



**CONCEPTUAL UNDERSTANDING OF ETIOPATHOGENESIS OF RENAL OEDEMA IN  
AYURVEDA W.S.R TO SHOTHA**

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**ABSTRACT**

Oedema can be caused by a variety of factors, including hypoproteinemia, in which a lowered concentration of plasma proteins decreases the osmotic pressure, thereby permitting passage of abnormal amounts of fluid out of the blood vessels and into the tissue spaces. Hence the oedema occurs when the small blood vessels (capillaries) leak fluid into the surrounding body tissue. This may happen as a result of increased pressure in (or damage to) the capillaries. The oedema may be unilateral, bilateral, pitting and non-pitting or it may be localized or generalized etc. Depending upon these features the co-relation can be made in understanding the Etiopathogenesis of Shotha with special reference to Renal Oedema. *Shotha* explained in the classics in different ways as a *lakshana* in many diseases and sometimes as a disease. In such conditions it should be studied as a separate disease only and not as considering as a *lakshana*. In *ayurvedic* classics we get the description of 3 different words as *Shotha*, *Shopha* and *Shwayathu*, all are the synonyms of *Shotha*. The *lakshanas* of *Shotha* that are explained in the classics can be correlated with the Oedema.

**KEYWORDS:** Renal oedema, *Shotha*, *Kleda*, Hydrostatic pressure, Renin Angiotensin.

**INTRODUCTION**

Oedema is the chronic effusion of fluid into the skin and subcutaneous tissues or into a serous cavity. 'Anasarca' is a form of generalized oedema and is a very constant feature of some forms of cardiac diseases. Generalized oedema is due to an increase in the volume of extracellular fluid. This is brought about by excessive renal tubular reabsorption of sodium and water; the mechanism of this reabsorption is complex and the renin-angiotensin-aldosterone system is only one of the factors involved. Hence, Oedema is a clinical condition which may manifest either by a local cause with a minimal tissue involvement as seen in an insect bite, thrombosed vein or as a consequence of multisystem tissue injury. The accumulation of fluid in the extra vascular space which causes oedema is determined by the relationship between the hydrostatic and oncotic pressures in the capillaries and the interstitial tissue.

Contemporary theories of oedema formation are often based on the idea that "effective" blood volume is reduced, and that sodium retention and oedema are a

result of the kidney responding, as in haemorrhage, to a perception by receptors in the circulation that blood volume is inadequate. This idea has enhanced understanding of the pathophysiology of such conditions as renal failure, cardiac failure and cirrhosis, but has obscured the fact that blood volume is almost always increased in oedematous states. Evidence is presented that an increase in renal venous pressure can cause sodium retention by a direct action on the kidney: a rise in venous pressure could thereby initiate a vicious circle by causing sodium retention, expansion of plasma volume, and further increase in venous pressure. The incidence of renal oedema is seen more in the age group of 30-60 yrs which is more than 55% of the total age group and almost 3 males are affected for every one female, according to the study.

Renal oedema is general in its distribution from the beginning, occurring in the legs and eyelids at the same time; though it is probable that the oedema around the eyes on rising in the morning first attracts the attention of the patient.

Vyadhi is the effect of a *nidana* or the conglomeration of *nidana*. *Nidana* reacts with the *shareerika doshas* and bring about the changes in the composition of the body. When there is marked change in the biological composition and functioning of the *doshas* that forms the disease or the *vyadhi*. The phenomena that explain all these processes from the affliction of *nidana* up till the manifestation of the disease is called as Pathogenesis or *Samprapti*.

According to *acharyas*, pain and discomfort frequently affects one's normal routine functions. *Shotha* is one such manifestation which may interfere one's normal health, especially when its origin is from *Shira*, *Hridaya* and *Basti*.

On the contrary the Renal oedema resembles with the *samprapti* and *lakshanas* which are explained in classics about *Shotha*.

To understand the *samprapti* of the disease the physiological backgrounds are essential, so that, upon which the changes can be understood and explained. *Shotha* is a disease where in *shareeraja jala dhatu* is unduly retained in the body. Hence it is necessary to understand the mechanism of fluid regulation in the body.

#### Fluid – Fate - according to Ayurveda

The body is primarily made of *jaleeya dhatu*, so is accepted in the present science too. '*Deham rasajo ambubhavo*'- by saying *Charaka* gave a message that major constitution of the body is *Ambu – Jaleeya dhatu*.<sup>[1]</sup> The Modern science explains around 60 % of the bodily content is fluid.

There are two sources of fluid for the *shareera*. One is *Ahara* i.e., one among the *chaturvidha ahara* is *jala*. The second one is *kleda*, formed inside the body during the process of digestion and metabolism.<sup>[2,3,4,5]</sup>

The terms such as *rasakleda*, *shonita kleda*, *mamsa kleda*, *dhatu kleda*, *sroto kleda* etc clearly suggests the formation of the *kleda* at different tissue level in the body.

'*Aapah kledah*'- Generally the term *kleda* refers to *Jaleeya dhatu*. (*Kledayati aardreekaroti – prakledana aardrabhavakara*).<sup>[6,7]</sup>

*Shareera kleda* refers to *jaleeya dhatu* of *shareera*. The term *kleda* is used in different contexts having different meaning with a common entity everywhere i.e., *Jaleeya dhatu*.

The essential components of the body such as *rasadhatu*, *raktadhatu* and other *dravadhatu* that are circulated by the *Vyana vata* from the *Hrudaya*. The functions of *Vyana vata* is a continuous one till the death. All the essentials of the *shareera dhatu* are carried by the *drava*

*dhatu* continuously, there by maintains the normal functions of each and every tissues. In this way *shareeraja jaladhatu* are circulating and fulfilling the needs of each and every tissue and hence maintains the normal functions of the body.

Body has three major routes of elimination of wastes and eliminates three forms of waste products. First one is related with the elimination of the wastes through the air i.e., through *pranavata*. *Charaka* opines that the *pranavata* should be protected at most when compared to the other four types of *Vata*. The life – '*Jeevana*' depends on the *Pranavata*. Among the *dhatu*s, the *restra karma* of *rakta* is *Jeevana*.<sup>[8]</sup> *Jeevana* refers to *Prana dharana* or *prana anuvartana*. *Rakta* is the abode for *pranavata*; hence both of them have the same functions, without which the life is impossible or the life does not exist.

Other major mode of elimination is as solid waste i.e., through the *pureesha*. The solid waste of the food is called *pureesha*. The gross food is digested with the help of *samanavata*, *pachakapitta* and *kledaka kapha*. Later the solid waste particles of the gross food are eliminated with the help of *apanavata*. Some amount of liquid is also eliminated in the process.

Finally, the bodily wastes are eliminated by means of water i.e., through *mootra* and *sweda*. It is mentioned that there are two sources of fluid in the body. They are the *aharaja* and *shareeragata kleda*.

'*Kittam annasya vinmootram*' if the amount of fluid is taken in excess is also absorbed in to the *shareera*. It is not just passed out along with *pureesha*. Though the *moola* of *ambuvaha srotas* is *talv* and *kloma*, it is also related with *kosta*.<sup>[9]</sup> *Ambuvaha srotas* is in relation with *pachaka pitta* and *samana vata*.

*Acharya Sushruta* has mentioned that, the *ahara* after *pachana* forms two parts. *Sara bhaga* and the *kitta bhaga*. The *Sara bhaga* forms the *rasa dhatu* and the *kitta bhaga* remains in the *dravavastha*. Thus the *drava mala* reaches the *pakwashaya* where it undergoes the *shoshana*. The hard part of it is the *purisha* and the liquid part is the *mutra* which reaches the *basti* through the *mutravahini siras*. In the same way we get the description explained by *Acharya Sushruta*, the *ahara pachana* occurs by the *pachaka pitta* and attains the *sarabhaga* and *kittabhaga*. *Sara bhaga* attains the *rasadi dhatu*s and *kitta bhaga* forms the *mala* and *mutra*.

The *Chaturvidha annapana* undergoes the *pachana* by the action of *pachaka pitta*, enters to the *pakwashaya* where it gets divided in to the *drava bhaga* and the *Ghana bhaga*. The *Ghana bhaga* is the *purisha* and the *drava bhaga* is the *malakhyia kleda* and is carried to the *basti* from the *pakwashaya* to the *basti*. The *annamala* and the *dhatumala* are also added to the *malarupa kleda* by the *dhatwagni vyapara*. The *malarupa kleda* which contains the *annamala* and the

*dhatumala* enters the *vankshana* and *nishyandana karma* takes place. Thus the *mutra* is formed and is carried to the *basti* and is eliminated out through the *medra* by the action of *Apana vayu*.

The amount of fluid is taken in to the system through the *ambuvaha srotas*. This fluid reaches the *hridaya* along with the *rasa* and *dosha*, from which it gets circulated along with the *rasa raktadi drava dhatu* with the help of the *Vyana vata*.<sup>[10]</sup>

The fluid is formed in the body during the metabolism in *dhatu* is considered as *Kleda*. Excess of *kleda* should be eliminated from the system to maintain the homeostasis. The *ambuvaha srotas* is in relation with the *basti* also. *Basti* is the *vishrama stana* for the *Ambuvaha srotas*. *Basti* is considered to be the *mootrashaya* and *maladhara* i.e., area where the *mootra* is formed and collected. *Basti avayava* is in relation with *Ambuvaha srotas* and *sarva srotas* is just like multiple small water channels that are joined to form an ocean. Here *basti* is in relation with the *sarvasrotas* through the *udakavaha srotas*. All the *moortimanta bhava* in an individual are nothing but variants of *srotas* itself.<sup>[11]</sup>

It means all the *paramanu* in the *shareera* are nothing but the variables of *srotas*. In this way all the *dhatu*, *dhatu avyava* are ultimately connected with the *basti* through the *ambuvaha srotas*. In this way *shareeragata kleda* ultimately reaches the *basti*. *Apanavata* eliminates the *mootra* from the *basti*.

*Mootra* is one among the *trividha mala* and it is *jaleeyaroppi mala*. The function of *mootra* are *kledavahana*, *basti poorana*, *vikledana*. *Kleda vahanam* refers to *kleda nirvahana* i.e it regulates the *kleda*. The *jaleeya dhatu (kleda)* that is required for the *shareera* is retained and the excess is eliminated out of the body in the form of *mootra*. *Kleda vahanam* also refers to *kledasya bahir nirgamanam*. It means excess of *kleda* is eliminated out. *Basti poorana* refers to the formed *mootra* is collected in the *basti*. *Vikledana* refers to *kleda vivekajam*.<sup>[12]</sup> *Kleda vivekajam* is the process where in surplus *kleda* is removed by maintaining the essential amount.

From this it is understood that the unwanted portion of *kleda* is eliminated as *mootra* in *bastiavayava*. *Sweda* is another *jaleeyaroppi mala* which is excreted over the *twacha*. The function of *sweda* is mainly *kleda vidhruti*. The root of the word *vidhruti* is *vidru* and it gives the meaning as clean or devoid of impurity. *Vidhruti* refers to organized or arranged. *Kleda vidhruti* is a process where the *kleda* is organized in the body. Excess *kleda* is removed and the required amount is always maintained. *Arunadatta* opines that in the conditions of *kleda abhava*, *sweda shoshana* takes place in the body. From this, it gets clear that *sweda* also regulates the *jaleeya dhatu* in the *shareera*.

### Fluid regulation<sup>[13]</sup>

The explanation in the current science regarding the fluid regulation is also the same. The ingested fluid is taken in to the system, reaches the heart becomes the part of the blood. Blood carries the essential nutrients as well as oxygen to the each and every cell. Blood reaches the cells by the cardiac activity. At cells, metabolism takes place producing the energy, metabolic wastes and the fluid. The metabolic wastes enter the circulation, reach the kidney and get excreted out. Sweat excretes some amount of fluid.

Even the exhaled air also excretes some amount of fluid. All these excretion are regulated from the different neuro-endocrinal system. In *Ayurveda* all the essential functions are regulated by the *Vatadosha* and probably its functions are compared to neuro-endocrinal system. The normal fluid homeostasis encompasses maintenance of vessel wall integrity as well as intravascular pressure and osmolarity within certain physiologic ranges. Changes in vascular volume, pressure or protein content or alterations in endothelial function, all affect the net movement of water across the vascular wall. Such water extravasation into the interstitial spaces is called the oedema and has different manifestations depending on its location.

### Etiopathogenesis of *Shotha* According to *Ayurveda*

The term *shotha* refers to *swayathu* where '*Utsedha*' is the *pratyatma lakshana*. *Uthseda* refers to swelling. *Shotha* is primarily because of *kapha prakopa* in the *shareera*. *Kapha* is responsible for *kledana* in *shareera*. '*Aapah kledah*' - Generally the term *kleda* refers to *Jaleeya dhatu*. Accumulation of *jaleeya dhatu* results in *uthseda* in the *shareera*. From the etiological factors it is learnt that different diseases are the *pradhanika* or *utpadaka* or *vyadhi hetu* of *Shotha*. All the *aharaja* and *viharaja nidana* are either *vyanjaka* or *dosha* or *vyabhicari hetu*. A person suffering from the *vyadhi* if he is indulging in the *aharaja* and *viharaja nidana* there will be further *dusti* of the *dosha* in the *shareera*.<sup>[14]</sup>

*Dusta tridosha* obstructs *vyanavata* in the *shareera* especially *ambuvaha*, *moortavaha* and *swedavaha srotas*. *Tridosha* are moving in all the *srotas* with the help of *vyanavata* along with the *rasa rakta adi dravadhatu* to maintain the respective functions of the *shareera dhatu* and *avyava*. Once the *dusta dosha* obstructs the *vyanavata* at *ambuvaha*, *mootravaha* and *swedavaha srotas vata* gets *prakupita* and brings the other *dosha* along with *rasa raktadi drava dhatu* to the *bahya sira*. *Bahya sira* refers to *agambheera sira* i.e., *sira* related with *twak mamsa pradesha*. In this way *dusta dosha* and the *shareeraja kledadi drava dhatu* reaches the *twak mamsantara pradesha* and vitiates the *sthanika dosha*, results in *uthseda* i.e., *Shotha vyadhi*.

After *sthana samsraya* at *twak mamsantara pradesha*, *dosha* vitiates the *sthanika dosha* and *srotas* causing *sirayama* i.e., dilatation of *srotas*. *Sirayama* is the

*poorvaroopa* of *shotha*. Because of *sirayama dravadhatu* leaks out of the *srotas* and accumulates in *twak mamsantara pradesha*. *Sira tanutwa* is the *samanya lakshana* of the *shotha*, where in *sirayama* ultimately results in *sira tanutwa* i.e, thinning of *sira*.<sup>[15]</sup>

In all the types of *shotha Tridosha* are invariably involved. Hence *shotha* is considered as *mahagada* and it is mentioned in the beginning of the chapter itself. Depending on the *doshadikyata* and the site of affliction it is being classified.

If *Kapha doshadikyata* is present in an individual usually *shotha* is observed in *urdvakaya*. If *Pitta doshadikyata* is present in an individual then the *shotha* is observed in the *madhya kaya*. If *Vata doshadikyata* is present in an individual then the *shotha* is observed in *adhokaya*. If

*doshastahanasamshraya* is in *sarvanga*, manifestation is *sarvadehaja* and *sthanasamshraya* in *ekadeshaja* the manifestation is in *ekadesha*.

#### Samprapti Ghataka

- *Dosha – Vata- vyana and Samana vataPitta – Pachaka pitta Kapha – avalambaka*
- *Dushya- Rasa, Rakta, Ambu*
- *Agni- Jaatharagni and Dhatwagni*
- *Ama- Jaatharagnijanya and Dhatwagnijanya*
- *Udbhavasthana- Koshta*
- *Sancharasthana- sarvashareera*
- *Adhithana- Twak mamsashraya*
- *Srotas- Rasa, Rakta, Udaka and Swedavaha srotas*
- *Srotodushti prakara- Sanga, Vimargagamana*

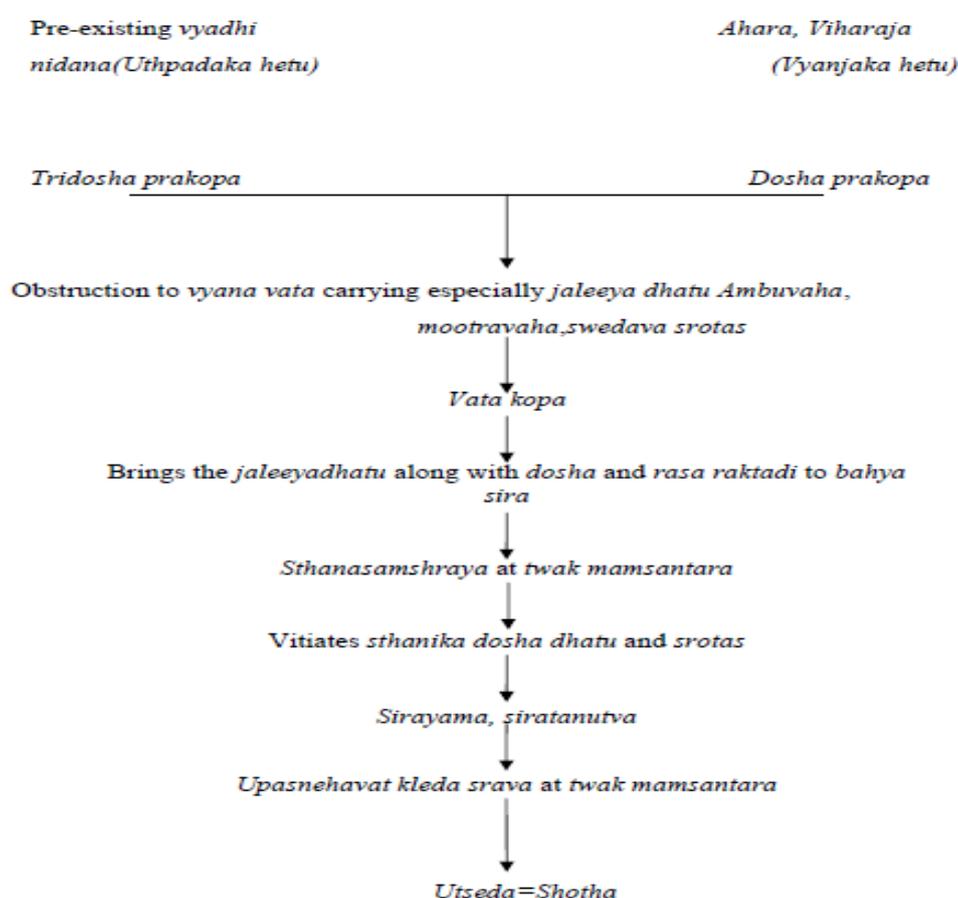


Figure 1: Flow chart of Shotha Samprapti in Ayurveda.

#### Pathophysiology of Renal Oedema

Generalized oedema occurs in certain diseases of renal origin such as in nephrotic syndrome, some types of glomerulonephritis, and in renal failure due to acutetubular injury.

Oedema in nephrotic syndrome-heavy proteinuria leads to hypoalbuminaemia causing decreased plasma oncotic pressure resulting in severe generalized oedema. The nephrotic oedema is classically more severe and marked

and is present in the subcutaneous tissues as well as in the visceral organs. The affected organ is enlarged and heavy with tense capsule. Oedema in Glomerulonephritis- it is due to excessive reabsorption of sodium and water in the renal tubules via renin-angiotensin-aldosterone mechanism. The protein content of oedema fluid in glomerulonephritis is quite low (less than 0.5g/dl). The nephritic oedema is usually mild as compared to nephrotic oedema and begins in the loose tissues such as on the face around eyes, ankles and

genitalia. Oedema in these conditions is usually not affected by gravity. Oedema in acute tubular injury – acute tubular injury followed by shock or toxic chemicals results in gross oedema of the body. Oedema is caused by mechanisms that interfere with normal fluid balance of plasma, interstitial fluid and lymph flow. The following six mechanisms may be operating singly or in combination to produce oedema:

1. Decreased plasma oncotic pressure.
2. Increased capillary hydrostatic pressure.
3. Lymphatic obstruction.
4. Tissue factors.
5. Increased capillary permeability.
6. Sodium and water retention

### 1. Decreased plasma oncotic pressure

A fall in the total plasma protein level results in lowering of plasma oncotic pressure thus it can no longer counteract the effect of hydrostatic pressure of blood, this results in increased outward movement of fluid from the capillary wall and decreased inward movement of fluid from the interstitial space causing oedema. Hypoproteinemia usually produces generalized oedema.

### 2. Increased capillary hydrostatic pressure

A rise in hydrostatic pressure at the venular end of the capillary which is normally low (12mm Hg) to a level of fluid at the venular end consequently leading to oedema.

### 3. Lymphatic obstruction

Obstruction to the outflow of the lymphatic channels causes localized oedema.

### 4. Tissue factors

The tissue factors are normally quite small and insignificant and they cause oedema in combination with other mechanisms.

### 5. Increased capillary permeability

The capillary endothelium is injured by various 'capillary poisons' thus the capillary permeability of plasma protein is enhanced due to development of gaps between the endothelial cells, this in turn causes reduced plasma oncotic pressure and elevated interstitial oncotic pressure which consequently produces oedema.

### 6. Sodium and water retention

#### Sodium and water retention is due to

- Intrinsic renal mechanism as occurs in hypovolaemia.
- Extra renal mechanism involving the secretion of aldosterone by the renin-angiotensin system.
- ADH mechanism, this hormone is stimulated by increased concentration of sodium in the plasma and hypovolaemia. Retention of sodium in turn leads to retention of water.

## DISCUSSION

The *ahara* after *pachana* forms two parts. *Sara bhaga*

and the *kitta bhaga*. The *Sara bhaga* forms the *rasa dhatu* and the *kitta bhaga* remains in the *dravavastha*. Thus the *drava mala* reaches the *pakwashaya* where it undergoes the *shoshana*. The hard part of it is the *purisha* and the liquid part is the *mutra* which reaches the *basti* through the *mutravahini siras*.

*Mutra* is one among the *trividha mala* and it is *jaleeyarooopi mala*. The function of *mootra* are *kledavahana*, *basti poorana*, *vikledana*. *Kleda vahanam* refers to *kleda nirvahana* i.e it regulates the *kleda*. The *jaleeya dhatu (kleda)* that is required for the *shareera* is retained and the excess is eliminated out of the body in the form of *mootra*. *Kleda vahanam* also refers to *kledasya bahir nirgamanam*. It means excess of *kleda* is eliminated out. *Basti poorana* refers to the formed *mootra* is collected in the *basti*. *Vikledana* refers to *kleda vivekajam*. *Kleda vivekajam* is the process where in surplus *kleda* is removed by maintaining the essential amount, from this it is understood that the unwanted portion of *kleda* is eliminated as *mootra* in *basti avayava*.

### Renal mechanism for regulation of blood pressure

The kidneys play an important role in the long term regulation of arterial blood pressure. When blood pressure alters slowly in several days/months/years, this neural mechanism adapts to the altered pressure and the pressure anymore. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called as long term regulation.<sup>[16]</sup>

### Kidneys regulate arterial blood pressure by two ways

1. By regulation of ECF volume.
2. Through renin-angiotensin mechanism.

### By regulation of Extra Cellular Fluid volume<sup>[17]</sup>

When the blood pressure increases, kidneys excrete large amounts of water and salt, particularly sodium by means of pressure diuresis and pressure natriuresis. Pressure natriuresis is the excretion of large quantity of water in urine because of increased blood pressure. Even a slight increase in blood pressure doubles the water excretion of large quantity of sodium in urine. Because of diuresis and natriuresis, there is decrease in the ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When the blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output resulting in restoration of blood pressure.

### Through renin-angiotensin mechanism

Secretion of renin- the juxta glomerular cells secretes renin. Along with angiotensins, renin forms the renin-angiotensin system which is a hormone system that plays an important role in the maintenance of blood pressure.

**Renin angiotensin aldosterone mechanism<sup>[18]</sup>**

In the kidneys, a fluid that resembles plasma is filtered through the glomerular capillaries into the renal tubules (glomerular filtration). As this glomerular filtrate passes down the tubules, its volume is reduced and its composition altered by the processes of tubular reabsorption (removal of water and solutes from the tubular fluid) and tubular secretion (secretion of solutes into tubular fluid) to form the urine that enters the renal pelvis. The composition of the urine can be varied, and many homeostatic regulatory mechanisms minimize or prevent changes in the composition of the extracellular fluid by changing the amount of water and various specific solutes in the urine. From the renal pelvis, the urine passes to the bladder and is expelled to the exterior by the process of urination or micturition.

The kidneys are also endocrine organs, making kinins, secreting renin and erythropoietin.

**The Renin Angiotensin System**

Renin- the rise in the blood pressure produced by injection of kidney extracts is due to renin, an acid protease secreted by the kidneys in to the blood stream. This enzyme acts in concert with angiotensin converting enzyme to form renin angiotensin 2. It is a glycoprotein. Like other hormones, renin is synthesized as a large prohormone in the body.

**Stimulants for Renin secretion****Secretion of renin is stimulated by four factors**

1. Fall in arterial blood pressure.
2. Reduction in the ECF volume.
3. Increased sympathetic activity.
4. Decreased load of sodium and chloride in macula.

Angiotensin- Circulating angiotensinogen is found in the

alpha 2 globulin fraction of the plasma. It is synthesized in the liver and its circulating level is increased by glucocorticoids, thyroid hormones, estrogens, several cytokinins and angiotensin 2.

**Renin – Angiotensin system**

When renin is released in to the blood, it acts on a specific plasma protein called angiotensinogen or renin substrate. It is the alpha 2 globulin. By the activity of renin, the angiotensinogen is converted into decapeptide called angiotensin 1. Angiotensin 1 is converted in to angiotensin 2 which is an octapeptide by the activity of angiotensin converting enzyme (ACE) secreted from lungs.

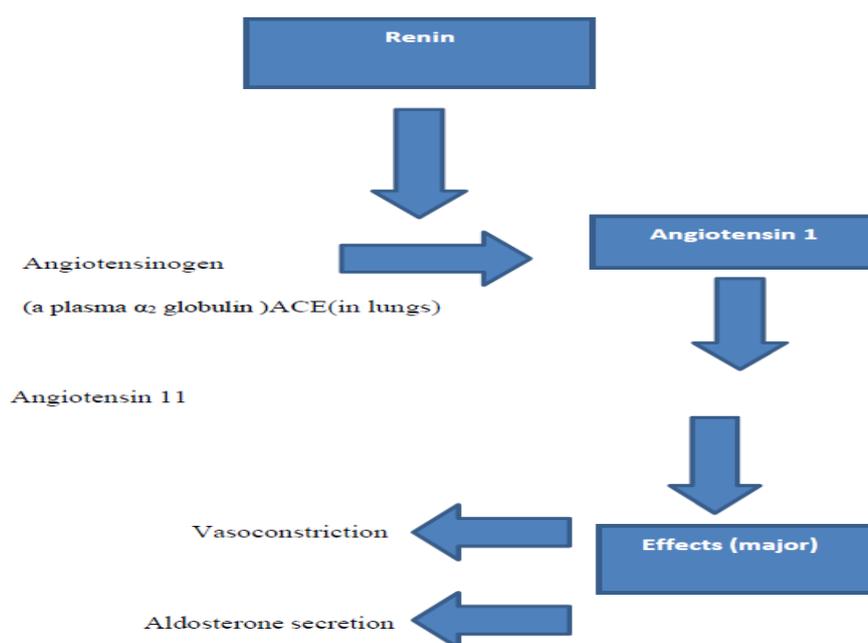
Angiotensin 2 has a short half-life of about 1-2minutes. Then it is rapidly degraded into a heptapeptide called angiotensin 3 by angiotensinases which are present in the RBC's and vascular beds in many tissues. Angiotensin 3 is converted in to angiotensin4 which is hexapeptide.

**Actions of Angiotensins<sup>[19]</sup>**

Angiotensin 1- it is physiologically inactive and serves only as the precursor of angiotensin 2

Angiotensin 2- it is the most active form and its actions on kidney are,

- 1) Angiotensin 2 regulates glomerular filtration by two ways-
  - It constricts the efferent arteriole which causes decrease in filtration after an initial increase.
  - It contracts the glomerular mesangial cells leading to decrease in surface area of glomerular capillaries and filtration.
- 2) It increases sodium reabsorption from renal tubules. This action is more predominant on proximal tubules.



**Figure 2: Flow chart of formation of Oedema.**

**CONCLUSION**

The *kleda* present in the body mainly in the *rasa, raktadi dhatus*. The *kleda* not only in the *dhatus* but it is also present in small amount in the *purisha*. The *prasadabhaga* of the *ahara* gets converted in to the *dhatus* whereas the *kittabhaga* in to the *mutra* and *purisha*. Hence the *dravaamsha* i.e., the *kleda* of the *kittabhaga* of *ahara* forms the *mutra* after reaching the *basti*.

According to the analysis of Etiopathogenesis of clinical features explained in the classics, it can be concluded that among the different types of *Shotha* according to the *doshas* the *Kaphaja Shotha* can be compared with the features of the Renal Oedema, depending upon the clinical features seen in the patients and the features mentioned in the classics.

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