pathology, in the treatment of menstrual disorders, the usage of herbal preparations in the treatment of the primary dysmenorrhea is increasing worldwide (13, 14). In certain conditions like *vatiki yonivyapat* (15), *vataja artava duṣṭi* (16), *udavartini yonivyapat* (17) etc. pain occurs during menstruation. To regulate uterine contractions and uterine tone, many effective ayurvedic regimens are described in ayurvedic classics and various studies have also been conducted; but a proper understanding and interpretation of the disease spectrum is not available, furthermore the documentation of these data in terms of research is lacking.

Aims and objectives

- To explore the various aspects of *Shoola* and *Kastartava* in Ayurveda.
- To correlate the concept of *Kastartava* in Ayurveda with modern biological and anatomical understanding

Materials and methods

Ayurveda classics were reviewed to analyse the various aspects of *Kastartava* and correlated with research papers establishing the concept of in terms of evidence-based physiological and anatomical advancements. Databases like PubMed, Scopus were searched using keywords like *Shoola, Kastartava, Udavartini, Vatiki Yonivyapat*, Dysmenorrhea, Primary Dysmenorrhea AND Ayurveda, Pain measurement, Quality of life, Inflammatory Markers, Biomarkers, Prostaglandin theory with the help of Boolean operators 'AND', 'OR' and 'NOT'. Filters like Clinical trials, within five years and free full text were applied.

Review of Literature

Concept of *Kashtartava*

Kashtartava is a symptom mentioned in various *yonivyapad*, *vata* being the main causative factor for this condition. As it is painful menstruation, it is commonly compared with dysmenorrhoea of contemporary science. The *Nidana* of *Kaṣṭartava* is not explained separately in Ayurvedic classics, though *Kaṣṭartava* is found as a symptom in following *Yonivyapat (Vatiki, Sannipatika, Paripluta, Udavartini and Mahayoni), Ārtava duṣhṭi* and *Asrgdara.*

Anatomical and Physiological pathways regulating uterine contractility

As the contraction of uterus plays a key role in the reproductive physiological activities, including menstruation, an attempt has been made for a detailed collection of literary review on the concept. There has been a great progress towards the understanding of the applied anatomy and the pathophysiology of reproductive axis, and this has resulted in many important interventions to regulate the menstruation. Hence a detailed understanding of urogenital anatomy and physiology is essential to formulate and test interventions that can treat various gynaecological conditions like dysmenorrhea.

Clinical Anatomy of Primary Dysmenorrhea

Uterus is a hollow, pear- shaped organ with thick muscular walls. It measures 8 cm long, 5 cm wide, 2.5cm thick. Length of cavity is 6 cm, thickness of muscle wall is about 1.2 cm. It is divided into fundus, body, cervix. They are quite distinct in function and in structure although the transition from musculature to fibrous tissue is gradual. The cavity of the cervix, the cervical canal, communicates with the cavity of the body through the internal os and with that of vagina through the external os (18). The external os is small before parturition and is sometimes called the os tincae. After the birth of a child it becomes a transverse slit as that of parous os. The cervical canal is having fusiform appearance and marked by folds called the arbor vitae. The anteflexed and anteverted position of uterus serves as a key factor for the normal menstrual function. Thus, in the erect position and with the empty bladder, the uterus lies in an almost horizontal plane by regulating the uterine tone (19). Based on the evidence that myometrial function plays a vital role in various gynaecological disorders including dysmenorrhea, the histological layers of uterus is to be understood. Among the three layers of uterus, the myometrium consists of three layers of smooth muscle fibres that are thickest in the fundus and thinnest in the cervix. The inner layer of uterus, endometrium is highly vascularized which is composed of an inner most layer composed of simple columnar epithelium with ciliated and secretory cells, an underlying endometrial stroma with a thick region of lamina propria which contains stromal cells, vessels and nerves and endometrial glands which are simple tubular in type lined by non- ciliated columnar epithelium which extend to myometrium. All these are attributed for changes components are changed during menstrual cycles. Endometrium is divided into 2 layers; stratum functionalis lines the uterine cavity and sloughs during menses. The deepest layer, stratum basalis is permanent and gives rise to a new stratum functionalis after each menstruation (20).

The blood collected for whole month by the *dhamani* (uterine vessels and their endometrial capillaries) assuming slight black colour and specific odour is brought downwards to the vaginal orifice for expulsion (21). *Acarya Viswamitra* has clarified that hair like thin vessels fill the uterus for whole month to receive *bija*. As believed by *Acarya Kasyapa*, the blood in adult females during their reproductive period enters into *garbha kosa* every month and there are *rajovaha sira* in the uterus which are the carriers of *artava* formed by the action of *agni* upon the *rakta dhatu* and they fills the uterus in one month and after that this

artava is expelled out by these *sira* at the interval of one month (22).

Contractile Apparatus in Primary Dysmenorrhea

As the uterine myometrial contraction in a regulated fashion is needed for a normal menstrual flow, the knowledge regarding characteristics of uterine contractions is necessary. Uterine contractions occur throughout the menstrual cycle in the non- pregnant as well as in the pregnant state. Over the past several years, remarkable information regarding myometrial contractions in non- pregnant women has been obtained from the use of 3D Ultrasound or MRI (23, 24). It is evident from the previous studies that, the contractions in the non- pregnant uterus appears to be different from that of a pregnant one (25). Recently imaging techniques have shown that these contractions are seen to be involving only the sub- endometrial layer of the myometrium. All these recent findings have led to a new concept of uterine applied anatomy that incorporates two separate zones of the myometrium. The innermost layer of muscle fibres which are arranged in a circular fashion around the uterine cavity is distinct and unique from the outer layer in its physiological properties as well as in various pathological conditions including dysmenorrhea, attributed to its different embryological origin. It has been stated recently that the inner junctional myometrium is derived from the paramesonephric ducts, but the outer myometrial layer originates from the non - Mullerian tissue (26). The presence of a functional inner circular layer of myometrium, could explain the mechanism for a peristaltic and anti-peristaltic activity that is well documented through the menstrual cycle depending on the local hormonal activity (27). The explanation of *pesi* as explained in the Ayurveda classics can be considered here. As in all muscle tissue, the predominant proteins expressed in uterine smooth muscle myocytes are myosin and actin. In case of uterine myocyte, there is 5-6fold more actin than myosin (28). "According to the research works on the uterine muscle activity, for achieving the uterine contraction, a force must be transmitted along the actin filaments from the longitudinal pole of the cell towards the centre of the cell and the actin filaments must be firmly attached to the myocyte". Accessory proteins including tropomyosin, calponin, etc. plays a remarkable role in regulating contraction. Uterine smooth muscle has a phasic pattern of contraction by regulating the resting tone potential with intermittent contractions of different frequency, amplitude and duration which is regulated predominantly by Calcium ions (29, 30).

It is stated that all the biological activities in the body are under the control of *Vyana vayu*. For the production of *artava, Vyana and Apana vayu* work friendly. Contraction and relaxation of the uterus and its related organs are done by *Vyana vayu* (31) after that, *artava* is expelled out by *anulomana kriya* of *Apana vayu* (32).

Neuro-Vascular Framework in Primary Dysmenorrhea

One of the main aims of treatment of menstrual pain is to regulate the lymphatic and venous drainage from the pelvis and to ensure balance of the nervous system. Branches of internal *iliac* artery which are called as uterine arteries, supply blood to uterus. These uterine arteries give off branches called arcuate arteries that are arranged in a circular fashion in the myometrium. These arteries again branch into radial arteries which penetrate deeply into myometrium. Just before the branches enter the endometrium, they divide into two kinds of arterioles in which the straight arterioles supply the stratum basalis and helps in the regeneration of stratum functionalis and the spiral arterioles supply the stratum functionalis and change markedly during the menstrual cycle. Blood leaving the uterus is drained by uterine veins into internal *iliac* veins. Nerve supply to the uterus is principally derived from sympathetic chain and partially from parasympathetic chain. The lower abdominal cramping pain associated with dysmenorrhoea are mediated through sympathetic afferents and hence may be referred to appropriate segments. "Sympathetic components are those from the motor segments of T5 and T6 and sensory segments of T10 to L1. The parasympathetic system is represented on either side by the pelvic nerve". The optimal functioning of spinal segments can help maintaining the input from the nervous system to the pelvic organs. The recent studies have shown that, for the treatment of dysmenorrhea from an anatomical base, a chance of improvement is there by improving the blood circulation and nerve supply to the pelvis.

Artavavaha Srotas

According to classics, *Artavavaha srotas* are two in number, having its *mula sthana* in *garbhasaya* and *Artavavaha Dhamani*. On injury to these, produces *vandhyatva, maithuna asahishnutva* and *artava nasa*. These *Srotas* may be considered as uterine arteries specially their capillary bed (33). *Artava Utpatti* is contributed to be from *Rasa Dhatu* (34)*, Rakta Dhatu* (35)*, Ahara Rasa* (36), *Upadhatu of Rasa Dhatu* (37) and *Upadhatu of Rakta* (38). According to Acarya Caraka, *suddha artava* has flow without pain, sliminess and burning sensation (39).

Neuro-endocrinology in Primary Dysmenorrhea:

The endocrine mechanism is the basic factor in the female reproductive cycle. The menstrual cycle is a transformative cycle, generated by the interplay of secretory sites, the hypothalamus, the anterior pituitary, and the ovaries, and of the hormones they produce and to some extend are influenced by the thyroid and adrenal glands. It leads to real changes in reproductive structures so that the relevant applied anatomy actually changes through the monthly cycle. The hormonal secretions appear to be the outcome of a rhythmic pulse. The anatomical changes in ovaries with the development and ripening of the follicle to ovulation and the formation of the new secretory apparatus, the

corpus luteum can be seen to provide the structural mechanism for the ovarian cycle. Any deviation in the hormonal pathway will alter the ovarian cycle and thereby the menstrual cycle. Associated with the monthly cyclical production of estrogen and progesterone by the ovaries in the ovarian cycle, there is an endometrial cycle in the uterus that functions through certain stages; 1) Regenerative phase 2) Proliferative phase 3) Secretory phase 4) Menstrual phase among which the physiology of secretory phase and menstrual phase plays a key role here.

All *Acarya* have mentioned twelve years as the age of menarche. There is no controversy regarding time of first *artava darshana*. Only in Kashyapa Samhita, it is mentioned that sixteen years is the age of menarche, it might be the description of approximate age for conception (40). He further says that this age can be influenced by specific *Ahara* and *Arogya*. *Artava Utpatti Hetu* particularly the *Kala, Dhatu paripurnata* and *swabhava* are attributed for the initiation of a proper Neuroendocrinological pathway and maintenance of neuroendocrinological homeostasis. According to Ayurveda classics, as in young flower or fruit in the earlier stage, fragrance does not manifest, *artava* does not appear in young age signifying the importance of *Kala* in *Artava Utpatti* (41). It means that the *Kala* has its own importance in the production of *artava* but the maturity of the genital organs or body as a whole is also essential for the same (42). Kashyapa Samhita says that after replenishment of *dhatu* and steadiness of body, the blood accumulated in the *garbhasaya* again leaves the *yoni* in appropriate time which signifies a neuroendocrinological homeostasis through *Dhatu Paripoornata*.

Pathophysiology of Pain:

Inflammatory mediators such as bradykinin, Prostaglandin E and leukotrienes contribute to the process of sensitization of primary nociceptors which are the primary pain receptors. The effect of the psychological factors on the severity of pain implies the existence of brain circuits that can modulate the activity of the pain transmission pathway and pain modulation (43). The intensity of pain has marked individual variations and is subjected to many behavioural, physiological and psychological influences. Receptors are directly stimulated by mechanical stress, severe vasodilatation, ischaemia, obstruction, inflammation etc or indirectly by PGs, bradykinin etc due to tissue damage. By the activation of the receptors, stimuli go to spinal segment and then passes to ascending tract and reach the pain centre of brain, then pain is identified, the same process of pain production takes place in dysmenorrhoea.

a) **Hormonal Imbalance:** Spasmodic dysmenorrhoea is found to be associated with hormonal fluctuation in the uterine axis. Progesterone stimulates myometrial contraction of the smooth muscle of the cervix and thereby constricts the cervical canal. Progesterone also stimulates the synthesis of prostaglandin F2α which in turn causes pain. Hormonal imbalance which takes place when excessive action of the corpus luteum causes the formation of a thick endometrium which is expelled in large fragments or as cast of the uterus; the expulsion of these large fragments causes excessive colicky contractions of the uterus (44).

b) **Myometrial Activity Theory:** It is not adequately explained that myometrial contractions produce pain, as all myometrial contraction are not found painful. However, the researcher Reynold has pointed out that when painful contraction is produced, the pain can be relieved by such a drug which reduces the strength of the contractions of myometrium. The dysperistalsis and hyperactivity of the uterine junctional zone are seen to be the important mechanism for Primary Dysmenorrhea (45).

c) **Myometrial Ischemic Theory:** During uterine contractions, endometrial blood flow decreases, which indicates that ischemia due to the hypercontractility is the primary cause for pain associated with Primary Dysmenorrhea (46).

d) **Psychogenic Theory:** All the psychogenic stress related factors will reduce the pain threshold. Most researchers consider pain to be of psychosomatic origin due to various factors like dependence, anxiety particularly in adolescent age group. Anticipation of severe dysmenorrhea each month can itself be expected to engender quite a bit of stress (47).

e) **Prostaglandins:** PGs are synthesised from free and unsaturated fatty acids, such as arachidonic acid etc. which are derived from conversion of phospholipids, triglycerides, cholesterol esters by enzyme acyl hydrolase, under the influence of cyclooxygenase COX, isomerase, reductase (Prostaglandin synthetase). Although PD is characterized by increased production of PG, it is not known whether increased PG production is mediated by COX-1 or COX-2. PG production in an ovulatory cycle specially (PG F2α) or of their activity in the uterus is the main cause of PD (48). It has been demonstrated that secretory endometrium contains more PGs than that of proliferative one. Progesterone exerts an important control on the secretion of PGs and the stability of *lysosomes*. At the end of the luteal phase, a reduced level of progesterone causes lysosomal instability which is accompanied by menstrual flow with generation of arachidonic acid. This in turn causes the production of PGs together with intracellular destruction and trauma accompanying the onset of menstrual bleeding. Pickles and colleagues were the first researchers to quantify the PGs in menstrual fluid and found out that dysmenorrhoeic women produce 8-13 times more PG. F than those of non dysmenorrhoeic women. PGs are known to increase myometrial contractions and constriction of endometrial blood vessels to produce ischaemia and breakdown of the endometrium, bleeding and pain. PG over production causes abnormal, dysregulated uterine contractions with an increased intrauterine pressure, vasoconstriction of small uterine vessels

leading to decreased uterine blood flow, increased sensitivity of pain receptors and ischemia of the uterine muscle, which ultimately produces pelvic pain. Increased PG. E in women results in higher uterine tone with high amplitude contractions causing dysmenorrhea. This has led to the popularity for the use of NSAID‟s, which act as COX inhibitors. The intensity of the menstrual cramps and associated symptoms of dysmenorrhea are directly proportional to the amount of PGF2s released (49).

f) **Leukotrienes:** Increased uterine leukotrienes are seen to be the cause for some cases of primary dysmenorrhea that which is not responding to NSAID‟s, because leukotrienes are formed by 5-lipooxygenase enzyme pathway, which will induce myometrial contractions. Increased leukotriene release by the endometrial and myometrial layers are observed by researchers throughout the menstrual phase in conditions of dysmenorrhea and menorrhagia especially leukotriene C4 and leukotrienes D4 than in women without dysmenorrhea (50).

g) **Muscular Inco-ordination:** Excessive amplitude and frequency of uterine contractions and a high resting tone between contractions is postulated to one of the main reasons for PD. During contractions, endometrial blood flow is reduced and there exists a positive correlation between minimal blood flow and maximal colicky pain, favouring the concept that ischaemia due to hypercontractility causes PD. This phenomenon could be explained by an imbalance in the autonomic nervous control on the uterine musculature, in which an overactive sympathetic system leads to hypertonicity of the circular fibres of the isthmus and internal os. During menstruation in case of a non dysmenorrhoeic woman, the active pressure is maximum (120 mm Hg), uterine resting tone is lowest (< 10 mm Hg), number of contractions (3-4 per minute). The increased release of uterine PGs adds a significant effect on to the myometrial hyperactivity. These abnormalities lead to poor uterine reperfusion and oxygenation, thus giving rise to pain (51).

h) **Vasopressin:** Increase in vasopressin levels without any difference in the oxytocin levels increases the PG synthesis which will increase the myometrial activity. But a recent study did not show the vasopressin elevations in primary dysmenorrhea and found out that treatment with a vasopressin antagonist, atosiban had no effect on the condition (52).

i) **Endothelins:** Endothelins causes myometrial contractions especially in the endo-myometrial junction. Endothelins in endometrium can induce PGF2α. Local myometrial ischemia caused by endothelins aggravates uterine dysperistalsis.

j) **Platelet activating factor (PAF):** These are vasoconstrictors and stimulates myometrial contractions.

k) **Blood stagnation in the uterus:** The strong and abnormal uterine contractions in dysmenorrheic women reduce uterine blood flow and cause myometrial ischemia, resulting in pain. Administration of an uterolytic, such as a calcium channel blocker or NSAID, regulates the hypercontractility and restores blood flow to normal pattern. The pulsatility index and resistance index of both uterine arteries and the arcuate artery were found to be significantly higher on the first day of menstrual cycle in women with primary dysmenorrhea, suggesting increased blood flow impedance and uterine vasoconstriction as the cause of the pain (53).

l) **Abnormal Anatomical and Functional Aspects of Uterus:** Unequal development of mullerian ducts such as separate or bicornuate uterus, Hypoplastic uterus, Cervical obstruction, Deficient polarity of uterus were found to be attributing factors for dysmenorrhea in some women.

Almost all *Acarya* have described regarding the symptom *shoola* but all references are scattered in description of different *roga*. Among the various synonyms of *kasta* like *ruk*, *ruja* etc, *sula* is of prime importance here. *Sula-rujayam* which means a sharp pain in belly as colic.

Types of Pain in Dysmenorrhea

Pain is defined as an unpleasant sensory and emotional experience associated with a potential tissue damage. According to some studies, around 60% of patient complaints of cramps and the rest have pelvic ache and discomforts with respect to dysmenorrhea. The major types of pain observed in dysmenorrhea are

- Cramping abdominal pain that increases to a maximum in its intensity and then suddenly ceases for a period of complete absence of pain, where the episodes repeat at intervals.
- Colicky pain which is sharp and almost similar to that experienced in abortion.
- Dull ache which is steady with a feeling of bearing down pressure, referring to suprapubic area and down the legs.

Shoola* in *Udavartini yonivyapad

Udavartini, one among the 20 *yonivyapat*, is due to *urdhvagamana* of *vata*, and is characterized by pain which is relieved when the flow of menstruation is properly established (54). According to Caraka Samhita, due to *vegavarodha* (natural urges), the *Apana vata* is aggravated and moves in reverse direction. This *Apana vata* pushes the *Raja* upward, causing pain and discomfort during menstruation. The lady gets relieved from the pain after the proper flow of menstrual blood is established.

Ayurvedic concept of pain related to *Udavartini*

According to principles of Ayurveda, without the involvement of *vata, shoola* or pain cannot exist. According to Caraka Samhita, no *yoniroga* can occur without the affliction of aggravated *vata dosa*. Role of

vata is inevitable as either *pradhana doṣa* or *anubandhi doṣa*. Considering *Udavartini yonivyapat*, it is classified under *vataja yonivyapad* and other *doṣa* are its *anubandha doṣa*. *Vata Prakopa* being the main cause of *Udavartini yonivyapat*, causes can be categorized in to *aharaja, viharaja and manasika*. *Acarya Susruta* has already mentioned that the one who does *vegadharana* for long duration, leads to *vata prakopa* and suffers from *udavarta roga*. All these *nidana* contribute to *srotodushti* which may occur either individually or in combined form giving rise to the disease. *Atreya Sampradaya* says the *vedana viseṣa* is predominantly due to vitiation of *doṣa* in *Srotodushti*. In *udavarta roga*, *vimarga gamana* of *apana vata* occurs due to *adhovegadharana*, resulting in the manifestation of disease and pain leading to *krcchra artava* or kashtartava and ruja.

Consequences of *vega dharana* mentioned in our classics can also be counted here during the evaluation of *Udavartini yonivyapat*.

Clinical features of *Udavartini*: Apart from the frothy and difficult menstruation along with pain, the classical clinical feature of *Udavartini yonivyapat* explained by *Acarya Caraka* is the immediate relief obtained by the patient just after the proper flow of menstruation is established.

***Samprapti* of *Kastartava*:** In the disease *kaṣṭartava* all the three *dosa* are involved with predominance of *Vata*. The probable mode of pathogenesis may be viewed as follows. *Vata doṣa* which is the primary factor in the pathogenesis of the disease may be vitiated or aggravated by three ways.

- Due to indulgence of *Vata prakopaka Ahara* or *Vihara.*
- Due to *dhatukṣaya*
- Due to *marga avarana*

Due to the intake of *Vata prakopaka Ahara* and *Vihara*, *vata doṣa* aggravates leading to *anuloma dhatu kṣaya* from *Rasa dhatu*. There will be a derangement in the *Upadhatu* formation. Hence *Artava* will be produced in less quantity than normal which will further vitiate vata doṣa which further will produce *garbhasaya kṣhobha*. This will lead to toda and vedana. The vitiated *vata* by *rukṣa, sukṣma* properties spread through *rasavaha srotas* and leads to *rasavaha, raktavaha* and *artavavaha Srotodusṭi*. *Doṣa duṣya sammurcchana* takes place in *garbhasaya*. Here due to vitiation of *vyana* and *apana vayu*, the *akunjana* and *prasaraṇa kriya* of *garbhasaya* does not take place properly, the state exactly that of dysrhythmia of uterine muscles, which will hinder the proper flow of menstrual blood leading to *kaṣṭartava.* The explanation of additional ten *pesi* in the *garbhasaya* by our classics can be considered here as the anatomical entities of *mamsa dhatu* which indicates the direct role of *pesi akuñjana* and *prasaraṇa* in *kastartava* by *vyana vayu prakopa*. The *sara, drava, uṣṇa, tikṣṇa* properties of vitiated *Pitta* plays an important role in the *Sthanika Rakta vrddhi* with the help of *Vyana* and *Apana Vayu*. As the functioning of *Vyana* and *Apana Vayu* gets impaired, it will result in *kaṣṭartava*. The vitiated *kapha* due to its *snigdha, guru, picchila* and *abhiṣyandi guna* will impair *agni* and will cause *Jatharagni* and *dhatvaagnimandyata*. That will create *artava pravritti rodha* or painful menstruation. The concept of *avarana* also seems to play role producing pain, especially when the condition of *kapha avrta vata* is concerned. As in *kapha vrita Vyana vata*, pain as an additional symptom has been stated by *Acarya Vangasena* and *Hamsraja Nidana*, Manasika factors like *bhaya, shoka, cinta, krodha* also aggravates the *vata dosa* resulting the pathology of *kaṣṭartava.*

Samprapti Ghataka in Kastartava

- Dosa: *Vata Pradhana tridosa*
- Dusya: *Rasa, Rakta, Artava, Mamsa*
- Agni: *Jatharagni, Dhatwaagni Mandya*
- Srotas: *Rasa, Rakta and Artava vaha*
- Srotoduṣṭi: *Saṅga* and *Vimargagamana*
- Udbhava sthana: *Amapakvashaya*
- Sthana samsraya: *Garbhasaya*
- Vyakta Sthana: *Tryavarta yoni*

Conclusion

Quality of life of adolescent population can be improved if the principles of Ayurveda are followed properly. The etiopathogenesis of *Kastartava* with special reference to Primary Dysmenorrhea is mostly depending on the *Ahara* and *Viharaja nidana.* The physiological anatomical understanding of primary dysmenorrhea should be incorporated in the prophylactic and therapeutic ayurvedic clinical approach towards any case who are susceptible for *Kastartava*. Moreover, there is a need to develop more diagnostic and assessment strategies for primary dysmenorrhea so as to find and test the scope of Ayurvedic management.

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