Prameha: The characteristics of abnormal urine in comparison to the modern perspectives.

Abstract

Prameha, which is a grave disease of urinary system, consists of various pathological conditions caused by different etiological factors. On the basis of pathophysiology, it is divided into three categories namely: Kaphaja prameha, Pittaja prameha and Vataja prameha. Each category consists of subtypes named according to the characteristics of abnormal urine. Udakameha is characterized by excretion of dilute urine in excessive volume. Any disease, which shows glycosuria belongs to Pittamah. Sita meha or Madhameha according to the severity and pathophysiology of such diseases mentioned in modern medicine. Ikshumah is the second sub type of Kaphaja prameha characterized by excessive passing of sweet urine that is similar to juice of sugar cane. Sita meha is another sub type of Kaphaja prameha in which patient passes large volume of cold, sweet urine. Sandramah, Sandraprasadameha and Alatameha are pathological conditions, in which patient excretes turbid and viscous urine consisting of protein, phosphate, oxalate, calcium, mucus and other secretions of prostate etc. Many types of Pittaja prameha show different degrees of haematuria and haemoglobinuria. In Madhameha of Vataja Prameha, the terminal stage of nephropathy, sweet urine occurs in addition to the other characteristics of urine that are similar to bees' honey. Those three conditions represent different stages of glycosuria of different etiology. Many types of Pittaja Prameha and Vataja prameha represent different stages of nephrotic syndrome. Diabetes Mellitus, which is a highest prevalent disease in Prameha category, runs through many types of Prameha beginning from Ikshumah and terminating with Madhameha.

KEYWORDS: Prameha, Polyuria, Turbid Urine, Diabetes

Introduction

An extensive elaboration on the disease “Prameha" is found in classical Ayurvedic texts. The classification, etiology and pathology of Prameha have been discussed comparatively1. The term “Prameha" itself signify one of the common characteristic features - polyuria of the disease. On the basis of pathophysiological involvement, Prameha is categorized into three groups as Kaphaja prameha, Pittaja prameha and Vataja prameha. Each category consists of different subtypes named according to the specific qualities of excreted abnormal urine. The objective of this article is to discuss, in comparison to modern scientific perspectives, the common features of the disease and specific characteristics of abnormal urine excreted in each subtype of Prameha. It provides guidance to understand the pathology, pathophysiology, and to plan the management.

Normal day and night volume ratio of urine is 2:1. A volume of more than 2000 ml in 24 hours is termed polyuria and any increase of urine volume causes diuresis. The pH of urine reflects the ability of the kidneys to maintain normal hydrogen ion concentration in plasma and extra cellular fluid. An average adult on a normal diet excretes about 50 – 100 mEq of Hydrogen ions in 24 hours to produce urine of about pH 6. The normal urine pH may vary from 4.6 to 8.2. It has a faint aromatic odor and may consist of white form. Odor is useful in diagnosis of certain diseases. Lack of odor in urine from patients with acute renal failure coincides with acute tubular necrosis rather than pro-renal failure2. Normally, there is a scanty amount of protein in urine up to about 150 mg/24h or 10 mg/dL, depending on urine volume3. Proteins are derived from plasma and urinary tract. About one third is albumin; remaining plasma proteins include many small globulins. Plasma proteins with molecular weight less than 50,000-60,000 daltons pass through the glomerular membrane and are reabsorbed by proximal tubular cells. Albumin (molecular weight 69,000)
is apparently filtered but only in very small amounts. Retinol binding, tu - microglobulin, immunoglobulin light chains and lysozyme are excreted in small amounts. Tamim - Horsfall glycoprotein (Uromucoids), secreted by distal tubular cells and cells of the ascending loop of Henle, constitutes one third or more of total normal protein loss. Immunoglobulin A in secretion of urinary tract, enzymes and proteins from tubular epithelial cells, and other desquamated cells and leukocytes are other proteins found in very small amount in normal urine. More than 200 urinary proteins have been identified. Proteinuria is a consistent finding of dehydration and strenuous exercise in healthy persons. Moreover, it can occur in the absence of urinary tract diseases in patients with haemorrhage or salt depletion and febrile illnesses. These may cause dehydration and relative renal ischaemia.

Clinical features of Prameha

Prognostic symptoms and signs
The premonitory clinical features of Prameha include the burning sensation of hands and legs, excessive growth of nails, filthy odor of the body, smoothness, viscousness and heaviness of the body parts, sweetness of clear colourless urine, dryness, fatigue, thirst, shortness of breathing, collection of garlic material on the surfaces of palates, pharynx and tongue, nodular hair, sweating, flaccidity of the body, desire for constant lying on the bed, sitting, sleeping and feeling as something being adhered to the chest, eyes, tongue, and ears, copresence of the body, excessive growth of hair, matting of hair, desire for cold, dryness of palate and throat, sweetness in the mouth and gathering of ants on urine and body (Su.Ni.B.5, Ca.Cl.8.97, Ca.Cl.B.13-14, Ca.Ni.4.119). Each symptom is resulted from different mechanism of pathophysiology. The premonitory symptoms and signs mentioned in classical texts are predominantly related to the alteration of carbohydrate and lipid metabolism. Thirst, dryness of throat and dryness of palate are due to the alteration of water regulation caused by increase in plasma osmolality, defects of ADH secretion or its function at collecting tubules, renal tubular acidosis, impaired serum electrolytes levels such as hypokalaemia and levels such as hypokalaemia and hypercalcaemia.

In Prameha, thirst and dryness of throat and palate may occur as a result of polyuria, which is a cardinal symptom of the disease.

Common Features of Prameha
Prabutamutrita (Polyuria)
Excessive passing of turbid urine is a general feature of Prameha. The term “Prabutamutrita” stands for the excessive urine excretion (AHG.Ni.10). Excretion volume of urine that is more than 2000ml in 24 hours is termed as polyuria, where the normal day and night volume ratio (2:1) will be lost. Nocturia is arbitrarily defined as the excretion of more than 500ml of urine at night by an adult with a specific gravity of less than 1.018.

Polyuria may occur due to many pathological causes. Excessive intake of water and use of diuretics such as caffeine, alcohol, thiazide, intravenous saline and glucose are the general causes of polyuria. Polyuria, in pathological conditions, is resulted with an abnormality of glomerular filtration and tubular absorption. Tubular damage causes sodium wasting or impairment of the counter current mechanism resulting polyuria. Pyelonephritis or interstitial nephritis causes predominantly tubular dysfunction with polyuria early in the disease. In chronic progressive renal failure, functioning renal tissues are lost and the kidneys lose their ability to concentrate urine. In diabetes insipidus, urine volume increases with deficiency of antidiuretic hormone (ADH) or its activity at the receptors of renal tubules. Renal tubules are unable to absorb the excessive amount of glucose in diabetes mellitus and tubular damages of other reasons also fail to absorb the glucose resulting in polyuria with glycosuria.

Avilamutrita (Turbidity of urine)
The second common characteristic feature of Prameha is Avilamutrita, the turbidity of urine. In pathological conditions, presence of red cells, epithelial cells, spermatoza and prostatic fluids and proteins in urine cause turbidity not cleared by acidification or heating of urine. Detection of turbidity of urine is a indicator of disease in urinary system. presence of white cells, phosphates, oxalates and mucus also produce turbidity of urine. Urinary tract infections, Neplusis, Nephritic syndrom, and diseases of prostate gland produce turbid urine. The subtypes of prameha shows different digs of turbidity according to the pathology and pathophysiology.
Specific characteristics of abnormal urine

There are two categories of patients with Prameha. Strong obese patients are included in one category while weak emaciated patients are in the other (Ca.Ci.6.109, Ca.Ci.6.158). Clinically, the different types of Prameha have been identified on the basis of the hematologic analysis of urine or characteristics of urine such as appearance, color, smell and taste. All types of Prameha exhibit the relevant characteristics of urine according to the pathophysiology and pathology (Ca.Ci.6.98, Ca.Ci.6.128).

Udakameha

Udakameha, a subtype of Kapajameha, is characterized by passing of clear (Achamvara), colourless (Sita colour), odourless, cold, slightly turbid and viscous urine, which is similar to water (Ca.Ci.6.79, AHS.Ni.10.618) in excessive volume, without pain (Su.Ni.6.106, Ca.Ni.4.618). Polyuria with colourless very dilute urine may occur in diabetes insipidus, in addition to the other reasons including excessive drinking of water, which may occur in the early stage of impaired glucose tolerance. In diabetes insipidus, the deficiency of vasopressin or its activity leads to polyuria, nocturia, thirst and compensatory polydipsia. Daily urine output may reach as much as 10-15 litres.

Ikhshubilkarasamhe (Ikhumeeha)

Ikhumeeha is characterized by passing of excessively sweetened, cold, slightly viscous, slightly turbid urine similar to the juice of sugar cane (Su.Ni.6.1018, Ca.Ni.4.6, Ca.Ci.6.69, AHS.Ni.10.18). In other words, Ikhumeeha comprises glycosuria with moderate polyuria of any reason. Pale urine consists of glucose with a high specific gravity, which may be found in diabetes mellitus. Glucose may appear in the urine at different blood glucose levels. The glucose level in blood, glomerular blood flow, tubular reabsorption rate and urine flow influence its appearance. Glycosuria usually occurs when the blood level is more than 180-260mg/dl.

Glycosuria and hyperglycaemia may accompany central nervous system diseases, brain tumor or haemorrhage, hypothalamic disorders, asphyxia, disturbance of metabolism associated with burns, infections, fractures, myocardial infarction, and uraemia. Liver diseases, glycogen storage diseases, obesity, and feeding after starvation, are also associated with glycosuria.

Pregnant women may produce glycosuria where the glomerular filtration rate increases and the total amount of filtered glucose may not be reabsorbed. Glycosuria without hyperglycaemia is usually associated with renal tubular dysfunction.

In renal tubular transport diseases, glycosuria is not a major finding but one of many. Reabsorption of water, amino acids, bicarbonates, phosphates, and sodium is impaired in conditions such as Fanconi's syndrome. Galactocoria, cystinosis, lead poisoning, and myeloma are a few conditions associated with renal tubular dysfunction and glycosuria. Fructose, Galactose, Lactose, Maltose, and L-sululose are also found in urine in patients with inherited metabolic disorders.

Small amount of disaccharides is normally excreted in the urine, about 50mg in 24 hours. With intestinal diseases such as severe sprue or acute enteritis, the level may rise to 250mg or more. With high levels of sugars in the gut in lactose intolerance is absorbed and excreted unchanged in the urine. Galactose is found in the urine in genetic disorders of galactose metabolism associated with a deficiency of galactose-1-phosphate uridyl transferase or galactokinase.

Diabetes Mellitus is well-defined metabolic syndrome, which produces glycosuria. Ilusmeha includes all conditions, which show sugars in urine with or without mild nephropathy. It excludes the condition of glycosuria with severe nephropathy, which belongs to the Madhurameha, the terminal stage of nephropathy of any aetiology.

Sandrameha

In this condition, condensation of urine, at keeping stand for some time, occurs in addition to the turbidity (Su.Ni.6.1018, Ca.Ni.4.5, Ca.Ci.6.88, AHS.Ni.10.18). Normal concentrated urine may show a sedimentary deposit if allowed to stand after cooling from body temperature. Precipitations may occur due to the excessive presence of organic and inorganic matters such as cells, proteins, phosphates and urates etc. Proteinuria is one of the most common signs of renal disease. The low molecular weight proteins such as α1 microglobulin, β1, microglobulin, light chain immunoglobulins and lysozymes are reabsorbed. When only albumin or smaller proteins are found, the pattern indicates minimal change disease with more favourable prognosis.
Glomerular diseases often cause heavy proteinuria, greater than 3-4g/day. Small amount of protein in urine, which can be found in diabetic patients, correlates with very early diabetic nephropathy. A loss or reduction of the fixed negative charge on the glomerular capillary wall allows albumin to permeate into Bowman's space in large quantities more than can be reabsorbed by the proximal tubular cells.

When serum albumin is lost in urine, other proteins of similar size or charge such as antithrombin, transferring, pre-albumin, α1 acid glycoprotein, α1 Antitrypsin etc are also lost. Microalbumin that is greater than 100μg/day indicates renal tubular damage. Persistent proteinuria (1-2g/day) in an asymptomatic person or when accompanied by haematuria has a poor prognosis than intermittent or postural proteinuria.

Large proteins such as α2, Macroglobulin and α Lipoproteins are not found in urine while the glomerulus is still selective. As larger proteins appear, the proteinuria is less selective, indicating greater morphological changes such as membranous nephropathy and proliferative glomerulonephritis. Nephrotic syndrome is principally associated with glomerular disease and is diagnosed when the protein excretion is greater than 3.0 - 3.5g/day or 2g/m²/24h. Sometimes losses of 10-20g of protein per day are also found. In addition to proteinuria, nephrotic syndrome is characterized by low serum albumin level, generalized oedema and increased serum lipids (Cholesterol, Triglycerides and Phospholipids). Low density and very low density lipoprotein are increased in serum where as high density lipoprotein, small molecule, has been demonstrated in urine. With lipid lost in urine, many granular cast, fatty cast, fat-filled renal tubular epithelial cells are found in urine.

**Sandraprasadamaha**

Sample of urine separates into two phases: Condensed and Clear (Ca.Ni.4.6, Ca.Ci.6.8). Mucus from the urinary passage may cause a fluffy, bulky deposit. This is increased in inflammatory diseases of urinary system and genital tract. Mucus from the urinary and genital tract is seen as small cloudy patches (Nubeculae) in normal urine. Benign and malignant tumors, urinary tract infections and urinary calculi produce different patterns of sedimentations formed by the precipitation of proteins, inorganic and organic matters, epithelial cells, red cells, pus cells, casts and crystals.

**Suklamaha and Pishlamaha**

The term Sukla stands for white colour. Suklamaha shows increase in frequency of urine and urines is in white colour (Ca.Cu.6.8) consisting a matter that is similar to flour (Ca. Ni.4.6). Pishlamaha refers to a similar condition called Pishlamaha, which is not mentioned in Carakasamhita, shows an erection of hair at the moment of passing of urine that is similar to flour dissolved in water (Su.Ni.6.10 - 116, A.H.S.Ni.10). The appearance of urine may be due to the presence of phosphate and carbonate in alkaline urine. Leukocytes and Phosphates in urine may also produce white clouds. White or cloudy urine is most commonly a result of phosphaturia. This is a benign condition in which excess ammonium phosphate crystals form in urine. Adding a drop of acetic acid to the urine sample will result in immediate clearing of the urine. Phosphaturia is usually intermittent, occurring following a meal or after ingesting a large quantity of milk. White urine is sometimes due to pyuria (abundant white blood cells) in association with an infection of the urinary tract. White cloudy urine can rarely be due to chyluria (lymph fluid), resulting from a communication with between the lymphatic system and the urinary tract. Phosphaturia occurs in primary and secondary hyperparathyroidism. Chronic renal failure is the most important renal disease affecting calcium and phosphorus metabolism and excretes high amount of phosphates in urine causing urinary calculi formation. Fanconi syndrome is characterized by increased urinary excretion of phosphate, glucose, amino acids and systemic acidosis.

**Sukramaha**

The term "Sukra" in general, refers to the seminal fluid. The frequency of urine increases in Sukramaha and urine may consist of Sukra or is similar to Sukra (Su.Ni.6.10, Ca.Ci.6.8). In the case of benign prostatic hyperplasia and prostatic carcinoma prostatic secretion may mix with urine and gives the characteristic appearance of urine.
Si tamaha
Si tamaha is not mentioned in Susratasamha. According to the Carakasamhita it shows passing of excessive volume of sweat and cold urine (CaNi.6.610^6, CaCI6.610^6). Sweating of urine can be attributed to the presence of sugars in urine. Heavy porya with glycosuria is a more severe stage of diabetes mellitus than the lakshamaha.

Sanrimeha
In this condition, patient passes urine in small volumes, frequently, with difficulty in low velocity. Urine consists of Kajha and is viscous (SuNi.6.10^6, CaCl6.610^6, CaCI6.610^6, AHSNi.1010^6). Frequency of urination and difficulty or delay in initiation of urination are early symptoms of prostatic hyperplasia. The viscosity may be due to the prostatic secretion mixed with urine.

Sikatehawa
In this condition, patient experiences painful passing of urine that consists of minute particles similar to sand (SuNi.6.10^6, CaCl6.610^6, CaCI6.610^6). The term “Sikata” stands for sand. The minute particles of urinary stones excrete with urine in urolithiasis. Crystals of calcium oxalate, calcium phosphate, urates and uric acid are the responsible agents in formation of urinary calculi. The nephrolithiasis, the deposition of calcium salts in the renal parenchyma, may be associated with urolithiasis. Aggregation of crystals with a small amount of protein and glycoprotein matrix material form urinary stones in urine outside the renal parenchyma. Most of the urinary stones are of multifactorial origin. Hypercalcaemic nephropathy, hyperuricosuric nephropathy, obstructive uropathy etc are the complications of Sikitamaha.

Ahalaiahaha and Lalameha
Ahalaiahaha is mentioned in Carakasamhita. In this condition patient passes viscous urine similar to the pukhaam bordered by a thread. The same pathological condition is known as Lalameha (AHSNi.1010^6, CaCl6.610^6, AHSNi.1010^6). In Susratasamhita, no reference is found for Lalameha or Ahalaiahaha. The appearance of urine in Ahalaiahaha or Lalameha may be due to the presence of large amount of protein, mucus, and prostatic secretions.

Surameha
Patient passes urine that is similar to wine; the top layer of urine is clear while the bottom layer is condensed on standing for some time (SuNi.6.10^6, AHSNi.1010^6). Carakasamhita does not refer to this condition. Individuals who suffer from diabetes commonly exhibit renal malfunction that results in an excessive amount of glucose and protein in their urine. In the presence of yeast or bacteria, urinary glucose can be converted to alcohol by fermentation. Thus, a person with diabetes and who has a urinary tract infection might have alcohol in their urine.

Lavanameha
Patient passes clear urine similar to saline (SuNi.6.10^6). This condition is referred to in Susratasamhita only. Many kinds of salts are present in normal urine and they give a characteristic salt taste of urine. Excessive concentration of sodium, potassium and chloride are the responsible agents for excretion of saline urine. This condition is associated with the defects of electrolyte and water metabolism.

Phrenameha
Patient passes urine frequently, which consists of form (SuNi.6.1012). This condition is referred only in Susratasamhita. This may occur due to the gas formation in urinary tract infections and vesico-rectal fistula.

Ksharamaha
Ksharamaha is characterized by passing of urine similar to an alkaline solution in colour, smell, taste and tactile (SuNi.6.1012, CaCl6.610^6, CaNi4.610, AHSNi.1010^6). In respiratory alkalosis, alkaline urine is produced and is associated with increased excretion of bicarbonate. In Metabolic alkalosis, alkaline urine with higher levels of bicarbonate is produced and ammonia production is decreased. The kidney may produce urine with higher pH value of 7.8. Alkaline urine may be induced by using some fruits and vegetables. Administrations of Sodium bicarbonate, Potassium citrate, and Acetazolamide are also the causes of Alkaline urine. In classic renal tubular acidosis, glomerular filtration is normal but distal tubular ability to form ammonia and exchange hydrogen ion for sodium is defective and systemic acidosis results and urine is relatively alkaline in the condition. In proximal renal tubular acidosis, bicarbonate wasting occurs as in proximal tubular diseases such as Fanconi’s syndrome. In respiratory alkalosis, alkaline urine is produced and is associated with increased excretion of bicarbonate.
**Kalamaha**
This condition is characterized by passing of excessive volume of black or dark brown coloured warm urine (Ca.Cl.6.6, Ca.Ni.4.89, AHS.Ni.10^12). Dark brown or black urine may be due to the presence of haemoglobin in acid urine, which forms methemoglobin. In the presence of unstable haemoglobin such as Haemoglobin Kell, the urine becomes red brown. Yellowbrown or greenbrown urine is most often associated with bile pigments, chiefly bilirubin. Dark brown urine can be seen in Akapunam (Homogentisic Acid) and Melanin. Colourless melanogens are converted into Melanin in acid urine.

**Nilameha**
It is characterized by passing of clear, formed (Su.Ni.6.10^12) sour tasted urine in bluish colour (Ca.Cl.6.69) of bird "Cashew" (Ca.Ni.4.89, AHS.Ni.10^10). Blue green coloured urine may be due to the presence of Indicans and Chlorophyll. It can be observed in small intestinal infections such as pseudomonal infections.

**Raktameha and Sonitameha**
In Raktameha, patient excretes salt tasted warm urine similar to raw flesh in odor and similar to Raka or Shonita in appearance (Su.Ni.6.10^5, Ca.Cl.6.8, Ca.Ni.4.89, AHS.Ni.10^10). This type of urine may occur due to the presence of blood in urine or haematuria. Haematuria may appear cloudy, smoky, pink, red, or brown in colour. Red brown colour of urine is due to the presence of erythrocytes in urine. The term "Sonitameha" refers to the similar condition in which patient passes urine that is similar to Sochita or blood (Su.Ni.8.11^6).

**Manjisthameha**
Passing of urine similar to raw flesh (Su.Ni.6.11^6) in odor and in the colour of Manjistholika (Water extract of Manjistha (Ca.Cl.6.98, AHS.Ni.10^10)) is the characteristic feature of this condition. Haematuria may appear as cloudy, smoky, pink, red, or brown in colour. Urine in the Haemoglobinuria is clear red, clear red brown or dark brown. The colour of urine in Manjishameha signifies the amount of haemoglobin or red cells in urine.

**Haridrameha**
In this condition patient passes urine similar to Haridrodaka in colour (Ca.Cl.6.68), Katuka (pungent) in taste and patients feel burning sensation on passing urine (Su.Ni.6.11^6, AHS.Ni.10^10).

In severe obstructive jaundice, the urine may be dark green, orange red or orangebrown due to presence of large amount of urobilin in urine.

**Ammamah**
Patient passes urine that is in sour taste and is similar to acid (Su.Ni.6.10^5). This condition is referred in Susnutasamthia only. Metabolic activity of the body produces non volatile acids that cannot be excreted by lungs principally sulphuric acid, phosphoric acid, and hydrochloric acid but small amount of pyruvic acid, lactic acid, citric acid and some ketone bodies. When protein intake is high, more phosphates and sulphates are produced, resulting in more acid urine. In respiratory acidosis acid urine is formed and the amount of ammonium excreted is increased. These acids are excreted by Glomeruli with cations, chiefly sodium bicarbonate is reabsorbed. The tubular cells exchanges hydrogen ions for sodium of the glomerular filtrate and urine becomes acid in reaction. Hydrogen ions are also excreted as ammonium ions (NH4). In metabolic acidosis, acid urine is produced and titratable acidity and concentration of ammonium ion is increased. In chronic acidosis as Diabetic Ketoacidosis, very large amount of hydrogen ions excreted, much of it as ammonium ion. When protein intake is high, more phosphates and sulphates are produced, resulting in more acid urine. Acid urine may be produced by a diet high in meat protein and in some fruits. In respiratory acidosis acid urine is formed and the amount of ammonium excreted is increased.

**Vasameha**
Vasameha is characterized by the presence of frequent passing of urine similar to Vasa (Lymph) or mixed with Vasa (Lymph) (Su.Ni.6.10^5, Ca.Cl.6.7, Ca.Ni.4.10^5, AHS.Ni.10^12). This can be interpreted as Chyuria, which shows milky appearance and it is resulted from lymphatic obstruction. Chyuria is rare. Urine contains Lymph, It is associated with the obstruction to lymph flow and rupture of lymphatic vessels into the renal pelvis, ureters; bladder or urethra. Filariasis (Late in the disease), abdominal lymph.
node enlargement, and tumors are associated with Chyluria. The appearance of urine varies with the amount of lymph present. It may appear normal, opalescent, or milky. It may clot if sufficient lymph is present, after meal. Urine may separate into layers showing the chylomicron on top and fibrin and cells beneath. Large number of cells may cause the pink colouration. Chylomicrons may not apparent microscopically unless they may have coalesced as microglobules.

**Sarpinmela**
Patient passes urine similar to Ghee (Su.Ni.6.111). This condition is referred in Susrita Samhita and can be identified as Lipiduria, which is resulted from Nephrosis, or crush injury. With lipid lost in urine, many granular cast, fatty cast, fat-filled renal tubular epithelial cells are found in urine. Endogenous lipids may float on the surface of urine. Fat globules may appear in the Nephrotic Syndrome. These are neutral fat (Triglycerides) and cholesterol. Lipiduria is also present in a significant number of patients who have sustained major skeletal trauma with one or more fractures to major long bones or pelvis. The source of the lipid presumably is exposed fatty marrow.

**Maljameha**
Maljameha shows frequent passing of urine mixed with Majja (Marrow) (Ca.Ci.6.79, Ca.Ni.49, AHS.Ni.10.10). This is closely related to Sarpinmela. The difference of the names is due to the appearance of the particles in urine.

**Hastimela**
Patient passes slowly a large volume of urine mixed with Laska (Ca.Ci.6.79, Ca.Ni.4.109, Su.Ni.6.116) and is similar to that of elephant gone amok (Su.Ni.6.111, AHS.Ni.10.10). Inflammatory conditions of the urinary tract are associated with this condition. The sloveness of voiding urine may be due to the defect of neurogenic control on bladder.

**Kshauiramacha**
Urine is similar to Kshaura in taste and colour (Su.Ni.6.111). The urine is madhura, and kashaya in taste and ruksha (rough) in quality. Kshauura is a variety of bee-honey that has light maroon colour. The colour of the urine represents the intensity of haematuria. Madhumeha and Kshauiramacha are closely related stages of Nephrotic syndrome.

**Madhumeha**
Madhumeha belongs to the category of Medodoshaja roga, which includes the conditions caused by defective functions of Meeta (Su.Su.24.6) and it is a Maharoga - Greatly incurable Disease (Su.Ci.14.115). The characteristics of urine is sweet and kashaya in taste (Ca.Ni.4.9, Su.Ni.6.10, Su.Ci.12.6-912) and is similar to that of Madhu, a variety of bee-honey, and the term "Madhumeha" is given because of colour, smell and taste of urine, which are similar to those of bee-honey (Su.Ni.5.10, Su.Ci.12.6-912, AHS.Ni.10.10). The colour of urine is pandu and is ruksha (rough) in the quality (Ca.Ni.4.9). In this condition oja is mixed with urine (Ca.Ci.5.712). Oja, which is the essence, includes the vital constituent that maintains life. The Madhumeha, which is the fatal stage of all type of Prameha, is characterized by abscess and other complications of Prameha. It is incurable and patient suffers restlessness (Su.Ni.6.2412). Diagnosis of Madhumeha is confirmed at the stage of patient, who is affected with Prameha Pidaka (Cordbundles) showing the characteristics of complications (Su.Ni.6.249).

**Complications of Prameha**
Prameha occurs as a result of defects of Vasti, one of the major vata intake organs of the body, which involves in regulation of body functions. Defects of Vasti may impair the functions of other organs resulting pathological changes, which are known as complications. The features of such complications in Kapaja Prameha include wasting of body tissues (muscules etc), looseness of limbs, anorexia, indigestion, salivary discharge, vomiting, somnolence, lassitude, catarh, cough, difficulty in breathing and gathering of flies on the body (Su.Ni.6.1312). The features of complications of Pitaja Prameha include tearing pain of scrotum, sever pain in the region of bladder, piercing pain in the penis; stabbing pain in the region of heart, acidic eruction to mouth, pyrexia, diarrhoea, anorexia, vomiting, generalized heat, burning sensation, thirst, generalized weakness (Ca.Ni.4.125, Su.Ni.6.1312), insomnia, anaemia, and yellow coloured urine and stools (Su.Ni.6.1312). The complications of Vataja Prameha may result in tightness of the chest, eager to take all types of taste, insomnia, rigidity of the body, tremor, stabbing pain, and constipation (Su.Ni.6.1312).

Pidaka such as putimansa (Gangrene), Aalaj and Vidradhi etc. are the major complications of Prameha.
Viulued medes and all the doha are responsible for the genesis of Prameha Pidaka (Su.Ni.6.712, Ca.Su.17.539). Saranka, Kakchaiky, Jalani, Sarsha, Alaj, Vinata, and Vidrith are the seven kinds of abscesses (Ca.Su.17.3.4) occurring in fleshly areas, joints and vital organs due to the poor management of Madhumeha (Ca.Su.17.38-39).

Investigations

Organoleptic investigations have been carried out in the diagnosis of Prameha. Carakasaamhita and Susrutasamhita mention the characteristics of urine, on the basis of which, each type of Prameha can be diagnosed precisely. Taste of urine had been detected by using indirect methods (Su.Su.10.512). Urine of patients is analyzed according to its nature, colour, smell, and appearance (Su.Ni.6.1012). The results have been elaborated as features of Prameha.

Diagnosis of Prameha

Diagnosis of Prameha depends on the clinical features and characteristics of urine and the confirmation of diagnosis is made on the following criteria:

- Appearance of premonitory symptoms and signs with moderate polyuria (Su.Ni.6.2212) or,
- Partial appearance of premonitory symptoms and signs with heavy polyuria (Su.Ni.6.2312).

Diagnosis of Madhumeha is made at the stage of patient, who is affected with Prameha Pidaka showing the characteristics of complications (Su.Ni.6.2412).

Differential Diagnosis of Prameha

Susruta mentions the features in differentiating the hereditary Prameha and acquired Prameha. The patients suffering from hereditary Prameha show the characteristics such as leanness and roughness of the body, excessive thirst, taking of small quantity of food and restlessness. The patients of acquired Prameha show the characteristics such as excessive taking of food, smoothness of the body, excessive sleeping and desire to have sedentary life style (Su.Ci.11.0312).

In conclusion, according to the classical texts of Ayurveda, Prameha is a pathological condition, which shows excessive passing of turbid urine due to many causes. The nosology of sub types of prameha represents the abnormalities of urine. Usually, the clinical course of Prameha begins with Kaphaja Prameha and then it turns into Pittaja Prameha and Vataja Prameha respectively; but according to the severity of aetiology, Prameha may start from any stage of the disease with one or more specific characteristics of different types of Prameha. According to Modern Medicine, Prameha includes a large number of diseases of different causes and pathology. A subtype of Prameha represents a particular clinical stage of the diseases of urinary system mentioned in modern medicine.

In Udakameha, patient excretes dilute urine in excessive volume. Any disease, which shows glycosuria belongs to Ikshumeha, Stitametha or Madhumeha according to the severity and pathophysiology of such diseases mentioned in Modern Medicine. Sandrameha, Sandraprasada-meha and Alasameha are pathological conditions, in which patient excrete turbid viscous urine consisting of protein, phosphate, oxalate, calcium, mucus and other secretion of prostate etc. Many types of Pittaja prameha show different degree of haematuria and haemoglobinuria. Ikshumeha is the second sub type of Kaphaja meha characterized by excessive passing of sweet urine that is similar to juice of sugar cane. Stitametha is another sub type of Kaphaja meha in which patient passes large volume of cold, sweet urine. In Madhumeha of Vataja Prameha, the terminal stage of nephropathy, sweet urine occurs in addition to the other characteristics of urine that are similar to bees' honey. These three conditions represent different stages of glycosuria of different aetiology. Many types of Pittaja prameha and Vataja prameha represent different stages of nephritic syndrome. Diabetes Mellitus, which is the highest prevalent disease in the Prameha category, runs through many types of Prameha beginning from Ikshumeha and terminating with Madhumeha.

References


