Review of Literature
ANATOMY OF FEMALE REPRODUCTIVE ORGANS

AYURVEDIC CONCEPTS

The organ concerned with reproduction of a new generation of human being is considered to be that of ‘Yoni’ in Ayurvedic classics. The word meaning itself denotes its function as that of helping in provision of new generation in the society. It is mentioned in Ayurvedic classics to denote the organs involved in the function of reproduction. However according to contexts it varies, as while describing the gynaecological disorders in general, the word ‘Yoni’ is used to denote all the organs under one heading and while describing the specific causes as different organs related to reproductive system.

Yoni is considered¹ to be one among the three extra external orifices present in female, through which the arthava raktha flows out.

Shape

The shape of yoni² is considered to be that of sankha nabhi or couch shell having three avarthas or circular layers inside-Garbhasaya is considered to be the organ situated in the third avartha of yoni.

There are differences of opinion among Ayurvedic scholars while describing the three avarthas separately. Some considered it as the different layers of tissue present in the vaginal canal as that of

1. Outer fibrous layer or fasciae.
2. Muscular layer.
3. Mucus layer.

However while taking into consideration the site of garbhasayam or uterus and that of the description about the avarthas along with that of bahya srotas one can consider that these three avarthas may be the different parts of reproductive system as that of

1. The introitus
2. The external os of cervix separating the vaginal folding from that of the uterus.
3. The internal os of cervix which open into the asaya of garbha or the uterine cavity.

The tissues included in between these demarcating circular parts can include that of the parts mentioned earlier as that of faciae, mucous membrane etc. These include the tissues that are mentioned under the name of internal genital organs in modern science.

Nadees

Acharya Bhavamisra\(^3\) gave a well explained description about the nadees of yoni. According to him the manobhavagara mukha of female have three nadis namely samirana, chandra mashi and gowri collectively known as nadithrayam.

*Sameerana* is the nadi present in the outer most portion of the yoni supplying the madanadapa pathra and it is said that the *sukra* if fall over this portion will become futile. The second chandramasi in present is kandarpageha and if *sukra* while by coitus fall on this area the women will deliver a beautiful female child. The third gowri nadi is said to be situated in the inner most portion *ie* the upastha garbha and if the *sukra* fall on this
area the women will deliver a male child and also she will be sexually satisfied.

The description about these nadis along with the effectiveness of coitus as with that of achievement of conception with different sex of child gives clue that this may be the description described about the different parts of the vaginal cavity.

Yoni as marma

The entire female reproductive system of female is included as marma by Susrutha⁴. According to his opinion injury to these parts causes severe pain and these are to be protected while doing any surgery on pelvic organs as vasthi, gudam etc.

Garbhasaya

It is considered⁵ to be the eighth asaya which is present only in female. As mentioned earlier it is the main organ concerned with reproduction and it is situated in the third avartha of yoni. The cavity inside the garbhasaya is considered to be the seat or the sight giving space as well as protection to the growing garbham (embryo or foetus) and so it is considered to be the garbha saya. This part contain dhamanis, siras, pesis etc. helping in the circulation of raktha, rasadhathu etc. which lead to formation of arthavam, rajas, garbham, ie. its nourishment and growth. This is the organ mainly concerned with transformation of raktha dhathu into arthava raktha and abnormalities in its function can lead to several abnormalities, and diseases as udavartha, vatiki etc.
Shape

Shape of garbhasaya⁶ is considered to be the same as that of mouth of rohitha fish ie angular or conical and short at the outer and becoming more wider and broad inside. The garbhasaya is generally described to be having the parts as garbhasaya gala and garbhasaya it self. In the garbhasaya gala the yonidwaram or rajomar gam is situated. The cavity or lacuna of it is very short (cervical canal). But that of uterine cavity is very broad.

Site or Relation

It is situated between vasthi and pakwasaya ie below vasthi (urinary bladder) and above pakwasaya (rectum). According to Kasyapa⁷ it is situated in the vipula kundala of Srotas ie inside the abdominal cavity or pelvic cavity in between the coiled intestines and is covered with jarayu (peritoneum).
Srotas

Garbhasayam is considered to be the root of origin of two arthava vaha srotas (uterine arteries) which are concerned with formation of arthavam and circulation of arthava raktam including that of menstrual blood.

Dhamani

Susruta described presence of two dhamanis (adhogami dhamanis) in female and divided them functionally into two. One is concerned with formation of arthavam and another with excretion of arthavam or rajas.

Siras

Presence of multiple number of small hair like thin siras are mentioned in classics which are concerned with formation of rajas or arthavam.

Pesi

Presence of extra ten pesis are being mentioned in classics among females at apathy patha. Among these three are situated outside that of garbhasayam three in garbhasayam and two at garbachidram which is responsible for entry of sukra and arthavam. There are difference of opinion among ayurvedic scholars in considering the classification of pesis as some include fasciae, muscle, tendon etc. These may be the muscle situated at introitus, vagina, uterus and fallopian tubes.
Pesis present in garbhasayam is generally under control of vata *ie* apanavayu and any derangement in the function of vayu can effect function of pesis. Arthava nirgamana is one of the many function of apanavayu and malfunction of these will lead to arthava dushti as kricharthavam, udavartha etc.

References

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ANATOMY OF
FEMALE REPRODUCTION ORGANS

The reproductive organs in female are those which are concerned with copulation, fertilization, growth and development of foetus and its subsequent exit to the outer world. The organs are divided into

I. External genitalia
II. Internal genitalia
III. Accessory reproductive organs

I. External genitalia

External genitalia of female are vulva. The vulva includes mons veneris, labia majora, labia minora clitoris, vestibule, and perineum. Mons veneris is a pad of subcutaneous adipose tissue lying in front of the pubis and in adult it is covered by hairs.

Labia Majora

The vulva is bounded by elevation of skin and subcutaneous tissue which from the labia majora. They are continuous where they join medially to form posterior commissure in front of the anus. They are covered with squamous epithelium and contain sebaceous glands, sweat glands and hair follicle. Labia minora are two folds of skin, devoid of feet. Anteriorly they divide to enclose the clitoris and unite with each other in front and behind the clitoris to form the prepuce and frenulum respectively. The lower portion of the labia minora fuses across the midline to form a fold of skins known as fourchette. The labia minora do not contain hair follicles. The
folds contain connective tissue, numerous sebaceous glands erectile muscle fibers and numerous vessels and nerve endings. Clitoris is a small cylindrical erectile body measuring about 2.5 cm. It consists of glans, a body and two crura. The glans is richly supplied by nerves. Vestibule is a triangular space bounded anteriorly by the clitoris. There are four opening into vestibule.

(a) Urethral  
(b) Vaginal orifice  
(c) Opening of bartholin’s duct  
(d) Two sken’s duct  

Vestibular bulbs are bilateral elongated masses of erectile tissue situated beneath the mucous membrane of the vestibule.

**Blood supply of vulva**  
**Arteries:**  
(a) Branches of internal pudendal artery  
(b) Branches of femoral artery  

**Veins drain into**  
(a) Internal pudendal vein  
(b) Vesical or vaginal venous plexus  
(c) Long saphenous vein  

Nerve supply: Vulva is supplied through bilateral spinal somatic nerves. Anterior superior part is supplied by the cutaneous branches from ilio-inguinal and genital branch of genito-femoral nerve and posterior inferior part by pudendal branches from the posterior cutaneous nerve of thigh. Vulva is supplied by the labial and perineal branches of the pudendal nerve.
Lymphatics

Lymphatics drain into

(a) Superficial inguinal nodes
(b) Intermediate groups of inguinal lymph nodes, gland of cloquet.
(c) External and internal iliac lymph nodes.

II. Internal genital organ

These include vagina, uterus, fallopian tubes and the ovaries.

The vagina

Vagina is the female organ of copulation, fibro muscular tube lined with stratified squamous epithelium, which extends from the vestibule, or cleft between the labia minora to the uterus, and is situated between the bladder anteriorly, rectum and anal canal posteriorly. It is inclined posterior superiorly, its axis forming with that of the uterus at an angle of over 90°; opening anteriorly but which varies with the condition of the bladder and rectum. Its length is 7.5 cm along its anterior wall and 9 cm along its posterior wall. Its width gradually increases from below upwards. It upper end surrounds the vaginal portion of the cervix uteri. Its attachment extending higher on the posterior than on the anterior wall of the cervix. To the arched recess between the cervix and vagina the term fornix is applied.

Structure: The vagina consists of a mucous membrane and a muscular stratum, the lamina propria of the former containing in its deepest layer a
large number of thin walled veins. The mucous membrane is firmly fixed to the muscular layer.

The epithelium of the mucous membrane is of the nonkeratinized stratified squamous variety. After puberty it becomes thick and rich in glycogen. Its glycogen increases in the post-ovulatatory phase and diminishes towards the end of the cycle. The fermentative action of certain bacteria (Doederlein’s bacillus) on the glycogen renders the fluid in the vagina acid. There are no glands in vaginal mucous membrane, which is thus lubricated by mucus derived from the glands of the cervix uteri.

The muscular layers are inner circular and outer longitudinal. External to the muscular coat there is a layer of areolar tissue, and is highly vascular.
Vessels and nerves

The arteries of vagina are derived from the vaginal, uterine, internal pudendal, and middle rectal branches of internal iliac arteries. The veins are drained through the vaginal veins into the internal iliac veins.

Nerve supply: The vagina is supplied by sympathetic and para sympathetic nerves from the pelvic plexus. The lower part is supplied by pudental nerve.

The uterus

The uterus is a hollow pyriform muscular organ situated in the pelvis between the bladder in front and the rectum behind.

Position

Its normal position is one of the ante version and anteflexion. The uterus usually inclines to the right so that the cervix is directed to the left and comes in close relation with the left ureter.

The uterus measures about 8 cm long 5 cm wide at the fundus and its walls are about 1.25 cm thick. Its weight varies from 50-80 gms. It has got the following parts.

1. Body
2. Isthmus
3. Cervix

   1. Body: The body is divided into three parts.

   (a) Fundus: This is the part which lies above the openings of the uterine tubes.
(b) The body proper: The body proper is triangular and lies between the openings of the tubes.

(c) The isthmus: The superiolateral angles of the body of the uterus project outwards from the junction of the fundus and body and are called the cornua of the uterus. The uterine tube, round ligament and ligament of ovary are attached to each cornu.

2. Isthmus

The isthmus is a constricted part measuring about 5 cm, situated between the body and the cervix. It is limited above by the anatomical internal os and below by the histological internal os.

3. Cervix

The cervix is the lowermost part of the uterus. It extends from the histological internal os and ends at external os which opens into the vagina after perforating anterior vaginal wall. It is almost cylindrical in shape and measured about 2.5 cm in length and diameter.
Cavity

The cavity of the uterine body is triangular in coronal section with base above and apex below. It measures about 3.5 cm. The walls consists of three layers from outside inwards.

Perimetrium: It is the serous coat which invest the entire organ except on the lateral borders.

Myometrium: It consists of thick bundles of smooth muscle fibres held by connective tissue and are arranged in various directions. There distinct layers of muscles are seen in myometrium. They are

1. Outer longitudinal
2. Middle interlacing
3. Inner circular

Endometrium: The mucous lining of the cavity is called endometrium. It consists of lamina propria and surface epithelium. The surface epithelium is a single layer of ciliated columnar epithelium. The lamina propria contains stromal cells endometrial glands, vessels and nerves. The glands are simple tubular and lined by mucus secreting non ciliated columnar epithelium. The components are all changed during menstrual cycle. The endometrium is changed to deciduas during pregnancy.
Cervix

The cervix is composed mainly of fibrous connective tissue. Only the posterior surface has got peritoneal covering. The glands, which dip into the stroma, are of complex type and are lined by secretory columnar epithelium. There is no stroma unlike the corpus and the lining epithelium rest on a thin basement membrane.

Secretions

The endometrial secretion is scanty and watery. The physical and chemical properties of the cervical secretion change with menstrual cycles and with pregnancy. The cervical glands secrete an alkaline mucus with pH 7.8. The mucus is rich in fructose, glyco protein and mucopoly succharides. It also contain sodium chloride. The fructose has got nutritive function to the spermatozoa. Under oestrogenic stimulation glyco protein network is arranged in parallel to each other thus facilitating sperm ascent. Cervical mucus contributes significantly to the normal vaginal discharge. A part forms the mucus plug which functionally closes the cervical canal and has got bacteriolytic property.

Blood supply

The arterial supply is from the uterine artery. Veins drain into internal iliac veins.
Nerve Supply

The nerve supply of the uterus is derived from sympathetic system and partly from para sympathetic system. Sympathetic components are from T₅ and T₆ and T₁₀ to L₁ spinal segments. The somatic distribution of uterine pain is that area of the abdomen supplied by T₁₀ to L₈. The para sympathetic system is represented on either side by pelvic nerve which consists of both motor and sensory fibres from S₂, S₃, S₄ and ends in ganglia of Frankenhauser.

Fallopian tubes

The uterine tubes are paired structures measuring about 10 cm and are situated in the medial ¾ of the upper free margin of the broad ligaments. Each tube has hot two openings; one communicating with the lateral angle of the uterine cavity called uterine opening of 1 mm in diameter and the other end called pelvic opening of 2 mm in diameter.

Structure

It consists of three layers.

1. Serous - Consist of peritoneum on all sides except along. The line of attachment of meso salpinx.

3. Mucus membrane: lined by columnar epithelium and is partly ciliated. Changes occur in the tubal epithelium during menstrual cycle but are less pronounced and there is no shedding.

The important functions are transport of gametes, to facilitate fertilization and survival of zygote through its secretion.

Blood supply

Arterial supply is from the uterine and ovarian arteries. Venous drainage is through the pampiniform plexus into the ovarian veins.

Nerve Supply: Is derived by branches from the uterine and ovarian nerves.

Ovary: The ovaries are paired sex glands or gonads in female which are concerned with germ cell formation, maturation, storage and its release and steroidogenesis.

Each gland is oval in shape and pinkish grey in colour. It measures about 3 cm in length 2 cm breadth and 1 cm thickness. Each ovary presents two ends tubal and uterine.

The ovaries are intraperitoneal structures. In nulliparae the ovary lies in the ovarian fossa.
Structure

The ovary is covered by a single layer of cubical cell known as germinal epithelium. The substance of the gland consist of outer cortex and inner medulla.

Cortex

It consists of stromal cells which are thickened beneath the germinal epithelium to form tunica albuginea. During reproductive period the cortex is studded with numerous follicular structures called functional units of the ovary in various phases of development. The structures include primordial follicles, maturing follicles, graffian follicle and corpus luteum. Atresia of structures results in formation of atretic follicles or corpus albicans.

Medulla

It consists of loose connective tissue few unstriped muscles, blood vessels and nerves. There are small collection of cells called hilus.

Blood supply

Arterial supply is from the ovarian artery; a branch of abdominal aorta. Venous drainage is through papiniform plexus to form the ovarian veins which drain into inferior vena cava on the right side and left renal vein on the left side.
Nerve supply

Sympathetic supply comes down along the ovarian artery from T_{10} segment.

III. Accessory reproductive organs

The Breast

The breasts are bilateral glandular structures and in female constitute accessory reproductive organs as the glands are concerned with lactation following child birth.

It usually extends from second to sixth rib in the mid clavicular line. It lies in the subcutaneous tissue over the fascia covering the pectoralis major or even beyond that to lie over serratus anterior and external oblique.

The areola is placed about the centre of the breasts and is pigmented about 2.5 cm in diameter. There are numerous sebaceous glands over it and contains few involuntary muscles. The nipple is muscular projection covered by pigmented skin. It accommodate about 15-20 lactiferous ducts and their openings.

Blood supply

Arterial supply

(1) Lateral thoracic – branches of the axillary artery
(2) Internal mammary artery
(3) Intercoastal artery

Veins: The veins follow the courses of the arteries.

Lymphatic drainage of breast tissues are different at different areas as...
1. Lateral hemisphere - anterior axillary nodes
2. Upper convexity - infra clavicular group
3. Medial convexity - mediastinal glands
4. Inferior convexity - mediastinal glands

**Nerve Supply**

The organs are supplied by IV, V and VI inter coastal nerves.

**Neuro anatomy and neuro physiology of the female genitalia**

a. Abdominal wall

The anterolateral muscles of the lower abdominal wall *ie.*, the internal and external. Oblique muscles rectus abdominis, transverse and pyramidalis receive their motor nerve supply from the anterior primary rami of the lower three thoracic and first lumbar nerves. These travel anteriorly from the intercostal spaces (T_{10} and T_{11}) or subcostally (T_{12} and L_{1}) to lie between the transverse abdominis and the internal oblique until reaching the lateral border of rectus abdominis. They pass posteriorly to the latter muscle and end within it. They give off lateral cutaneous branches which pass through the anterior. Sheath of the rectus abdominis and supply sensory fibres to the anterior abdominal wall.

A few branches derived from T_{12} communicate with the anterior primary rami of L_{1} & L_{2} from which the ilio hypogastric, ilio inguinal and genito femoral nerves are derived. The anterior cutaneous branch of the liohypogastric becomes relatively superficial at a point about 2 cm medial to
the anterior superior iliac spine, and it then runs forwards to provide the sensory nerve supply of the suprapubic area. The ilioinguinal having passed through the transverses abdominis near the anterior part of the iliac crest, pierces the internal. Oblique and supplies sensory fibres to the mons pubis and the labia majora. The genital branch of the genito femoral nerve arises from its parent branch retroperitoneally lateral to the ureter and passing behind the external iliac artery enters the inguinal canal. It accompanies the round ligament of the uterus and provides sensory nerve fibres to the mons pubis and labia majora.

b) Pelvis

The muscles of the posterior wall of the pelvis mainly quadratus – lumborum, psoas and coccygeus receive motor nerve supply from root values T₁₂, L₁-L₄, but of greater moment their sensory nerve supply is same.

c) Pelvic floor

The muscles of the pelvic floor obturator internus, periformis, levator ani and coccygeus – derive their motor nerve supply from the dorsal branches of the sacral plexus (L₁ to S₄).

d) Perineum

The perineal muscles may be considered in two groups anal and urogenital. The sphinter ani externus which is in an unopposed state of tonic contraction is supplied by motor fibres arising directly from the fourth sacral nerve and also by fibres derived from the inferior, haemorrhoidal
nerve, the latter is a branch of the pudental nerve, from which it springs during the course of the latter’s traverse of the pudental cannal. The inferior haemorrhoidal nerve also supplies sensory fibres to the lower part of the anal canal and to the skin around the anus.

e) Vulva and vagina

The muscles related to the vagina, clitoris urethra are supplied by the perineal branch of the pudental nerve (S₂, S₃ and S₄). This is the larger terminal branch of the pudental nerve which continues to traverse the pudental canal and approaching the pubic arch-passes behind the pudental artery before distributing its motor fibres as described, and its sensory fibres to the labia and clitoris. The sensory supply of the labia and the adjacent skin of the perineum is provided not only by the pudental nerve but also by the perineal branch of the posterior, cutaneous nerve of the thigh. The latter arises from the dorsal branches of S₁ and S₂ and the ventral branches of S₂ and S₃, and gives off its perineal branch as it courses below the ischial tuberosity, at which point the perineal nerve passes the fascia lata and running beneath the superficial fascia of the perenium reaches the labia. The sensory supply of the vagina and urethra is provided by the perineal branch of the pudental nerve as is the motor supply to the urethra and its sphincter.

The uterus is supplied by motor nerves of both sympathetic and parasympathetic origin. The root value of the sympathetic contribution extends from T₅ to L₂. The preganglionic fibres of the thoracic roots travel down the posterior wall of the thorax anterior to the heads of the ribs and just beneath the coastal pleura. The fibres from T₅-T₉ coalesce to form the
greater splanchnic nerve, those from T_{10} and T_{11} to form the lesser splanchnic nerve and those from T_{12} are identified as the lowest splanchnic nerve. Each of these structures pierces the diaphragm in the vicinity of the crus to synapse in the celiac ganglion, aorlico renal ganglion or in the case of the lowest splanchnic, the inferior mesenteric of the hypogastric ganglia to which the pre ganglionic fibres from L_{1} and L_{2} are also directed.

The first ganglionic fibres travel caudally in close proximity to the aorta and its major branches. Those destined to supply the pelvic structures unite to form the two hypogastric or pre sacral nerves, which eventually intermingle to form the hypogastric plexus, situated anterior to left common iliac vein, the fifth lumbar vertebra and the sacral promotary, between the two common iliac arteries. From the hypogastric plexus are derived the right and left pelvic plexus. The latter are joined by preganglionic parasympathetic fibres of root value S_{2} and S_{3} and possibly S_{4}. Fibres destined to supply the uterus accompany the internal iliac arteries and subsequently the uterine arteries. The fundus of uterus and fallopian tube receive their post ganglionic sympathetic motor supply from nerves which emerge from renal plexuses.

The sympathetic motor supply to the myometrium appears to be of little functional importance in respect to either pregnancy or menstruation. The preganglionic parasympathetic fibres synapse in minute ganglia situated within, or very closely adjacent to the myometrium. Their post ganglionic fibres subserve a predominantly vasomotor function.

Ovaries and Fallopian tubes
The sensory supply of the ovaries, fallopian tubes and the entire uterus is subserved by sympathetic nerve of root value T_{11} and T_{12}. Fibres from lower part of uterus, including the cervix travel in the base of the broad ligament in company with uterine artery via the pelvic plexus, eventually to reach the spinal cord through the appropriate dorsal nerve root. Fibres, which supply sensation to the fundus of the uterus, the tubes and ovaries, reach the same destination via the ovarian plexus and renal plexus.
ARTHAVAM

Arthavam is one among the main reproductive function of women. It is considered to be the main identifying feature of a woman attaining capacity to reproduce and is considered to be the turning point from childhood to adulthood. The presence of arthavam is denoted by

1. Cyclical monthly bleeding per vagina and
2. By presence of conception.

Achievement of conception is considered to be the main objective of function of arthavam and so it include various processes including the formation of beeja, change in garbhasaya along with raja-raktha sanchayam. Absence of conception will lead to expulsion of those things prepared for the same with presence of bleeding *per vaginum*.

**Definition**

It arises from the root “rte bhavam arthavam” which denote its specific characteristic of occurrence in a cyclical manner. Generally normal arthavam is characterized by periodic cyclical loss of blood through yoni for 3-5 days which is bright\(^1\) red like ‘indragopa’ or with slight black\(^2\) tinge, without any particular smell, burning sensation or unbearable pain.

Synonyms
The word arthavam is mentioned in different contexts with different meanings including

a. beejam (ovam including ovulation)
b. rajas (menstrual blood including menstrual cycle)
c. dhathu (metabolites in blood including hormones)

Arthavam as beejam

The words like sonitha, raktha³ beeja are used to denote beejam.

Arthavam as rajas

Raktha⁴, rajas⁵ and pushpa⁶ are used to denote the meaning as rajas.

Arthavam as dhathu

Vagbhata, Kasyapa⁹ and Delhana accept presence of raja from very childhood and is said that it will not be visible due to inconspicuousness or presence in very minute quantity. Chakrapani⁷ has written that arthavam which is explicit at 12 years is formed during embryonic life. Susrutha⁸ while describing the formation of dhatus accepts formation of arthavam along with sukra in females. Susrutha accepts the development of breasts, uterus, vagina, and vulva etc. during adolescence due to gradual accumulation of arthavam. These descriptions give clue for the presence of hormones present in blood which get circulated through out body along with helping in the changes including that of attainment of adulthood, puberty, menstruation, ovulation an achievement of conception along with its maintenance. Bhavamisra¹⁰ mentioned that women posses an extra dhatu
just as presence of an extra asaya thus have arthava as seventh dhathu. All the functions relating to conception are performed by arthavam.

Uthpathy of rajas

Based on the theory of dhatu parinama the ahara we take will change in due course of time into seven dhatus. They are rasa rakta, mamsa, medas, asthi, majja, sukra which constitute the whole body parts.

The raktha formed from rasa circulates through out the body reaches the garbhasaya getting circulated through hair like thin arthava vaha srotas in one month of time assumes certain specific changes and it comes out as arthavam.

Rajas

Though the rasa dhathu formed out of ahara rasa is soumya in character but by the parinama or paripaka occurring inside the garbhasaya transforms it into agneya in character with addition of agni mahabhoota. So rajas or arthavam is considered to be ushna, theeksha and agneya.
There are differences of opinion regarding the formation and origin of arthavam among Ayurvedic scholars as mentioned in the table below.

Table 1
Opinion of different acharyas regarding the

<table>
<thead>
<tr>
<th>No.</th>
<th>Classics</th>
<th>Dhatu from which arthavam is formed</th>
<th>Character</th>
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<tbody>
<tr>
<td>1.</td>
<td>Susrutha(^{12})</td>
<td>Rasa</td>
<td>Soumyam</td>
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<tr>
<td>2.</td>
<td>Vagbhata(^{13})</td>
<td>Rasa</td>
<td>Soumyam</td>
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<td>3.</td>
<td>Dalhana</td>
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<td>4.</td>
<td>Chakrapani(^{14})</td>
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<td>5.</td>
<td>Vagbhata II(^{15})</td>
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<td>6.</td>
<td>Arunadatha</td>
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<td>7.</td>
<td>Sargadharma(^{16})</td>
<td>Raktha</td>
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<tr>
<td>8.</td>
<td>Bhavamisra(^{17})</td>
<td>Raktha</td>
<td>Agneya</td>
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Arthava Vibhagam

While going through the classics it is understood that there are two varieties of arthavam (or the things occurring cyclically) are being mentioned. As description about the amount, smell etc. are mentioned as that of a kind of arthavam at one place with specific duration, it can be made out that it is conspicuous or it can be felt by the sense organs whereas as of the other variety, only its timing is mentioned without other characteristic and
cannot be made out by sense organs. It occurs on 12th day from 1st day of arthavam (menstruation) or raja srava darsanam. So we can assume that it is another variety which is not conspicuous, probably it may be occurring inside the body. Due to these characteristics some commentators have classified arthavam into

1. Anthararthavam
2. Bahya aarthavam

Anthararthavam

As per description in Astanga Sangraham18 it can occur on 12th day starting form first day of arthava raktha sravam, 16th day or any day within one month period. As this is the process in which formation of stree beejam happens this is considered to be the period at which conception can occur.

Presence of this arthava generally produces some change in the psychology of the women and the women in whom there is presence of arthavam with specific symptoms is denoted as ritumati.

Ritu mati lakshanam

Charaka19 says that after the preveous raja is gone and new one has settled and after taking bath the women possessing healthy yoni, garbhasaya, and sonitha is termed as ritu mati.

According to Susrutha20 the rtumati women looks bright and healthy, her mouth and teeth are moist, she is anxious to hear love stories and to have
sexual relations her flanks eyes and hair are lax, she is quivering over arms, breasts, pelvis umbilicus thighs and hips and is happy and excited.

The importance of rtukala is for achievement of conception. Charaka has emphasized on the healthy condition of reproductive system and ovalation, lest abnormalities of these hamper fertilization. Psychological changes appearing in a r tumati will also be the signs favourable for achievement of conception.

Bahya arthavam

It is the condition in which presence of arthavam can be made out by external appearance or expulsion of rajas or arthava raktham. Susrutha\textsuperscript{21} and Vagbhata II\textsuperscript{22} describes this as the process in which the blood collected for whole month by both dhamanis assuming black colour and specific odour is brought downward to vaginal orifice for excretion.

Characteristics of Arthava or Rajas

Arthavam is agneya\textsuperscript{23} in character.

Basic mahabhootas

It is a combination of bhoomi and agni

Srotas – 2 arthava vaha srotas

Susrutha\textsuperscript{24} mentioned about arthava vaha srotas as two, originating from garbhasaya and arthava vahi dhamanis. Vandhya-thwa, loss of midhuna sakthi, arthava dosa are its complications if it is injured.
Pramanam

Quantity of it is four anjali\textsuperscript{25}.

Arthava darsana kalam and nashta kalam

In women the arthava\textsuperscript{26} darsana kalam is 12 years and nashta kalam is generally 50 years.

**Prasastha arthavam**

Arthava having the qualities that are not influenced by any doshas, which can produce a good progeny is called prasastha arthava. The normal menstruation\textsuperscript{27} is that which has inter menstrual period of one month duration, of five days, not associated with pain or burning sensation exerted blood is not unctuous, very not scanty or excessive in amount, the colour resembles the red juice of lac, red lotus flower or rabbit’s blood.

Bhavamisra\textsuperscript{28} had explained that the difference in colour is due to prakriti of individuals and vitiated doshas are responsible for symptoms like pain and burning sensation.

Rtu chakram or arthava chakram

Critical study on the period of each menstrual cycle makes one to understand that the whole one month period can be divided into 3 stratas as or phases as

1. Raja srava kalam – 3 to 5 days\textsuperscript{29}
2. Rtu kalam – 12 or 16\textsuperscript{31} days
3. Rtu vyatitha kalam – 9-13\textsuperscript{30} days

1. Raja srava kalam

This is the period in which the blood collected inside the garbhasaya through arthava vaha dhamanis will be expelled out by the help of vata with certain change in colour as that of slight black. According to the description of Charaka\textsuperscript{32} it occurs generally for 5 days and with generally of bright red in colour, without much disturbances as pain or burning sensation. But according to Bhavamisra\textsuperscript{28} pain can occur as one among the symptoms due to vitiation of dosha mainly that of vata. Pain can also occur if there is arthava kshaya along with its absence of occurrence in every month.

Table 2

Table showing the opinion of acharyas regarding raja srava kalam

<table>
<thead>
<tr>
<th>Classics</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Charaka\textsuperscript{32}</td>
<td>5 days</td>
</tr>
<tr>
<td>Vagbhata\textsuperscript{33} I &amp; II\textsuperscript{34}</td>
<td>3 days</td>
</tr>
<tr>
<td>Hareethan\textsuperscript{35}</td>
<td>7 days</td>
</tr>
<tr>
<td>Bhavamisran\textsuperscript{36}</td>
<td>3 days 5 days upto 16 days*</td>
</tr>
<tr>
<td>Susrutha\textsuperscript{37}</td>
<td>7 days</td>
</tr>
</tbody>
</table>

*Bhavamisra classifies normal menstrual days into three. If with excessive amount of bleeding normal menstruation (rajasra-vakalam) can be of 3 days. Where as with moderate amount of bleeding for 5 days and if very scanty bleeding of 16 days. However bleeding for 3 to 7 days can generally be accepted as normal.
Certain specific regimen is generally prescribed in Ayurvedic system of medicine for a women who is in her raja srava kala. As this is the period in which loss of dhathu (ie raktha) occurs, excessive exercise during this period may increase the amount of blood lost along with causing weakness. So if she practice the specific regimen during the period, that may help is decreasing bleeding as well as general debility.

**Raja swala charya**

From the day of onset\(^3^8\) of arthavam the women should observe chastity and avoid sleeping in day time, application of mascara, shedding of tears, bathing, anointing, massaging, cutting of nails, fast racing laughing and excessive exercise. So she should sleep on the bed made of darbha spread over ground and eat havisya taken in a utensil made of clay or leaves. She should not dorn herself with ornaments. Use of nasya, swedana\(^3^9\) and vamana are contraindicated. She should take less quantity of food and avoid tikshna, katu, lavana things.

2. Ritu kalam

It can be\(^4^0\) on 12\(^{th}\) day, may be on 16\(^{th}\) day starting from the 1\(^{st}\) day of arthavam. If the reproductive system is healthy it can occur on any day of entire month. Sometimes ritukala may come up without meustruation.

**Table 3**

Kasyapa and Bhavamisra mentioned certain

<table>
<thead>
<tr>
<th>Religion</th>
<th>41 Kasyapa</th>
<th>42 bhavamisra</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brahmana</td>
<td>12 days</td>
<td>-</td>
</tr>
</tbody>
</table>
Bhavamisra\textsuperscript{43} says that this period is the proper time for conception.

It is the period in which the arthavam or streebeejam will be fully formed \textit{ie} the period starting just after raja srava kala till the 12\textsuperscript{th} or 16\textsuperscript{th} day can be considered as this rtukala or aanthara-rthavakala. Specific changes\textsuperscript{44} occures inside the garbhasaya during this period particularly for accepting the sukra for achievement of conception. Yoni will be more opened during this period helping in ascend of sukra into the grabhasayam.

Any medicine given during that period will be easily absorbed into the garbhasaya and uterine cavity.

Ritu Vyathita kala

This is the period after rtukala in which further more changes occur in garbhasaya helping in growth and development of garbha, if once it has happened. The changes include that of constriction or closure of yoni marga just like that of lotus\textsuperscript{25} flower closing after sunset. It will not render or help in ascend of sukra any more. This period is followed by raja sravam if formation of garbha fails.

Predominant doshas during each stage of arthava chackram
By accepting the specific changes occurring in garbhasaya and other body parts during the whole arthava chakram the specific changes occurring inside the body at different phases with different doshas can be madeout.

(a) Rajasrava Kalam

As rajasrava kalam is the period in which there is expulsion of rajas or arthava raktham and the main function of apanavayu is also considered to be that of arthava rakthasrava, it can be presumed that vata is the main predominant dosha helping in the changes during rajasravakalam. Ayurvedic scholars as Vagbhata and Charaka also have accepted this function as that of vata. The change in arthava raktha to that of light black with particular smell is also considered to be that due to involvement of vata and so vata can be considered as the predominant dosha during rajasravakalam.

(b) Ritu Kalam

This is the period in which formation of arthavam happens. According to charaka rajasrava or exertion or expulsion of purana rajas will be followed by formation of naveena arthavam. Arthava is considered to be formed out of rasadhatu formed from the assimilated ahararasa which is of sowmya in character. Formation of any tissue mainly that of soumya and seetha character is considered to be that due to kapha dosha. So it can be understood that rtukalam is predominated by kapha dosham.
Rituvyathita Kalam

This is the period in which there is transformation of naveena rajas into purana rajas which involve pakam. Any type of paka or pachana karma inside the body is only under the function of pithadosham. Absence of formation of garbham will result in change of purana rajas into rajas or arthava raktha sravam, which involve specific changes including that of addition of agneyatha. By all this it can be concluded that the rituvyathita kalam is the period predominated by pitha which need mainly that of the function of pitha dosham.

Table 4

**Chart showing doshic involvement**

<table>
<thead>
<tr>
<th>Stage</th>
<th><strong>Duration</strong></th>
<th>Condition</th>
<th>Predominant dosham</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rajasrava kalam</td>
<td>3-5-7 days</td>
<td>Expulsion of purana rajas</td>
<td>Vata</td>
</tr>
<tr>
<td>Rtu kalam</td>
<td>12-16 days</td>
<td>Formation of naveena rajas</td>
<td>Kapha</td>
</tr>
<tr>
<td>Rtuvyathita kalam</td>
<td>9-13 days</td>
<td>Formation of purana rajas</td>
<td>Pitha.</td>
</tr>
</tbody>
</table>

All these descriptions about arthava chakram similate the things mentioned in modern science under the heading of menstrual cycle. Cyclical changes described in each stage of the whole menstrual cycle including that of pre ovulatory, post ovulatory an menstrual are the same as that of the descriptions given in Ayurvedic classics under the
heading of ritu kalam, rituvyathita kalam, and rajasravam kalam which highlights the 
wisdom of ancient rishis on the field of Ayurvedic science of life.

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3. Ch. Sar. 3/3, 8/6, 2/12
4. Su. Sar. 3/10
5. Ch. Sar. 4/7
6. Ch. Sar. 8/9
7. Ch. Sar. 4/30
8. Su. Soo. 14/15
10. B.P. Pookh 3/188
11. A.H. Soo 1/13
12. Su. Soo. 14/10
13. A.H. Sar 1/8
14. Ch. Sar 4/30
15. A. San Sar. 1/21
16. Sar. Sam Poo 5, 18
17. B.P. 3/204
18. Ch. Sar. 4/5
19. Ch. Sar. 3/6
20. Su. Sar. 3/6
21. Su. Sar. 3/7
22. A. San Sar. 1/40
23. Su. Soo. 14/9
<table>
<thead>
<tr>
<th></th>
<th>Name</th>
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<tbody>
<tr>
<td>24.</td>
<td>Su. Sar.</td>
<td>9/22</td>
</tr>
<tr>
<td>25.</td>
<td>A. San Sar.</td>
<td>5/69</td>
</tr>
<tr>
<td>26.</td>
<td>Su. Soo</td>
<td>14/7</td>
</tr>
<tr>
<td>27.</td>
<td>Ch. Chi</td>
<td>30/225</td>
</tr>
<tr>
<td>28.</td>
<td>B.P. Gar</td>
<td>3/206</td>
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<tr>
<td>29.</td>
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<td>30/225</td>
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<tr>
<td>30.</td>
<td>Su. sar</td>
<td>¾, 5</td>
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<tr>
<td>31.</td>
<td>B.P.</td>
<td>3/38</td>
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<tr>
<td>32.</td>
<td>Ch. chi</td>
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<tr>
<td>33.</td>
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<td>A.H. sar</td>
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<tr>
<td>35.</td>
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<tr>
<td>36.</td>
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<td>68/10</td>
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<td>37.</td>
<td>Su. Soo</td>
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<td>38.</td>
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<tr>
<td>40.</td>
<td>Su. Sar</td>
<td>¾, 5</td>
</tr>
<tr>
<td>41.</td>
<td>K. Sam. Sar. Ja.</td>
<td>5</td>
</tr>
<tr>
<td>42.</td>
<td>B.P. Poo. Kh. Gar</td>
<td>3/2</td>
</tr>
<tr>
<td>43.</td>
<td>B.P. Gar</td>
<td>3/2</td>
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<tr>
<td>44.</td>
<td>A.H. Soo</td>
<td>19/77</td>
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<tr>
<td>45.</td>
<td>B.P. Chi</td>
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</tbody>
</table>
MENSTRUATION

Presence of menstruation in a female indicates her well being as well as efficiency to attain mother hood. Achievement of conception or in other words becoming mother is the cherish desire of every women. Menstruation is the best sign of normally functioning genital organs of female the absence or untimely occurrence of which may cause several psychosomatic problems in the women.

Presence of menstrual bleeding is generally associated with other symptoms as pain, malaise, mental irritability etc. It is generally considered to be due to the change occurring in the neuro humoural systems which occur cyclically from puberty till climatric or menopause. The entire endocrine orchestra gets involved in the cyclical changes occurring in the genital organs. The changes include that of development of endometrial tissue along with discardation etc. Though development of endometrial tissue aimed at preparing proper bed for growth of fertilized ovum, the absence of latter leads to distroyal of the same which cause bleeding which can be evidenced by the presence of bleeding per vagina generally known by the name menstruation.

Definition

Cyclical discharge of blood, mucus and certain other substances from the uterus in the reproductive life of female, at an average interval of 28 days is called menstruation.

Menarche
The first menstruation occurs between 11-15 years with a mean of 13 years. It is closely related to bone age than to chronological age.

Age

The menstrual years can be divided into three phases.

I. 5-7 years following the menarche

II. 20-25 years of mature reproductive life.

III. 5-7 years that precede the menopause.

(Sherman & Wallace 1985)

The years immediately following the menarche are characterized by irregular menstrual cycle and long cycles (more than 36 days). This is due to immaturity of the hypothalamus and pituitary and menstrual cycles will be anovulatory.

The middle years of reproductive life in normal women are characterized by regular menstrual cycles and regular ovulation. In the perimenopausal years menstrual cycles often become irregular again due to the decreased number of ovarian follicles and their increased resistance to the gonadotrophin stimulation with longer cycles.

Characteristics of menstrual bleeding

(a) Composition

The menstrual blood is made up of

1. Dark altered blood 30-40 ml.
2. Stripped off endometrium
3. Mucus
4. Leucocytes
5. Vaginal epithelial cells
6. Prostaglandins
7. Enzymes
8. Bacteria etc.

(b) Cycle-interval

The variation in cycle length in different women not using any contraception is considerable being a mean of 29.6 days. There is a tendency for mean cycle length to decrease with age from 30.1 days at the age of 20 and 27.2 days at the age of 40. Variation in the length of the cycle in the individual patient also decreases with age, with a standard deviation of 3.9 days at age 20, a standard deviation of 2.8 days at 40, and a minimum of 2.5 days at age 36 (Treloar et al., 1967).

(c) Duration

The duration of menstrual blood loss varies normally in different women from 2 to 7 days with a mean of 5 days (Guilleband & Bonnar 1978).

(d) Amount of blood loss
The best measure of the total amount of menstrual bleeding is estimated from the total haemoglobin extracted from all tampons, towels and measured by the alkaline haematin or other standard method. (Van Eijkeren et al., 1986). A round figure of 40 ml for the average menstrual blood loss (MBL) suffices for practical purpose. (Dewhorst 1995) 79 ml is generally accepted as the upper limit for the normal range (Hallberg et al., 1966). About the relationship between MBL and duration, Rybo (1966) found that if the duration of menstruation exceeded 7 days the mean MBL was greater than 50 ml. It is generally agreed that 90% of the MBL occurs in the first 48-72 hrs in both normal menstruation and also in menorrhagia.

Endocrine mechanism of menstruation

Play of sex hormones from hypothalamus in brain, anterior pituitary gland, ovary causes menstrual bleeding from uterine endometrium. This is called hypothalamus – pituitary – ovarian – uterine axis.

The hypophysial-portal system of blood vessels represents a pathway of information transfer from the hypothalamus to the adenohypophysis. Gonadotrophin output is controlled by messages, which reach from the brain, and the locus of the feed back action of oestrogen may be in the central nervous system.

In the brain, hypothalamus acts as switch to endocrine mechanism of menstruation and starts the process by secreting gonadotrophin-releasing hormone. (GnRH) or luteinising hormone releasing hormone (LHRH) by peptidergic neuron. The latter is controlled by aminergic neuron. Environment influences the menstruation via cerebral cortex and
hypothalamus. GnRH flows down from hypothalamus via pituitary portal vessels to anterior pituitary gland (gonadotroph cells) liberating, follicle stimulating hormone (FSH) and luteinizing hormone (LH) in blood circulation to initiate growth of ovarian follicle in both ovaries.

Ovarian follicles are grown in a menstrual cycle in 3 steps.

(a) Ovarian follicles are grown from primordial follicles. A single graffian follicle matures and become dominant by effect of FSH while other follicles undergo atresia.

(b) Oestradiol is secreted by maturing ovarian follicle which in circulation stimulates hypothalamus and ant pituitary to cause surge of LH & FSH hormones in blood on day 12 of menstrual cycle.

(c) Ovulation: It occurs generally on day 14th of menstrual cycle. Corpus luteum is formed in the shell of mature graffian follicle following ovulation due to LH effect. Corpus luteum remains mature from 19 to 26 days degenerates on 27 and 28 days if no pregnancy occurs in menstrual cycle.

![Schematic outline of the neuro-endocrine control of reproduction in the female Phospholipids](image-url)
The development and maturation of a follicle, ovulation and formation of corpus luteum and its degeneration constitutes an ovarian cycle. All these events occur within 4 wks. They consists of

1. Recruitment of groups of follicles.
2. Selection of dominant follicle and its maturation
3. Ovulation
4. Corpus luteum formation
5. Demise of corpus luteum

(1) Recruitment of group of follicles

The cohort of the growing follicles undergoes a process of development and differentiation which takes about 85 days and spreads over 3 ovarian cycles. The growth of primordial follicle into primary follicles is independent of FSH. It may be related to oocyte or epithelial growth factor. The oocyte is surrounded by an acellular barrier of polysaccharide produced by the follicular cells called zona pellucida.

(2) Follicle maturation

The graffian follicle was named after the Dutch physician and anatomist Reijnier de Graaf (1673). The development of antrum containing secondary follicle depends upon FSH. There is production of follicular fluid.
which is primarily an ultra filtrate of blood from the vessels within theca interna.

(3) Dominant follicle

As early as 5-7 days one of the follicles out of so many becomes dominant and undergoes further maturation. It seems probable that the one with highest antral concentration of oestrogen and lowest androgen: estrogen ratio and whose granulose cells contain the maximum receptors for FSH, becomes the dominant follicle. The rest of the follicle become atretic by the day 8. The follicular fluid is increased in amount. The fluid contains oestrogen, FSH, trace amount of androgen, oocyte maturation inhibitor, luteinization inhibitor, inhibin, proteolytic enzymes, plasmin etc.

A fully mature graffian follicle just prior to ovulation measures about 20 mm.

(4) Ovulation

The dominant follicle shortly before ovulation reaches the surface of the ovary. The cumulus becomes detached from the wall, that the ovum within the surrounding cells floats freely in the liquor folliculi. The follicle wall near the ovarian surface becomes thinner. The cumulus escapes out of the follicle by a slow oozing process taking about 1-2 mts along with varying amount of follicular fluid. A plug of plasma closes the stigma.

Factors causing ovulating

Endocrinal causes
Luteinizing hormone surge: (LH surge) Sustained peak level of oestrogen for 24-48 hrs in the late follicular phase results in LH surge from the anterior pituitary. Ovulation approximately occurs 24-36 hrs after the LH surge. LH peak level persists for about 24 hrs.

FSH rise: Preovulatory rise of 17 α-hydroxy progesterone facilitates the positive feed back action of oestrogen to induce FSH surge → increase in plasminogen activator → plasminogen → plasmin → helps lysis of the wall of the follicle. Thus the combined LH/FSH mid cycle surge is responsible for the final stage of maturation rupture and expulsion of oocyte.

Corpus luteum formation

After ovulation the ruptured graffian follicle develops into corpus luteum. The cells become larger poly hedral with pale vesicular nuclei and frothy cytoplasm. The cells are called granulose lutein cells. The colours of it is grayish yellow. Within 24 hrs small capillaries grow into it. But 4th day luteal cells attain the maximum size, have a distinctive yellowish colour. On 22-23 day of cycle retrogression starts. Corpus luteum has a life span of 12-14 days. The prostaglandin F₂ liberated from the ovary is luteolytic. Oestradiol is also considered to have luteolytic effect.

Oestrogen and progesterone are secreted by the corpus luteum. Together they maintain the environment for the growing ovum before the same is taken over by the placenta.

Oestrogen
The main and most powerful oestrogen produced by the ovary is oestradiol and less active oestrone is also secreted and both are found in circulation. Oestradiol is produced by the membrana granulose and the theca interna in increasing amounts as follicle ripens. The theca cells are the most important source of oestradiol. The total quantity of oestradiol formed during one cycle is estimated to be 10 mg.

Action on uterus

The primary effect of oestrogen on the uterus is to increase its vascularity and because of this as well as of its direct action on tissue cells. It leads to hypertrophy of myometrium. In presence of oestrogen it contracts regularly and forcefully and is sensitive to oxytocic drugs. Oestrogen causes proliferation of endometrial glands and growth and compaction of the stroma. It restores the endometrium including its coiled arteries. Endometrium suddenly deprived of an oestrogen influence breaks down and bleeds. By controlling the releasing factor of the hypothalamus oestrogen depresses the output of FSH, but stimulate the production of LH.

Blood

Oestrogen increases the coagulability of blood by raising the factors VII, VIII and X. They cause sodium and fluid retension in the body.

Progesterone

It is secreted by the theca lutein cells and granulose cells. Prior to ovulation when the theca interna begins to luteinize, and during luteal phase
the plasma progesterone level rises to 6 to 63 n mole/L falling in follicular phase to 1.3-6 n mol/L. The total amount of progesterone produced by the ovary during one cycle is 300-400 mg. Progesterone relaxes the smooth muscles throughout the body.

Endometrial cycle

The first four days are occupied with menstruation when two thirds to four fifth of the endometrium is shed. During the remaining 24 days the histological cycle consists basically of a proliferative and secretory phase.

Proliferative phase

At the conclusion of a menstrual period the remaining endometrium is necrotic, disorganized and devoid of surface epithelium, sometimes only the basal layer remains. The glands are macerated and broken. Repair proceeds quickly and the surface is re-covered with epithelium which grows from glands and stroma within hours of the completion of the menstrual phase. By the end of 2 or 3 days the surface intact new vessels are growing from the stumps of the old and the glands are reformed. At this stage the glands are small and lined by cuboidal epithelium, they have a regular outline. The glands increasing in size and becoming perpendicular to the surface. The epithelial cells become columnar with their nuclei situated basally but no secretory activity. The stromal cells become spindle shaped and assume a compact arrangement. At the end of proliferative phase the endometrium measures 2.3 mm.
Secretory phase

The secretory phase begins at approximately the 14th day of a 28-day cycle. During the phase the endometrium continues to grow to reach a maximum thickness of 5-7 mm. The glands increase in size and become active. The secretion collects first as a globule in the base of the cell to give rise to the appearance of sub nuclear vacuolation. The globule is latter seen on both sides of the nucleus but eventually collects near the lumen of the gland acinus and pushes the nucleus to the base of the cell. At the end of 5-8 days the secretion enters the gland lumen on its way to the uterine cavity, leaving the cells with their edges frayed. This secretion rich in glycogen, fructose and glucose has a nutritive function for any fertilized ovum reaching the uterus. The stroma as a whole becomes more vascular and oedematous. These give a reticular appearance.

Endometrial growth ceases 5 or 6 days before menstruation and within 1 or 2 days shrinkage is apparent. This occurs as a result of dehydration of stroma, decreased blood flow and discharge of secretion from the glands. The stroma gets infiltrated with leucocytes and some red blood cells. Menstruation is heralded by extravasation of serum and blood, which collect in small pools near the endometrial surface. These fragments the now necrotic endometrium and break through the surface epithelium carrying pieces of it away in the bloody discharge which is the menstrual flow.
Correlation of endometrial and ovarian cycles

Just before the onset of a menstrual period a new follicle begins to ripen in the ovary. Endometrial proliferation therefore occurs during the follicular phase in the ovary. Ovulation marks the change over from the proliferative to secretory phase in the endometrium. Secretory activity and decidual reaction are manifestations of the luteal phase in the ovary. The shrinkage of the endometrium premenstrually coincides with commencing failure of corpus luteum activity and is the direct result of withdrawal of the supporting effect of oestrogen and progesterone.

As a result of ovarian cycle the uterus is subjected to the influence, first of oestrogen and then combination of estrogen and progesterone. Both are withdrawn when the corpus luteum degenerates and menstruation occurs within a few days.

Mechanism of normal menstruation

In normal menstruation one half to three quarters of the menstrual discharge is blood, the rest being fragments of endometrial tissue and mucus. Menstrual blood does not normally clot and consists of aggregations of endometrial tissue, red cells and degenerated platelets and some fibrin. Large amount of fibrin degradation products are also present as the endometrium release fibrinolytic substances that normally cause any blood clots to disintegrate.

Changes in endometrium
The unique feature of primate female who menstruate is the existence of spiral arteries in the endometrium. In the proliferative phase the spiral arteriols grow upwards from the basal to the more superficial layers of the endometrium where a capillary network develops. In the luteal phase there is a marked increase in length and coiling of the spiral arteriols, which are also dilated. Premenstrually the endometrial glands empty of secretion, the fluid from stroma is reabsorbed, the endometrium shrinks and the spiral arteriols become more coiled upto light loops. Immediately before menstruation the spiral arteriols constricts intensely for a period of 4-24 hours and then dilate with a massive extravasation of erythrocytes into the stroma of the endometrium, particularly the spongiosa layer. Numerous bloodlakes appear and the areas of haemorrhage coalesce to lift off islands of devitalized endometrium. Spiral arterioles are essentially end-arteries supplying separate narrow longitudinal segments of endometrium without any anastomoses, making the superficial layers or endometrium vulnerable to ischaemia but also facilitating haemostasis. Bleeding occurs from the coalesced blood lakes and from the latter being slower and continuing longer. 75 per cent of menstrual blood loss is arterial and 25 per cent is venous.

Only about one quarter of the total endometrium is shed, the majority involutes and is reabsorbed, Markee (1950) postulated that the key event in menstruation is the vasoconstriction of the spiral arterioles due to the liberation of an unknown substance in the endometrium which produce vasoconstriction, resulting in damage to the walls of the spiral arteriols and necrosis of the superficial layers of endometrium.

Role of eicosanoids
The role of eicosanoids including prostaglandins PGF$_2$$\alpha$ and PGE$_2$ and the prostanoids prostacyclin (PGI$_2$) thromboxane (TxA$_2$) and leucotrienes has been reviewed by Abel (1985), Smith (1985), Van Eijk’eren (1989). Prostanoids are not stored in the tissues but are synthesized and released as required and are mainly formed from free arachidonic acid which is released from phospholipids in cell membrane by phospholipase A$_2$. This may be released physiologically but is also released in considerable quantity with cell ischaemia and death. Arachidonic acid is initially metabolized by cyclo-oxygenase into unstable endoperoxide (PGG$_3$ & PGH$_2$) which are rapidly converted by specific synthetase into PGF$_2$$\alpha$ which is vasoconstrictor and weakly platelet aggregatory PGE$_2$ (Vasodilator and weakly platelet anti aggregatory). PGD$_2$ a platelet aggregation inhibitor PGI$_2$$\alpha$ potent vasodilator and inhibitor of platelet aggregation and TxA$_2$ a potent vasoconstrictor and platelet aggregator. All prostanoids are rapidly metabolized and inactivated and are believed to act primarily at their site of synthesis.

Normal menstruation

In the proliferative phase the endometrium synthesizes roughly equal amounts of PGF$_2$$\alpha$ and PGE$_2$. But in the luteal phase the levels of PGF$_2$ progressively increase under the influence of oestradiol and progesterone. In normal menstruation the ratio of PGF$_2$ - PGE$_2$ in menstrual fluid is roughly 2:1 so that it is the vasoconstrictor and platelet aggregatory action predominate. There is considerable evidence that PGF$_2$ is the vasoconstrictor substance originally postulated by Markee (1950) and the
smooth muscle stimulant first demonstrated in the menstrual fluid by Pickles (1957).

Though the endometrium produces large amounts of PGF$_2\alpha$ and PGE$_2$ and also small amount of PGD$_2$ it produces virtually no PGI$_2$ or TxA$_2$ though the failure to demonstrate the later could be due to methodological difficulties. The myometrium produces considerable amounts of PGI$_2$ and its stable metabolite PGDF$_1$ having limited ability to synthesize PGI$_2\alpha$ from arachidonic acid but able to generate large amounts from intermediate endoperoxide produced in the endometrium. Endoperoxides are believed to diffuse from the endometrium into the myometrium stimulating PGI$_2$ synthesis PGI$_2$ may then diffuse back into the endometrium producing vasodilatation and inhibiting platelet aggregation. In normal menstruation it is postulated that the PGF$_2\alpha$ synthesized in the endometrium first produces vasoconstriction of the spiral arterioles, and as a result an increased proportion of the endoperoxides produced from arachidonic acid by

![Diagram of Prostaglandin Synthesis](image-url)
prostaglandin synthetase are deviated into the myometrium which then produces a surge of PGI$_2$. This surge may then diffuse back into the endometrium, producing the dilation which follows the vasoconstriction of the spiral arterioles immediately preceding the onset of menstruation.

Role of leukotriens

Leukotriens are eicosanoids produced by the action of lipoxygenase on arachidonic acid where as prostanoids are produced by the action of cyclo-oxygenase. They are responsible for the vascular lesion and endothelial gaps in the spiral arterioles and venules which precede menstruation.

Fibrinolytic and lysosomal enzymes

Menstrual bleeding follows the withdrawal of oestrogen and progesterone and also of progesterone alone. Progesterone has been shown to promote the formation of lysosomes in the endometrium (Henzl, 1972) and ultra structure studies have shown that the progesterone has a stabilizing effect and oestrogen has a labilizing effect on the lysosomes in the endometrium. The withdrawal of progesterone preceding menstruation probably causes a breakdown of lysosomes and the release of phospholipase A$_2$. This in turn causes the formation of large amount of arachidonic acid from phospholipids in the cell walls and initiates the prostanoids and the synthesis of PGF$_2$$\alpha$, PGE$_2$ and PGI$_2$. The sudden increase in prostaglandins particularly PGF$_2$$\alpha$ is responsible for the spasmodic contraction of the spiral arterioles and for menstruation. Progesterone has a paradoxical effect in that both progesterone and oestrogen are necessary to produce the maximum
synthesis of prostaglandins by the endometrium. This results in a progressive increase in corpus luteum insufficiency or anovulatory cycles and eventually in cessation of menses.

Components of menstrual flow

Blood escaping from endometrial capillaries and arterioles and shed functional layers of the endometrium are the major components of the menstrual discharge. The amount of blood loss approaches three fourth of the menstrual flow. The mean blood loss ranges between 10 and 60 ml. This corresponds to the average loss of iron per menstrual period of 14 mg.

Lack of clotting is a characteristic feature of the menstrual blood. Menstrual blood does not contain fibrinogen and that it possesses fibrinolytic properties led to the assumption that clotting flowed by fibrinolysis occurs in the uterus. Plasminogen activators were demonstrated in high concentration in the menstrual blood. The activity of these enzymes in the uterine endometrium increases progressively during the secretory phase, reaching maximum level just prior to menstruation. (Rybo, 1966) Another plasminogen activator similar to urokinase has been recovered from human uterine extracts during the proliferative phase (Kok, 1979). It has been found in increased amounts in women wearing intrauterine devices. Menstrual clots are not true clots, since they do not contain fibrin deposits. They are mere aggregates composed of red cells, mucoid substances, mucoproteins and glycogen. The high fibrinolytic activity of the endometrium is probably necessary to effect emptying of the uterus during
menstruation. An increase of this fibrinolytic activity may result in excessive menstrual blood loss.

In addition to blood cells and endometrial cells, several other cellular elements are found in the menstrual discharge. This includes macrophages, histiocytes, mast cells and vaginal epithelial cells. Also a variety of chemical substances found normally in blood or cellular material have been identified in the menstrual flow. Among these chemicals are heparin like substances and prostaglandin. Heparin like activity in concentrations of 2 to 3 u/ml has been found in the uterine fluid with a tendency to increase towards the end of the cycle and decrease again during menses (Foky, 1978). The presence of such a substance which is probably derived from mast cell granules present in the endometrium and myometrium have a role in maintaining the menstrual blood incoagulable.
The disease ASRUGDARA was known to mankind ever since the year of puranas. A vivid description regarding its aetiology, pathogenesis, symptomatology etc. are given in puranas. Description about streerogas are also available in Vedas and the management was mainly daiva- Vyapasraya.

All Ayurvedic classics including Charaka Samhita, Susrutha, Samhitha, Ashtanga Samgraha, and later works like Bhavaprakasa, Yogatharangini, Bhasava Rajeeyam, Vangasena etc contain description of rekta pradara or asrugdara in detail.

In Ayurveda a single disease may be known by different synonyms in different classics. This is true for Asrugdara also. Charaka Acharya has described its nidana, and samprati in detail in the chapter ‘Yoni Vyapat’ of Chikitsa Sthana (30th chapter)\(^1\). He liberally used the term pradara. Susrutha Acharya in Sukla Sonitha\(^2\) Adhyaya of Sareera Sthana described asrugdara as excessive menstrual bleeding. Vagbhata in ‘Puthrakameeya’ adhyaya of Sareera sthana\(^3\) has said that rakta yoni, asrugdara and pradara are one and the same disease. Where as in Ashtanga Hridayam description about rekta yoni is given and nothing is mentioned about asrugdara or pradara.
NIRUKTHI (DEFINITION)

Asrugdara is derived from two words.  1. Asruk meaning blood or rekha.  2. Daram meaning stream or continuous excessive flow.  Thus asrugdara means flow or stream of excessive blood flow. Various acharyas have defined asrugdara as follows.

1. Excessive flow during menstruation.
2. Prolonged flow during menstration.
3. Excessive flow occurring at menstrual or intermenstrual period.

SYNONYMS
1. Pradara – due to pradeerana (excessive excretion) of rajas.
2. Rekta pradara – Pradeerana of rekta through yoni.
3. Rektayoni – Athisruthi (excessive flow) of rektha through yoni.

Main symptom is excessive menstrual blood flow. This disease is mainly described in Astanga Hridayam and Samgraham. Vagbhata I considers pradaram and Rektayoni as one and the same disease.

4. Asruja – Excessive flow of as asruk even after conception.

Main symptom is excessive bleeding. This disease is described only in Charaka Samhita. It arises due to vitiation of rekta and pitha. Bleeding may be of prolonged duration as it is said that the bleeding occurs even after conception.
As asrugdara is caused by multiple factors a through knowledge of nidana, poorva rupa, samprati, lakshnas, and upasaya enable the physician to better understand the disease and thus prevent its progression.

Nidana (Aetiology)

Nidana can be divided into 1. Sannikridhta nidana and 2. Viprakrishta nidana. Sannikrishna nidana is dosha prakopa and is brought about by viprakishta nidana. The latter is further divided into 3.


The aetiological factors of asrugdara can be grouped into

1. Factors which vitiate doshas
2. Factors which vitiate rekta
3. Factors which vitiate both

General aetiology (Samanya nidanam)

A. Aharaja

1. Excessive intake if lavana (salt) amla (sour) and katu (pungent) rasas.

2. Excessive intake of food having the characters of guru (heavy) snigda (unctuousness) and vidahi.

3. Regular use of mamsa such as gramya mamsa, odaka mamsa.

4. Regular use of madya, dadhi, sukta, mastu.

5. Use of krisara and payasa excessively.

6. Use of virudha (incompatible) ahara.
7. Intake of food before the digestion of previous food taken (Adhyasana).

**B. VIHARAJA**
1. Doing work more than one’s own strength. (Ativyayamam)
2. Running, riding etc.
3. Day sleeping (Diva swapna)
4. Weight bearing
5. Excessive coitus (Athi maiduna)
6. Excessive thinking, worrying

**C. KALAJA**
1. Sarat kala
2. Afternoon

Specific aetiology (Vishesha nidanam)

Excessive use of rooksha (dry), cold and astringent foods, hard working, weight bearing etc are specific aetiological factors for provoking vata and leads to vataja asrugdara. Excessive use of amla, lavana, kshara and agni sevana etc leads to pithaja asrugdara. Regular and excessive use of guru, snigda, Seetha ahara and divaswapna etc. leads to vitiation of kapha and terminates in kphaja asrugdara.

Due to the aetiological factors above described, doshas are vitiated and also the dushyas mainly rekta, and deranges the rthu chakra, leading to the abnormality of arthava, resulting in asrugdara.

Women suffering from gynaecological disorders, (yoni rogas), infertile women (vandya) and one who had repeated abortions are more prone to get asrugdara.
Poorva roopa

Poorva roopa or prodromal stage are those symptoms and signs, before the actual manifestation of the disease. It is otherwise called as sthana samsraya state (Fourth kriyakala). Due to sroto vaigunya, the kicakisation if vitiated doshas in particular areas of body takes place and the dosha – dushya samoorchana occurs.

In pradara, poorva roopas are not seen. Since asrugdara is a disease with the cardinal feature of excessive menstruation, the prodormal stage which is occurring before menstruation is generally insignificant.

Classification of pradara

In Ayurveda, diseases are classified as per doshic predominance. Pradara is classified into four types

1. Kaphaja
2. Pithaja
3. Vataja
4. Sannipathaja

Susrutha acharya has not given any classification. But has said that treatment should be based on doshic predominance or character of rekta vitiated by different doshas. This is emphasised by Vagbhata also. Consisting these openions, dwidoshaja asrugdara should also be included in the above list. Charaka acharya has described the treatment for vatapitha pradara, indicating the importance of dwidoshhaja type. But in critical analysis it can be seen that, the main dosha being vata and main dushya being rekta and wherever rekta is vitiated, one can expect the vitiation of pitha also, as there
exists an asruya – asrayee bandha. So vitiation of pitha is a rule in all types. Thus it is better to avoid dwidoshaja variety and accept the four varieties of pradara.

Roopa (Lakshana)

Disease is indicated by the symptoms. Roopa or state of manifestation of a disease is the fifth stage of shad – kriyakalas of Susrutha. Lakshanas can be divided into two

1. Samanya lakshanas
2. Vishesha lakshanas

1. Samanya lakshanas
   1. Excessive menstrual/intermenstrual bleeding.
   2. Pain all over the body

2. Vishesha lakshas

   Vishesha lakshas arise from specific dosha dushti. Kaphaja pradara.

   Menstrual blood in slimy, heavy, unctuous, cold, pale and discharged with mild pain. Associated features like vomiting, distaste, anorexia, dyspepsia, cough and dysnoea are also present.

   Susrutha acharya has said, in addition to the above symptoms of menstrual blood passage of clots or threads, vasa gandha, and dalty taste will also be present in arthava.
Pithaja pradara

Menstrual blood is bluish red or yellowish red, hot continuous flow, and there is intermittent pain. Susrutha acharya has said that, since these is continuous blood flow, clots are usually absent, and it smells like gomuthra and has got a pungent taste and looks like makdhika.

Associated general symptoms present are burning sensation, increased body temperature, thirst, vertigo and loss of consciousness.

Vataja pradara

Menstrual blood looks like thin, dry, pale and blakish red. Additional symptoms of menstrual blood mentioned by susrutha acharya are; it is cold to touch, flows rapidly without clots and is clear. It has got astringent taste and loha gandha. Associated general symptoms includes intense pain at back, lower abdomen, sides, inguinal region etc. Excessive flow may or may not be associated with pain.

<table>
<thead>
<tr>
<th>Features</th>
<th>Vataha</th>
<th>Pithaja</th>
<th>Kaphaja</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Color</strong></td>
<td>Blackish red</td>
<td>Yellowish red</td>
<td>Pale red</td>
</tr>
<tr>
<td>2. Appearance</td>
<td>Thin &amp; frothy</td>
<td>Viscous but not clotted</td>
<td>Heavy &amp; clotted on with threads</td>
</tr>
<tr>
<td>3. Nature of flow</td>
<td>Speedy</td>
<td>Continuous</td>
<td>Slow</td>
</tr>
<tr>
<td>4. Pain</td>
<td>Intense</td>
<td>Intermittent</td>
<td>Mild</td>
</tr>
<tr>
<td>5. Feel to touch</td>
<td>Dry and cold</td>
<td>Hot</td>
<td>Slimy &amp; cold</td>
</tr>
</tbody>
</table>
6. Taste | Astringent | Pungent | Salty  
--- | --- | --- | ---  
7. Smell | Loha Gandhi | Matsya Gandhi | Vasa Gandhi  
8. Associated features | Pain on black sacral region low abdomen | Increased temperature thirst, burning sensation, vertigo | Vomiting dyspepsia, swasa, kasa  

Sannipathaja pradara

In Sannipathaja type, all the features mentioned above will be present. Menstrual blood is foul smelling, slimy yellowish and with burning sensation, flow looks like sarpee (ghee) or majja (bone marrow).

Samprathi (Pathogenesis)

The manner in which dosha-dushya samoorchana takes place, leading to the manifestation of a disease is termed as samprapti. This evolution stage starts with chaya (accumulation of doshas at their own place) and end in Vyakti of bheda. Thus the complete picture of a disease is the vitriated doshas and morbid condition of one or more tissues and organs of the body.

Pradara is included under one of the rekta dohsha vyadhies. The moola sthana of rekta vaha srotas are yakrit and pleeha. The defective function of these organs leads to rekta dushti and dosha dushti, mainly pitha dushti. Excessive in take of amla, lavana, katu rasas, madya, sukta and sura etc will increase the pramna (quantity) of rekta and this rekta instead of circulating to all body parts equally, passes more through arthava vaha.
srotas. This abnormal passage of blood through arthava vaha srotas is hinted by Bhela acharya as dushta marga pratipanna sonitha.

Thus the quantitatively increased and qualitatively changed rekta along with pitha will pass through sookshma siras of rektadhaara kala of garbhasaya and covers or obstructs the apana vata. Thus the vitiated apana vata excretes the dushta arthava through yoni marga as excessive menstrual bleeding. The character of menstrual blood and its flow will vary according to the degree of vitiation of doshas.

Diagram Shows the Samprati of Asrugdaram
Vyakthi  
Bheda  

Excessive bleeding

Asrugdaram
Upadravas (Complications)⁸

Upadrava or complication is defined as a symptom or a disease which has its origin in the original disease and which is developed after the primary disease.

Dourbalya (weekness), bhramam (giddiness), moorcha (unconsciousness), mada (psychosis), thrisha (thirst), daha (burning sensation of body), pralapa (delereum), pandu (anaemia), thandra (uneasiness or sleepiness), akshapaka (convulsions) and disorders of vata, sopha (oedema) etc are the complications of pradara. Most of the upadrava vyadhese of pradara arise from the excessive menstrual bleeding and to dhathu kshaya, and dosha dushti. This may upset the psychology of the women, exaggerating the symptoms.

Sadhya – Asadhyata (Prognosis)

Sannipathaja pradara is asadhyata or incurable. Women suffering from excessive continuous menstrual bleeding, suffering from thirst, having very short internenstrual period are also considered asadhyata, according to Madhava acharya. All other types are sadhya or curable.

Vyava cheda nidana (Differential diagnosis)

1. Arthava dusthi

In arthava dusthi, symptoms and signs of arthava vitiated by single or combination of doshas are described. They represent only a temperory pathological condition. They do not cause any associated general symptoms. Even though the characteristics of discharged menstrual blood in arthava
dushtti resemble that of pradara, but the excessive menstrual bleeding, irregularity of the rthu chakra and associated symptoms are only seen in pradara.

2. Vatiki

Vataki pradara has to be differentiated from vataki yoni vyapth in vataki, characteristics of menstrual blood resembles that in vatika pradara but excessive bleeding and inter menstrual bleeding are not seen. Local symptoms like the feeling of displacement of vamkshana are seen in vatiki.

References

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3. A. Sam. Sareera Sthana
4. Ch. Sam. Chi. 30/206
5. Ash. Hri. Ni. 26/45 Arunadatta tika
6. Ch. Chikitsa 30/204
7. Ch. Chi. 30/211
8. Su. Sa. 2/16
DYSFUNCTIONAL UTERINE BLEEDING (DUB)

The gynaecological health of a woman depends to a large extend on the normaly or otherwise of her menstrual cycle. Among the menstrual abnormalities Dysfunctional uterine bleeding (DUB) is a common disease comprising about 15% - 20% of new patients attending the hospital. In every case primary fault lies in the in-appropriate hormone production.

DEFINITION

Dysfunctional uterine bleeding (DUB) is defined as abnormal bleeding from the uterus, in the absence of organic disease of genital tract (Dewhurst, 1995).

DUB is not a condition with one aetiology but is a group of disorders characterized by dysfunction of uterus, ovary, pituitary, hypothalamus or other part of reproductive system which results in abnormal or excessive uterine bleeding. Diagnosis of DUB is a diagnosis by exclusion. Menstrual blood loss (MBL) is said to be excessive when it exceeds 80 ml/cycle (Dutta, 2000).

CLASSIFICATION

Menstrual bleeding mainly depends on development of endometrial tissue which in turn depends on the hormones and their metabolites. Duration, interval, and amount of bleeding may vary according to the phase of development of endometrium. Based on that DUB can be divided into following groups.
Based on aetiological factors, DUB can be divided into two (Dewhurst and Dutta, 2000)

1. Primary – Due to dysfunction of uterus, ovary, pituitary, hypothalamus

2. Secondary – To IUCD, contraceptive pills, thyroid dysfunction, blood disorders.

OVULATORY DUB

A) Functional equimenorrhoea and equimenorrhagia

Here menstruation occurs too frequently at interval of three weeks or less. It usually occurs following childbirth, abortion, during adolescence and premenopausal period and in pelvic inflammatory disease.

B) Functional Menorrhagia
Heavy or prolonged menstrual loss at normal intervals. Here the endometrium will be of secretary type as shown by studies of Dutta (2004) and Jeffcoate (2005). This may fall into following two varieties.

1) Irregular ripening endometrium

In this case endometrium is without adequate hormonal support. So slight losses or spotting occurs for many days before the proper flow starts. The deficiency of progesterone may be recognized on histopathological examination. This is due to inadequate functioning of corpus luteum.

2) Irregular shedding endometrium

Here slight bleeding continues intermittently for several days after the proper flow. Incomplete degeneration of corpus luteum is the cause. A persistent corpus luteum may result in continued secretion of oestrogen and progesterone, and absence of the normally sharp fall in oestrogen and progesterone secretion, which proceeds menstruation. This will result in irregular shedding.

While studying DUB cases Jeffcoate (2005) described the mixed pattern and found that the fault lies in corpus luteum.
ANOVULATORY DUB

1. Metropathia Haemorrhagica (Schroeder’s disease)

The characteristic symptom of this type of DUB is that there is continuous heavy bleeding for 2-8 weeks, proceeded by a short period of amenorrhoea of 2-3 months. Jeffcoate (1992) in his work on DUB showed the presence of large thin walled venous sinuses in endometrial hyperplasia and the rupture of the same is responsible for bleeding. In this case the persistent follicle produces continuous oestrogen but ovulation fails to occur and there is no progesterone. So bleeding is painless. Dewhaurst (1995) found that bleeding in metropathia haemorrhagica is due to hyperplasia of endometrium in response to hyper oestrogenism from unruptured follicles.

If the higher levels of oestrogen are maintained, benign hyperplasia, adenomatous hyperplasia and eventually carcinoma of endometrium may occur. In Metropathia Haemorrhagica (M.H.) the characteristic finding in endometrium is the swiss cheese pattern of glands termed as cystic glandular hyperplasia. M.H. comprises about 1/4 - 1/3 of all cases of DUB. It commonly occurs few years preceding menopause. Persistent graafian follicle is due to abnormal gonadotrophin stimulus, causing absence of secretion of LH or ovaries becoming resistant to LH.
2) Threshold bleeding

The classical symptom is prolonged bleeding. With insufficient follicular development, there is inadequate production of oestrogen and inadequate proliferation of endometrium without secretory change, resulting in a thin or deficient or atrophic endometrium. Oestrogen may reach the threshold level to cause bleeding but not enough to produce full proliferative phase. Ovary inactiveness is thought to be reason for this type of DUB. This is seen in young girls and also in women aged 45-50 yrs.

**PATHOLOGY OF DUB**

**PRIMARY DUB**

Till the end of the last century dub was thought to be due to chronic inflammation and uterine muscular insufficiency. But the current concept concludes that abnormal bleeding is most likely due to local causes in the endometrium postulated mechanism is,

1. Excessive endometrial secretion of PGE$_2$
2. Excessive prostulated PGI$_2$
3. Excess fibrinolysis leading to failure of defective plugs.
4. Defect in spiral vessels.
5. Increase in endometrial vascular system is affected by sympathetic nervous system.
6. Environmental vascular system is affected by sympathetic nervous system.
Environmental Stress, undernutrition, anxiety etc affect the endometrial vascular function.

**Role of Eicosanoids**

Towards the end of secretary phase oestrogen and progesterone levels falls.

This causes the break down of lysosomes liberating phospholipidase A<sub>2</sub>, which act on phospholipids of cell well producing arachidonic acid. Arachidonic acid in initially metabolized by cyco oxygenase into unstable endoperoxides, which are converted by specific synthetases to

- \( \text{PGF}_2\alpha \) - Vasodialator and weakly platelet aggregatory
- \( \text{PGF}_2 \) - Vasodialator and weakly platelet antiaggregatory
- \( \text{PGD}_2 \) - Platelet aggregator inhibitor
- \( \text{PGI}_2 \) - a potent vaso dialoter an inhabitor of platelet aggregation
- \( \text{TxA}_2 \) - a potent vaso constrictor and platelet aggregartor.

\( \text{PGF}_2\alpha \) is first produced in large amount bringing the vaso constriction of spiral arterioles. This intense spasm will cause stasis of blood and necrosis of tissues and vessel walls (Dewhurst, 2000)

**Role of Leukotriens**

Leukotriens are ecicosanoids produced by the action of lipo oxygenes on arachidonic acid. Leukotriens are responsible for the vascular lesions and endothelial gap in the spiral arterioles and venules which proceed menstruation. They are produced
predominantly by leucocytes and leucocytic infiltration is characteristic of late secretory endometrium. Degree of MBL is roughly proportionate to degree of infiltration.

Phospholipids

Arachidonic acid

Lipoxygenase
Pathway

Cyclo oxygenase
Pathway

Prostaglandin endoperoxides

Specific PG

Synthesis

Tx A₂ PGE₂ PGF₂α PGD₂ PGI₂

Thromboxane

In anovulatory DUB there is absence if progesterone. So deficient synthesis of PGF₂α resulting in decrease in PGF₂α: PGF₂ ratio leading to vasodialation and increased MBL.

In ovulatory DUB the amount of prostaglandins produced is same but there may be change in relative activities of synthetases leading to the predominance of PGE₂ or PGI₂ resulting in vasodialatation.
In corpus luteal insufficiency the deficient production of progesterone leads to decrease in PGF$_2$α : PGE$_2$. While in persisting corpus luteum continuous secretion of oestrogen and progesterone leads to inadequate release of phospholipidase A2.

Role of Fibrinolytic and Lysosomal enzymes

Progesterone has got stabilizing effect and oestrogen has got labilising effect on lysosomes, in the endometrium. Withdrawal of progesterone preceding menstruation probably cause a break down of lysosomes and release of phospholipidase A2. Excess MBL could be due to an increased formation of lysosomes with an increased synthesis of phospholipidase A2 and of arachidonic acid and prostaglandins at menstruation, is believed to occur in Ovulatory DUB. Marked increase in plasminogen activities and fibrinolytic plasmin in DUB are shown by Sheppard and Bonner.

Role of Ovarian hormones

Recent works showed that endometrial oestrogen and progesterone receptors are more in late secretory phase in women with ovulatory DUB. Actually the ovarian hormones are working through prostaglandins and fibrinolytic enzymes. Endometrial hyperplasia seen in anovulatory DUB suggests that hormones forms a major aetiological factor in DUB.

SECONDARY DUB

1. Secondary to steroidal contraceptives and IUCDs
Excess and irregular DUB occurs with continuous oral administration of low-dose progestogens, injectable depot progestogens and IUCDs. (Diezfalussy et al., 1980) Low dose progestogens cause under development of spiral arterioles and dialated (Maqueo 1980) Large dose progestogens cause large superficial dialated venules and atrophic endometrium. IUCDs superficial ulceration of endometrium and increased vascularity and, marked leucocytic infiltration (Foley et al., 1978; Aparicio et al., 1979).

2. Secondary to systemic diseases

DUB occurs in bleeding disorders like thrombocytopenia thrombocytopenic, afibrinogenaemia, deficiency of factor II, V, VII, X & XI, anaemia, leukemia, diseases like some cases of chronic hypertension, heart diseases with chronic congestive failure, chronic nephritis, undernutrition – avitaminosis, hepatic dysfunction; emotional disturbances, like worry, sorrow, prolonged taking of drugs like aspirin, anticoagulants, psychotrophic medications; diseases producing pelvic congestion like PID; endocrine disorders like obesity, PCOD, hypothyroidism, etc. (Scott and Mussey, 1964; Simpson and Christakos, 1969).

CLINICAL PRESENTATION AND DIAGNOSIS

a. Adolescents and Teenagers

Abnormal uterine bleeding (AUB) in adolescent girls is almost always dysfunctional and organic disease and malignancy are particularly rare. DUB in this age group is primarily due to immaturity of hypothalamo-pituitary – ovarian – endometrial axis. Mostly anovulatory DUB.
b. Adults

DUB between the age of 20-30 years is most commonly due to benign disease of the genital tract. (Including PID, fibromyomas) and also due to DUB which is mostly ovulatory.

c. Perimenopausal group

DUB in women over 40 years is most commonly dysfunctional though fibromyoma, carcinoma are also seen. Anovulatory DUB is predominant in this group.

II. By bleeding pattern

a. Regular cyclic bleeding

Excessive regular cyclic bleeding is due to DUB, or PID or fibromyoma, or rarely malignancy. DUB is mostly ovulatory.

b. Irregular or Acylical bleeding

This is due to organic disease, carcinoma and DUB. DUB is mostly anovulatory.

c. Intermenstrual bleeding

Intermenstrual bleeding in association with normal regular bleeding is often dysfunctional. This pattern of bleeding is also seen in endometrial poly, submucous uterine fibromyoma and cervical carcinoma.

**DIAGNOSIS**

1. History
Includes age, parity and fertility, amount, duration and pattern of uterine bleeding, any associated gynaecological problems etc.

2. Examination
   1. Systemic
   2. Pelvic

3. Investigations
   1. Haematology – Hb, BT, CT, DC, TC, ESR, platelet count
   2. Endometrial curettings
   3. Hysterography
   4. Hysteroscopy
   5. Laproscopy
   6. Cytology
   7. Hormonal assay

Endometrial curettage remains the commonest investigation in DUB, if serves 3 purposes.

1. To exclude intra uterine disease like fibroid, polyp, Cancer, etc.
2. To determine the functional state of endometrium – secretory, proliferative, mixed, atrophic.
3. To arrest bleeding if it is severe or persisting.

<table>
<thead>
<tr>
<th>Pattern of bleeding</th>
<th>Time for curettage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyclic</td>
<td>5-6 days prior to periods</td>
</tr>
<tr>
<td>a. Menorrhagia</td>
<td>3rd or 4th day of menstruation. Soon after periods starts</td>
</tr>
<tr>
<td>b. Irregular sheeding</td>
<td></td>
</tr>
</tbody>
</table>
Based on bleeding pattern and the type of endometrium obtained through curettage, DUB can be easily classified and the cause can be identified.

### TABLE CLASSIFICATION OF POSSIBLE ENDOCRINE ABNORMALITY ASSOCIATED ENDOMETRIAL HISTOLOGY AND TYPICAL BLEEDING PATTERN IN DUB

<table>
<thead>
<tr>
<th>Type</th>
<th>Endocrine abnormality and, endometrial histology</th>
<th>Typical bleeding pattern</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Ovulatory</td>
<td>Short cycle-short proliferative phase normal endometrium</td>
<td>Polymenorrhoea Menorrhoea Menorrhagia</td>
</tr>
<tr>
<td></td>
<td>Longcycle – Long proliferative phase normal endometrium</td>
<td>Premenstrual spotting Menorrhagia</td>
</tr>
<tr>
<td>Corpus Luteum abnormality</td>
<td>Inufficiency – short luteal phase – irregular or deficient secretory endometrium (mixed</td>
<td>Premenstrual spotting Menorrhagia</td>
</tr>
<tr>
<td>Pattern</td>
<td>Description</td>
<td>Associated Conditions</td>
</tr>
<tr>
<td>---------</td>
<td>-----------------------------------------------------------------------------</td>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>Persistant-irregular endometrium-mixed pattern</td>
<td>Prolonged Menstruation</td>
<td></td>
</tr>
<tr>
<td>Anovulatory</td>
<td>Insufficient follicle - short cycle, atrophic endometrium</td>
<td>Polymenorrhoea Menorrhagia</td>
</tr>
<tr>
<td></td>
<td>Persistent follicle PCOD prolonged cycle, proliferative or hypereplastic endometrium</td>
<td>Oligomenorrhoea Metropathia Haemorrhagirca</td>
</tr>
</tbody>
</table>

**ENDOMETRIAL PATTERN IN DUB**

1. Normal secretary endometrium
2. Endometrial hyperplasia
   a. Proliferative
   b. Cysto glandular hyperplasia
   c. Adenomatous hyperplasia
3. Mixed endometrium
4. Atrophic endometrium

Normal secretary endometrium
The surface epithelium columnar, nuclei are placed at the middle, ciliated at some places, glands are larger, there is subnuclear vacuolation, followed by its exudation and crenated appearance. Glands are cork screw shaped. Marked spiraling of vessels. Stromal cells are larger and polyhedral. Infiltration of leucocytes and monocytes in stroma are seen towards the end of the phase. Thickness of endomertium is 5-6 mm.

Proliferative endometrium

Epithelium changing from cuboidal to columnar with nuclei at base. Epithelium of one gland becomes continuous with the neighbouring gland. Mitosis evident in stromal cells. Glands are tubular and lie parallel or perpendicular to surface. Stromal cells are compactly arranged. Spiral vessels sends loose capillary network. Evidence of sub epithelial congestion. Thickness of endometrium is 3-4 mm or more.

Cystic glandular hyperplasia

Endometrium markedly overgrown, thick and polypoidal. Glands increase in number but varies in size. Some of them become dialated and cystic. This give rise to swiss cheese pattern. There is no secretion, so glands are empty. Stromal cells becomes abundant and compact. There is hyperplasia of all elements in the endometrium.

Adenomatous hyperplasia
There is disparity between the proliferation of gland and the stroma. Endometrial glands increase in number with bud like glandular projections into stromas (Dawn, 2000).

Mixed endometrium

In irregular ripening, the endometrial curetting done on first day of menstruation will show patchy secretary changes amidst proliferative endometrium. In irregular shedding, curetting done on 4th or 5th day will show mixed secretory and proliferative endometrium. There is total absence of absence of any surface epithelium.

MANAGEMENT OF DUB

1. General

   Bed rest, assurance, sympathetic handling, are helpful particularly in adolescents. Anaemia should be corrected by nourishing diet, haematinics and blood transfusion in severe cases.

2. Conservative

   Hormone therapy is given to stop the acute episode bleeding and to regulate the cycle.
Table shows Hormonal Management of DUB based on HPR

<table>
<thead>
<tr>
<th>History</th>
<th>Interpretation of abnormality</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absent/atrophic/deficient proliferation</td>
<td>Deficient oestrogen or excess progesterone (depot or oral)</td>
<td>A/C Premarin 25 mg, IV, oestrogen dominant oral contraceptive pill (OCP)</td>
</tr>
<tr>
<td>Irregular ripening/proliferative/hyperplasia</td>
<td>Anovulation or corpus luteum insufficiency progesterone inadequate</td>
<td>A/C large dose progesterone. Progestogen dominant OCP 15 to 25&lt;sup&gt;th&lt;/sup&gt; day 5 to 25&lt;sup&gt;th&lt;/sup&gt; day</td>
</tr>
<tr>
<td>Normal secretary</td>
<td>? Non normal ? Receptor defect</td>
<td>A/C antifibrinolytics IV low dose OCP, or anti PG synthetase</td>
</tr>
</tbody>
</table>

**Non hormonal drugs**

Non steroidal anti inflammatory drugs (NSAIDS) they block the formation of prostaglandin PGI<sub>2</sub> etc. eg. Mefenamic acid (250 mg) 2 cap tds. for 3 days. Anti fibrinolytic agents like epsilon amino carporic acid (EACA), Tranexamic acid etc. Clomiphene citrate especially in menorrhagia with anovulation.

**Surgery and Radiotherapy**

1. Uterine curettage: Predominantly done as a diagnostic tool but it has got haemostatic, therapeutic effect by removing necrosed and unhealthy endometrium.
2. Endometrial ablative procedures: Hysteroscopic laser photo vaporization and also transcervical resection of endometrium using a 26 F gauge resectoscope. This is a best procedure alternative to hysterectomy.

3. Hysterectomy: This has got a number of advantages, like providing complete cure, avoidance of long term hormone therapy, and removal of any missed pathology. The primary deciding factors are severity of bleeding, age and parity and wishes regarding future pregnancies.

Radiotherapy

This has now got a small place in treatment of DUB. It is suitable where the patient is unfit for operation. Radiation menopause is free of immediate risk but leaves behind a potentially damaged organ which may develop into pyometra, haematometra or carcinoma (Dewhurst, 2000).

**COMPARISON OF PRADARA TO DYSFUNCTIONAL UTERINE BLEEDING**

After going through the various aspects of aetiopathogenesis of pradara described in Ayurvedic classics and DUB mentioned in modern medicine, it can be observed that both conditions are almost similar. Various points substantiating this view are shown below in the table.

<table>
<thead>
<tr>
<th>No.</th>
<th>Features</th>
<th>Pradara</th>
<th>DUB</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Description</td>
<td>As a disease</td>
<td>As a single entity</td>
</tr>
<tr>
<td>2.</td>
<td>Chief symptom</td>
<td>Excessive, prolonged or intermenstrual bleeding</td>
<td>Same</td>
</tr>
<tr>
<td>3.</td>
<td>Menstruation</td>
<td>Cyclical or acyclical</td>
<td>Same</td>
</tr>
<tr>
<td></td>
<td>Associated symptoms</td>
<td>General weakness, body pain, fatigue etc.</td>
<td>Same</td>
</tr>
<tr>
<td>---</td>
<td>---------------------</td>
<td>------------------------------------------</td>
<td>------</td>
</tr>
<tr>
<td>5.</td>
<td>Structural abnormalities</td>
<td>Not mentioned</td>
<td>Excluded</td>
</tr>
<tr>
<td>6.</td>
<td>Aetiopathogenesis</td>
<td>Arthava vaha srotho dushti, along with vitiation of vata and pitha causing irregular menstruation.</td>
<td>Imbalance in hypothalamo-pituitary-ovarion axis, causing hyperplasia of endometrium, irregular ripening and shedding of endometrium etc.</td>
</tr>
<tr>
<td>7.</td>
<td>Principle of treatment</td>
<td>Rekta sthapana oushada along with dosha samana</td>
<td>Haemostyptic measures along with regularization of hormone level in blood</td>
</tr>
<tr>
<td>8.</td>
<td><strong>Prognosis</strong></td>
<td>Prompt and early treatment gives good result. Only sannipathika type is incurable</td>
<td>Same</td>
</tr>
<tr>
<td></td>
<td>Hormone resistant cases needs hysterectomy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9.</td>
<td>Complications</td>
<td>Arise due to blood loss as anaemia, oedema, general weakness, burning sensation of whole body, psychosis.</td>
<td>Same</td>
</tr>
</tbody>
</table>

With all these points, it can be summarized that pradara can be correlated to dysfunctional uterine bleeding.

**HORMONE THERAPY IN DUB**
As it is found that DUB arises due to imbalance of endocrine system mostly, leading to irregular or decrease secretion of ovarian hormones, management of the disease with such kind of preparation will be more effective. Among the ovarian hormones progesterone preparations are more suitable with less side effects, however the effectiveness of combined preparation (oestrogen and progesterone) can not be excluded mostly in the case of puberty menorrhagia, Commonly available progesterone preparations are,

1. Medroxy progesterone acetate – (Provera, farlutal etc.)
2. Nor ethisterone – (Primolute-N)
3. Nor ethisterone – acetate (Steranil, Regesterone)

Among the combined preparations widely available, tablet form of the same with lowest dose (Ovaral G, N) were selected for the study.

PROGESTERONE THERAPY

Progesterone is a steroid hormone containing cyclopentano phenantherene ring nucleus. Synthetic progesterone are either derivatives of testosterone such as ethisterone or 19 nor, steroids. Such as nor – ethisterone derivatives of progesterone such as 17 a progesterone caproate.

MECHANISM OF ACTION

Progesterone acts through progesterone receptors but induces a decrease in the cytoplasmic oestrogen tissue receptors. It activates 17 hydroxy steroid dehydrogenase which converts oestradiol to less active
oestrone. Thus unopposed oestrogen and endometrial hyperplasia are inhibited.

PHARMACOLOGICAL ACTIONS

1. Endometrium – Pregesterone causes secretary phase and decidua formation, if the endometrium has been previously primed with oestrogen.

2. Vaginal epithelium and secretions – Progesterone prevents the cornification of vaginal epithelium and brings about increased glycogen deposition. Vaginal secretion becomes thick, viscid and scanty.

3. Mammary gland – it promotes acinar growth

4. Ovulation inhibition – If inhibits ovulation only in large doses and given during pre-ovulatory stage.

5. Metabolic actions – Progesterone rises body temperature. It has got a catabolic action.

6. Anti neoplastic action – Progestines are used in advanced cancer of breast and endometrium.

ABSORPTION, FATE AND EXCRETION

Majority of oral progestins are effective orally and they are metabolized in liver and are excreted through urine.

ADVERSE REACTIONS

Progestins in general are safe drugs. Derivatives of testosterone and 19 nortestosterone can cause nausea, breast discomfort, headache, fatigue, mental depression and very rarely liver damage. Nor ethisterone cause
weight gain acene, hirsutism due to androgenic actions. Progesterone can cause increase in the coagulability of blood, predisposing to intra vascular clotting with the resulting deep venous thrombosis and thrombo embolic disease.

**THERAPEUTIC USE IN DUB**

Progesterone for arrest of haemorrhage: In endometrial hyperplasia large doses of progestogens helps in arresting excessive bleeding.

Luteal phase treatment – When corpus luteum insufficiency has been diagnosed progesterone is given from 15\textsuperscript{th} – 25\textsuperscript{th} day. Whole cycle treatment – For endometrial hyperplasia progestogens are given for 5\textsuperscript{th} – 25\textsuperscript{th} day.

**CONTRA INDICATIONS**

Hypertension, active liver disease like jaundice, conditions like thrombophlebitis etc.

**COMBINED OESTROGEN AND PROGESTERONE THERAPY**
Combined oestrogen and progesterone cyclical therapy in the form of oral contraceptives is the most widely used and effective hormone therapy. This combination therapy has got the great advantage of correcting any abnormality in the menstrual cycle and of the production of regular cyclical bleeding as well as reducing the amount of menstrual blood loss. The combined pills contain ethinyl oestradiol or its 3 methyl either as the oestrogen and a progesterone either belonging to 19 nonestrosterone group or derived from progesterone.
MECHANISM OF ACTION

The combined preparations suppresses both FSH rise and LH peak and thus helps in regularizing the level of ovarian hormones in blood. Thus this help in correcting the imbalance in the secretion of helping in regularizing growth and shedding of endometrial tissue with normal ovarian and menstrual cycles.

PHARMACOLOGICAL ACTION

Endometrium - The full secerory activity of endometrial glands is achieved with 3-4 days of starting these pills. Later the endometrium becomes thin and hypoplastic.

Pituitary ovarian axis - Prolonged use of these pill for 3 cycles does not cause any structural change in the pituitary or ovary.

Metabolic effects - There may be hyperglycaemia, glucosuria & impaired glucose tolerence. Pill cause slight increase in the plasma lipids.

ABSORPTION, FATE AND EXCRETION

Oestrogens are quickly inactivate by converting to oestriol. Oestriol is conjugated in liver with glucoronic acid. The conjugated oestrogen is excreted party in bile and partly in urine. (Absorption fate & excretion of progestorgens are as described earlier).
ADVERSE REACTIONS

Gastrointestinal symptoms like nausea, anorexia and vomiting are common. Fluid retention, breast engorgement, mastalgia, chloasma and increase vaginal secretion are also seen. There is increased chance of deep vein thrombosis, cerebral thrombosis, and pulmonary embolism.

CONTRA INDICATION

1. History of liver disease or jaundice
2. Thromboembolic disorders
3. Epilepsy
4. Carcinoma of breast, cervix or uterus, hypertension, diabetes, severe allergy etc.

DOSHIC VARIATIONS

Vatika pradara

By observing the symptoms mentioned under vatika pradara it is found that there is a close relationship with impairement of autonomous nervous system. All types of gynecological disorders are caused by vitiation of vata. Among the pancha vayu vitiation of apana vayu is the basic cause of vatika pradara.
**Pita pradara**

In Ayurvedic concepts pita has close relationship with rakta. So in paithika pradara all the disorders of rakta like that of bleeding disorders, defect in cloating mechanism, impairment in tissue metabolism which will produce harmful metabolites.

**Kapha pradara**

According to the character of menstrual discharge in kapha pradara it may be due to some swelling from inflammations, certain types of polyps, structural deformity which may cause hypertrophy of the uterine musculature.

**Sannipathika**

All the above three conditions together will contribute the Sannipathika pradara.
ENVIRONMENTAL HAZARDS

Work is a major part of life. Virtually every patient has spent eight or more hours of every day and many months and years of life working. In their jobs people can be exposed to dangerous chemicals, hazardous physical agents, and emotional stress, and they suffer trauma. Any of these exposure can cause disease – sometimes immediately and sometimes after an interval of years or decades.

Environment is another constant factor in human life that also can cause disease. Air pollution, pesticides are examples of environmental agents that can cause illness and death. Tens of millions of people are exposed regularly to environmental toxins. They cause broad range of illness and these diseases can involve virtually every organ systems. Newer entities recognized only in recent years such as, stertility in women. Some of these diseases are acute, others are chronic. Some are manifest through obvious symptoms, where as others involve more subtle degrees of dysfunction.

Environmental diseases are underdiagnosed. Many are incorrectly attributed to other causes, because frequently these diseases are not distinct in their clinical presentations and can closely resemble chronic diseases caused by other factors.
The keys to properly diagnosing environmental diseases are

1. Obtaining an adequate history of environmental exposure for every patient.

2. Possessing basic knowledge about the pathogenesis and clinical presentations of the major types of environmental diseases.

3. Knowing to report suspected cases of such illness to public health authorities so that additional cases caused by the same exposure can either be recognized or prevented.

Four factors contribute most significantly to female health problems are personal lifestyle, environment, hereditary and medical care systems. It has been suggested that our medical care system contribute to only a very small way to our overall health. By contrast our personal life styles contribute most significantly, with diet being a major aspect.

At the turn of the century the leading causes of death were infectious diseases, but today most of the leading causes of death are largely attributed to specific life styles. Heart diseases, cancer, stroke accounts for two third of all deaths, one third of will die of coronary artery disease. Changes in the eating patterns parallel these disease trends. In lieu of the high fibre, low fat foods, once used, refined starches, sweets, saturated fats, and salt makeup a major portion of today’s typical diet. Of the 10 current leading causes of death 5 can be attributed to an unhealthy diet. In addition diet contributes greatly to hypertension, hypercholesterolemia, obesity which are associated with significant morbidity.
Anaemia is one of the most common manifestations of disease worldwide. Any blood disorder characterized by coagulation defects, or by excessive capillary fragility can cause endometrial haemorrhage in females. Anaemia is said to cause menorrhagia but is much more likely to be the effect. Hypertension when it is associated with arteriosclerosis in the vessels of the uterus.

Psychological upsets

Emotional and nervous disorders may cause excessive uterine bleeding rather than amenorrhea. Changes in environment, nervous tension, anxiety states, stress situations, ever work are examples of factors which are commonly to blame. These factors operate possibly through the endocrine system which is influenced by the hypothalamus, but more probably through the autonomic nervous system which controls the blood vessels supplying the pelvic organs. Active or passive congestion causes hypertrophy of the myometrium and endometrium so that the uterus can become from two to six times normal in size. Psychological factors are the commonest reason for patients perceiving bleeding as excessive or abnormal.

Environment tobacco smoke

Considerable evidence indicates that exposure to environmental tobacco smoke (ETS) ie passive smoking is harmful to the health of non-smokers. ETS consists of smoke that is generated while the cigerette is smoldering, as well as mainstream smoke that has been exhaled by the smoker. Seventy five percent or more of the total combustion product from a cigerette enters the air.
Some toxins such as ammonia, formaldehyde, and nitrosoamines are present in much higher concentrations in ETS than in main stream smoke.

Reproductive hazards of tobacco use are reduced fertility, premature birth, lower birth weight, spontaneous abortion, abruptio placentae, premature rupture of membranes, increased perinatal mortality. From the above mentioned health hazards reproductive system is deeply influenced by the environmental tobacco smoke either in gynaecological and obstetrical perpectives.

Ayurvedic classics also gives due importance to the environmental and dietetic habits of women which causes all the gynaecological diseases not only pradara. Acharya Charaka has included abnormalities of dietics in the causes of pradara. Dietic abnormalities can produce emaciation, excess consumption of wine can damage the liver thus produce pelvic congestion. Emaciation, pelvic congestion are causes of pradara.

Grief influences woman’s psychology, which in turn may influence production of ovarian hormones via hypothalamus – pituitary and ovarian pathway or else produce vasomotor disturbances, both these are causes of pradara.

People are often confused about the magnitude of the potential adverse health effects of exogenous physical and chemical agents. There is wide spread concern about the potential chronic or delayed effects of exposure to low levels of contaminants in air, water and food and hence patients frequently seek advice and information from their
health care professionals about the risk of disease associated with specific environmental occupational and iatrogenic exposures (Leigh et al., 1997).

Occupational exposures contribute a wide range of illness, including cancer and cardiovascular, cerebrovascular diseases, leading premature death (1992). Environmental protection agency estimates that more than 60,000 chemicals are currently used in United States, approximately 1500 are pesticides, and 5500 are food addictives that affect our water and food supplies (Michell, 1992). Industrial chemicals, production byproducts, and metals are commonly detected at hazardous waste sites. Physicians can help patients identify specific environmental and iatrogenic hazards that may cause adverse health effects educate them about the nature and magnitude of these risks, and encourage behaviours that reduce these risks.

Mechanism of toxicity

Exogenous chemicals are absorbed after ingestion, inhalation, or skin contact, then distributed to various organs. Chemicals are often metabolized by multiple pathways, to products that may be more or less toxic than the parent chemical. One or more of these products then interacts with the target macromolecules, resulting in a toxic effect. The site of toxicity is frequently the site where metabolism or excretion of toxic metabolites occurs.

Exposure

Absorption at portals of entry
1. This xenobiotics metabolism facilitate their transport in the blood stream by lipoproteins and penetration through lipid membranes.

2. Multiple pathways may be involved in metabolism of a chemical toxicant, between different species sexes and age groups.

3. Endogenous factors such as nutritional and hormonal status alter enzyme activity involved in xenobiotic metabolism.

4. Exogenous factors like chemicals, drugs, ethanol and stress can induce activities of xenobiotic metabolizing enzymes.
Common Environmental Exposures

Personal exposure

Tobacco

Tobacco use also increases the prevalence of peptic ulcers, smoking impairs healing of ulcers and increases the likelihood of recurrence. Smoking may also increase pyloric reflux and decrease bicarbonate secretion from the pancreas.

Alcohol abuse

Ethanol is the most widely used and abused agent throughout the world. The metabolism of ethanol is directly responsible for most of its toxic effects. In addition to its acute action as a central nervous system depressant, it can cause wide range of systemic effects. Ethanol can cause fatty changes, acute alcoholic hepatitis, cirrhosis. In reproductive system it causes testicular atrophy, spontaneous abortion etc. decreased fertility in both men and women.

Drug abuse

Drug abuse, addictions and overdose are serious public health problems. Commonly abused drugs can be classified as central nervous system stimulant, depressants, narcotics, etc.

Outdoor air pollution

Air pollution is a serious problem in many industrialized countries. The major sources of ambient air pollutants are
1. Combustion of fossil fuels: There are divided into mobile sources such as motor vehicles, stationary sources such as power plant, factories, and other sources like fire places. Tailpipe emissions from motor vehicles are a complex mixture of carbon monoxide, oxides of nitrogen, hydrocarbons, diesel exhaust particles, and other particulates including lead oxide from tetraethyl lead contained in leaded gasoline.

Waste incinarators, Industry smetters

These point sources release acid aerosols, metals and organic compounds, that may be hazardous for human health.

Lungs are the major targets of common outdoor air pollutants.

Ozone: Ozone is a major component of smog that accompanies, crush, chest discomfort and inflammation in lungs.
Nitrogen dioxide

Nitrogen dioxide dissolves in water in the air way to form nitric acid and nitrous acid which damage the airway epithelial lining.

Acid aerosols

Primary combustion products of fossil fuels are emitted by tall smoke stacks and transported to air. In the atmosphere sulphur and nitrogen dioxide are oxidized to sulphuric acid and nitric acid. There are irritants to air way epithelium and alter mucoceliary action

Indoor air pollution

Rising every costs have led to increased insulation and decreased ventilation of homes that elevates the level of indoor air pollutants. They are gas, cooking stoves, furnaces, wood stoves, construction material, furniture, allergens associated with pets, dust, mites, fungal spores and bacteria.

Carbon monoxide: This is a byproduct of combustion produced with burning oil, coal, wood and natural gas. It can cause aggravation in myocardial ischemia. Exposure can lead to significant toxicity of the central nervous system and heart. It convert 50 per cent of the body’s hemoglobin to Carboxy-haemoglobin, it is ineffective for delivering oxygen resulting in some body parts not receiving oxygen needed.

Nitrogen dioxide: Gas stove and kerosene space heaters can raise indoor levels of, causing lung infections.
Wood smoke: This is a complex mixture of nitrogen oxides. High concentration of wood smoke in poorly ventilated houses increases the incidence of lung infections.

Industrial exposure

The spectrum of human diseases associated with occupational exposure affects almost all organ system. They include generative changes in nervous system, reproductive dysfunction etc.

Volatile organic compounds

Aliphatic hydrocarbons: (Methane, ethane, ethylene, acetylene) These compounds are the most widely used industrial solvents and dry cleaning agents. All of these chemicals are readily absorbed through the lungs, skin, and gastrointestinal tract.

Aromatic hydrocarbons

Benzene, benzopyrene, studied as a carcinogen, are oil spill pollutants, or atmospheric deposition. They are known carcinogens and are linked to other health problems.

Petroleum products

Gas, kerosene, mineral oil, are highly volatile. Inhalation of these causes dizziness, incoordination etc.
Aromatic hydrocarbons

Benzene, xylene are widely used solvents in the rubber and shoe industries and in printing and paper coating. It cause aplastic anaemia.

Agricultural hazards

Although agricultural productivity has been improved by the use of fertilizers and pesticides, they cause diseases in those exposed to them, pesticides residues are found on foods and they contaminate soil and water supplies. There is considerable adverse health effects of these persistent pesticides and their metabolites, especially concerning their relationship to breast cancer and to reproductive abnormalities.

Agricultural pesticides are divided into five categories depending on the target pest, insecticides, herbicides, fungicides, rodenticides and fumigants. All pesticides are toxic to some plants, farm animals. Fungicides are characterized as moderately toxic. Acute toxicity of insecticides for mammals ranges from low to high. Fumigants used to eliminate insects from enclosed spaces and rodenticides are highly toxic. There are all indirectly or directly effect human health, mainly to the reproductive organs.

Warfarin the main rodenticide inhibits the synthesis of biologically active forms of the Vitamin K dependent clotting factors II, VII, IX, X as well as the regulatory factors protein C, protein S, and protein Z. They require carboxylation of their glutamic acid recidues to allow the coagulation factors to bind to phospholipid surfaces inside blood vessels, on the vascular endothelium. The coagulation factors are produced but have decreased functionality due to under carboxylation. The end, the warfarin
diminish blood clotting in the patient. Pesticides contaminants land and water when it escapes from production site and storage tanks. They cause air pollution, by suspending in air as particles, carried by wind.

In the united states pesticides were found to pollute every stream of water and 90 per cent wells. The residues also been found in rain and ground water.

Many food crops including fruits and vegetables contain pesticide residues. A study published by us national research council 1993, determined that the exposure to pesticides is through diet.

US national academy of science estimates that 4000, and 20,000 cases of cancer are caused by pesticide residue in food.

Some scientists think that pesticides in uterus may have negative effects on fetus that may manifest as problems as growth and behavioural disorders or resistance to pesticides toxicity later in life.

Natural toxins

These mycotoxins and phytotoxins may contaminate food. Animal toxins can be ingested by eating fish, snails, or molluscs. Mycotoxins is a toxin produced by an organism of the fungus kinddom. Some toxins cause identifiable diseases or health problems, some weaken immune system. Aflatoxin are produced by aspergillus. Species from ground nuts, - Most toxic Aflatoxin B$_1$, is a potent carcinogen. Patulin, is a fungal species and is found in fruits, vegetables, cereals and other foods. It is carcinogenic and is reported to damage the immune system and nervous system. Fusarium which infect the grain of developing cereals like wheat and maize.
The major sugar substitutes for sweeteners are succharin, aspartame, neotame etc. At high doses saccharin causes bladder cancer and aspartame causing brain tumour. They contain aflatoxic B\textsubscript{1} is produced by fungi that contaminate peanuts, causing liver cancer.

All of the toxins have an adverse effect on the vital organs of the body like heart, lungs and liver. The blood purification does not take place when the lung tissues are damaged and also arterial defects may lead to bleeding disorders. Liver is the main organ of metabolism, and the hormones are metabolized there are impaired due to the damage of hepatic tissues causing hormones imbalance leading to bleeding.

Iatrogenic factors

Some factors induced by physician causes problems inpatients. In Pradara one of the commonest causes are exogenous estrogens, administered by various routes for a variety of conditions such as pruritis vulvae, climatic symptoms, and even for the control of uterine bleeding. Other forms of uterine bleeding can also complicate the taking of oral contraceptives containing oestrogen, progesterone or both.

Exogenous estrogens and oral contraceptives

Estrogens and oral contraceptives are discussed separately because (1) oestrogens for post menopausal syndrome may be given alone and are usually natural estrogens and (2) oral contraceptives contain synthetic oestrogen, always given with progesterone.

The adverse effect of estrogen therapy
1. Endometrial carcinoma: Unopposed oestrogen therapy increase the risk of endometrial carcinoma 3 to 5 times increased after the use of 5 years.

2. Breast carcinoma: Some studies continue to point to increased risk of this form of cancer with the use of unopposed oestrogen.

3. Cardio vascular disease: Myocardial infarction and stroke are among the leading causes of death in post menopausal women. It also causes bleeding disorders in uterine vessels.

Oral contraceptives

These drugs nearly always contain synthetic estradiol and variable amounts of progesterone. They cause

Breast carcinoma, endometrial dysfunction causing bleeding, cervical cancer, ovarian cancer, hypertension, cardiovascular diseases, hepatic adenoma, etc.

Unethical use of oral contraceptives may derange the hormone balance causing endometrial bleeding which may resulting into dysfunctional uterine bleeding.
AYURVEDIC ETIOLOGY

Abnormal\textsuperscript{1} dietics and mode of life abnormalities of arthava and both the bija (both sperm and ovum) and in the absence of apparent cause the diseases are said to arise due to curses of God are the causative factors of all the gynaecological disorders as said by Charaka.

Both\textsuperscript{2,3} vagbhatas accepting abnormalities of arthava and bija as well as abnormal diet, are causes of the gynaecological disorders.

Considering descriptions of all the classics collectively, following etiological factors emerge out.

1. Mithyacara

The heading includes midhya ahara (abnormal diet) midhya vihara (abnormal mode of life) both. Various environmental factors operating either during embryonic life of the girl (congenital abnormalities) or at a later life also come under this heading.

(a) Abnormal diet

Abnormality in diet refers to excessive mal or inadequate diet along with non-congenial, unwholesome, unhygienic and incompatible food. The diet influences dosas and dushyas of body, main causes of all the disorders.
(b) Abnormal mode of life

ABNORMALITY IN LIFE, I.E., SLEEP DISTURBANCES, UNTIMELY FOOD, OVER-EATING, LACK OF REST COMES UNDER THIS TITLE.

2. Pradushta arthava

The word arthava refers to female sex hormones, ovum and menstrual blood. Ovum is already described separately, menstrual blood is a result of endometrial changes brought about by hormones and reflects the status of reproductive system as well as hormones, thus arthava here refers to hormones. Ovarian hormones are causative factors for a good number of gynaecological disorders.

Formation of Raja

From Rasa, the raktha named as raja is formed. Raktha reaching uterus and coming out for three days in every month is called arthava. Susrutha, Vagbhata II, Dalhana, and Chakrapani has the opinion as raja is formed from ahara rasa. During the process of formation of arthava is soumya due to influence of rasa, while at the time of its excretion due to specific changes it assumes agneya character. The raja is considered as formed from rasadhathu, because it is mainly plasma which supplies nourishment to pituitary and ovary and carries hormones to ovary, ovarian hormones to endometrium. So the diet, and mode of life influence the formation of ahara rasa, if there is any abnormal production, or lacking the qualities of ahara rasa that may cause the formation of raktha impaired, thus production of hormones which may indirectly cause abnormal functioning of
endometrium. So dietic habits and environmental factors influence the production of hormones.

Mode\(^5\) of living during menstruation

From the day of onset of menstruation the lady should observe chastity, avoid day sleeping, fast racing, excessive exercise, laughing, listening to so many types of topics. She should eat sali rice with ghee and milk. Do not do panchakarmas, and take less quantity of food. Avoid punchant, hot, and salty substance. She should concentrate on thinking good or auspicious things. On fourth day after taking bath, with new garments, with garlanding with flowers, with the enchantation of religious hymns, should first see her husband.

These are the regimes described in the classics during the time of menstruation. By this, she may be getting proper rest, good thoughts, satwika aharas the ovum released will be of good qualities and the hormones will be balancing and no abnormalities in bleeding may occur. Now a days there regime is not following, due to the day to day work of the females, hard work, family matters she will be thoughtful about the family that will alter her hormonal status, causing so many diseases and the important among them is pradara.
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