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REVIEW ARTICLE **OPEN ACCESS**

Traditional Medicinal Plants Used in Treatment of Urolithiasis In Maharashtra Region : A Review

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ABSTRACT
Kidney stone is the common disorder of world as it affects 2% of world population. Urolithiasis is the more common disorders & it is recurrent disorder which having relapse rate 50% in 5-10 years. Advancement in technologies has only dramatic improvement in urinary stone. As the recurrence rate is much more and it is more costly. From an ancient time human being is dependent on indigenous medicinal system like Ayurvedic, Unani and Siddha. In recent years use of traditional plants is on rise due to its advantages. herbal remedy are reported as they are effective & having no side effect it have motivated people to return to nature for safe remedies. In the present review an attempt has been made to focus on the medicinal plants which are used for treatment of kidney stone which are available in Maharashtra region.
KEYWORDS: *Urolithiasis, kidney stone, TMP*

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Introduction-

Renal or Urinary calculi also called Urolithiasis. It is the word derived from Greek word ouron,"urine" Lithos, " stone". Urolithiasis is the condition where calculi are formed or located anywhere in the urinary system. It affect about 4-15% of world population approximately. In India 12% of population is expected to have urinary stones out of which 50% may end up with loss of kidney or renal damage. [1]

It is the third most common disorder of urinary system after urinary tract infection & prostate disease in past years, incidence of kidney stone is increasing in both male and female. As it affects all ages. 50-60 is the peak age for incidence of Urolithiasis. Calcium stones with female to male ratio (1:2) which is due to larger muscle mass of male & high presence of testosterone it favours formation. Whereas estrogen helps in prevention of stone in female. As it keeps urine alkaline and increases level of citrates.

Eighty percent stone are generally calcium stones. Whereas, two types of calcium stones are there calcium oxalate and calcium phosphate while 10-15% is mixed stones 15-30% are stuvite, 6-10% cystine & uric acid stone 2-10% kidney stones are hard, solid particle. The mechanism of calculi formation is a complex process concerned with super saturation, nucleation, aggregation, growth & retention of crystals in renal tubules. The etiopahogeneis involve anatomic, environmental infections, metabolic & dietary habits.

Prevention of Urolithiasis is carried out by inventing symptomatically, physiologically & change in lifestyle increasing fluid intake, painkiller for alleviation of colic pain, administration of salt monitoring drug helps in management of stone. [2] but the medicines are not effective at all stages surgical management like extracorponeal shock wave lithotripsy are latest option to manage & tract stones. But it is so expensive with so many side effect & also it do not prevent recurrence rate. That's why people are coming back to nature for their effectiveness [3]. Now-a-days, herbal medicine gained much popularity because they are more effective, having less side effect, reduce recurrence rate of stone formation. Hence search for Antiurolithiatic drug from natural sources has assumed greater importance. In ayurveda ,many plants having the property of disintegrating &dissolving stones termed as "Pashanbhedi". Man has been dependant on the plant based medicine from ancient time [4]. Belief & observations regarding traditionally used medicinal plants, enhancing interest of people to use natural medicine for their primary health care needs. [5]

AETIOLOGY

Urolithiasis is the chronic disease that affected human being from last so many centuries. In these disease pain get precipitated due to calcium oxalate stone in kidney or in any part of urinary tract. Perfect cause is not known but age, sex, diet, life style, medical history responsible for it. [1]

From ancient time people have been using plants & animal source as a source of medicine. The overuse of synthetic medicine results in higher incidence of adverse drug reaction has motivated people to return to nature for safe remedies.

Herbs & herbal medicine have created interest among the people by it's clinically proven effects. So, there is need for detailed scientific validation of traditional medicine to establish their efficacy & safety in light of modern science. [2]

Reasons of herbal treatment-

1. Low cost
2. No side effects
3. Re occurrence rate is less.

TYPES OF KIDNEY STONES

Uroliths or the renal stones which are formed in the kidneys are of various forms based on their chemical composition and their structures. Based on the predominant chemical composition, uroliths are classified as-

1. Calcium containing stones
a. Calcium oxalate stones (CaOx) 60%
b. Calcium phosphate stones (CaPh) 20%
2. Uric acid stones 10%
3. Struvite stones 8%
4. Cystine stones and 1.5%
5. 2, 8-Dihydroxyadenine stones 0.5

1. Calcium nephrolithiasis: All calcium stones are radio-opaque, and calcium oxalate and calcium phosphate stones are black, grey, or white and small (1 cm in diameter), dense, and sharply circumscribed on radiographs. Most stones contain calcium combined with oxalate, phosphate, or occasionally uric acid. Different conditions contribute to calcium stones. Such disorders include:

(1) Primary hyperparathyroidism, which results from an adenoma in 85% of cases, and is associated with mild to moderate hypercalcaemia. People who form stones and have hypercalcaemia are almost certain to have this parathyroid problem. Hypercalciuria is a result of excess parathyroid hormone, which cause overproduction of 1,25– dihydroxyvitamin D in the kidney; both factors promote bone resorption, increasing the filtered load of calcium and hence calciuria. [68]

2) Other disorders that induce hypercalcaemia can also result in hypercalciuria, malignancies, granulomatous diseases, sarcoidosis, thyrotoxicosis, and immobilisation.

(3) Idiopathic hypercalciuria is a familial disorder affecting both sexes equally, in which urinary calcium concentration is raised despite normal concentrations of blood calcium. the primary mechanisms for such hypercalciuria can occur individually or in combination. [69]

2. Uric acid nephrolithiasis:

Uric acid stones are smooth, round, yellow-orange and nearly radiographically transparent—unless mixed with calcium crystals or struvite, Uric acid becomes insoluble at low urinary pH and urate stones may form secondary to hyperuricaemia or acidic urine alone. Diets high in purines, especially those containing organ meats and fish, result in hyperuricosuria and in combination with low urine volume and low urinary pH (as a result of impaired renal ammonia production), can exacerbate uric-acid stone formation. Uric acid salts out calcium oxalate, and can precipitate out in acid urine even in the absence of raised serum or urinary uric-acid concentrations. [68]

3. Struvite nephrolithiasis:

Radiographs show struvite stones as large and laminated. They are associated with substantia morbidity including bleeding, obstruction, and urinary tract infection. Signs of storuvite stones include urinary pH greater than 7, staghorn calculi, and urease that grow bacteria on culture (proteus, klebsiella and pseudomonas). Stones develop if urine is alkaline, has a raised concentration of ammonium, contains trivalent phosphate, and contains urease produced by bacteria. [68]

4. Cystine nephrolithiasis:

Cystine stones should be suspected in patients with a history of childhood stones or a strong family history. They are greenish-yellow, flecked with shiny crystallites, and are moderately radio-opaque with a rounded appearance. Calcium stones are denser than vertebral bodies, whereas cystine stones are less

dense. People who are homozygous for cystinuria (an autosomal recessive disorder) excrete more than 600 mg per day of insoluble cystine. More than half the stones in cystinuria are of mixed composition, and many patients have associated physiological problems such as hypercalciuria (19% of patients), hyperuricosuria (22%), and hypocitraturia (44%). [68]

5. Dihydroxyadenine stones (2, 8-Dihydroxyadenine stones): This is a rare inherited form of renal stone disease, secondary to adenine phosphoribosyl- transferase (APRT) deficiency. Awareness of this type of stone is required as it may lead to progressive renal Failure. Stones are radiolucent and may vary from microscopic to gravel and staghorn calculi.The diagnosis may be made using urine microscopy where urinary sediment shows spherical brownish crystals.[68,69]

PATHOPHYSIOLOGY OF RENAL STONE FORMATION

Calcium stone formation involves different phases of increasing accumulation of CaOx and Calcium phosphate (CaP) nucleation, crystal growth, crystal aggregation, crystal retention. [68]

1. Nucleation:

Nucleation is the formation of a solid crystal phase in a solution. It is an essential step in renal stone formation. The term supersaturation refers to a solution that contains more of the dissolved material than could be dissolved by the solvent under normal circumstances. The level of supersaturation of a salt is expressed as the ratio between the actual ion activity product (AP salt) and the solubility product (SPsalt). The ion-activity of a salt is calculated from the free ion concentrations and the activity coefficients corresponding to the charge of the ions in the salt. The point at which saturation of a solution is reached, and crystallization begins is commonly known as thermodynamic solubility product (Ksp). Urine contains inhibitors of crystallization and can hold large concentrations of solute above the Ksp, a metastable state. If the concentration of solute increases further and a point is reached where it cannot be held in solution, this concentration is known as KF, which is the point of formation of product in urine. The process of nucleation in a pure solution is known as homogeneous nucleation. In secondary nucleation, new crystals deposit on pre-existing crystal surfaces of similar type. Secondary nucleation results in the'mass production' of crystals. [69]

2. Crystal growth:

After nucleation, crystal growth is the next major step of stone formation. What causes crystals to ''grow''? The driving force for crystallization is a reduction in the potential energy of the atoms or molecules when they form bonds to each other. The crystal growth process starts with the nucleation stage. Several atoms or molecules in a supersaturated liquid start forming clusters; the bulk free energy of the cluster is less than that of the liquid. The total free energy of the cluster is increased by the surface energy (surface tension), however, this is significant only when the cluster is small. Crystal growth is determined by the molecular size and shape of the molecule, the physical properties of the material, SS levels, pH, and defects that may form in the crystal's structure. Crystal growth is one of the prerequisites for particle formation. Using the powerful atomic-force microscope (AFM), Laboratory researchers are discovering complex growth mechanisms and three-dimensional structures of solution-based crystals. [69]

3. Crystal aggregation (crystal agglomeration):

In this process crystals in solution stick together and form a larger particle. Aggregation of particles in solution is determined by a balance of forces, some with aggregating effects and some with disaggregating effects. A small interparticle distance increases attractive force and favours particle aggregation. In addition, Tamm-Horsfall glycoprotein and othermolecule may act as glue and increase viscous binding. Furthermore, aggregate may be stabilized by solid bridges formed by crystalline material connecting two particles. The main force that inhibits aggregation is the repulsive electrostatic surface charge, known as Zeta potential. [69]

4. Crystal retention:

Urolithiasis requires formation of crystals followed by their retention and accumulation in the kidney. Crystal retention can be caused by the association of crystals with the epithelial cells lining the renal tubules. Crystal formation predominantly depends on the composition of the tubular fluid; crystal retention might depend on the composition of the renal tubular epithelial cell surface. A non-adherent surface of the distal tubules, collecting ducts, ureters, bladder, and the urethra may provide a natural defence mechanism against crystal retention, and may become defective when the anti-adherence properties are compromised. [7]

3. Complications of Urolithiasis -

Urinary stones often remain asymptomatic for long periods. As the time surpass the stones develop into macroscopic sizes and if not treated for long time they lead to severe complications. The brutal complications include-

- Recurrence of stones
- Proteinuria
- Obstruction of the ureter, acute unilateral obstructive uropathy
- Kidney damage, scarring
- Decrease or loss of function of the affected kidney.
- Calculi
- Stag horn calculus
- NephrocalcinosiS
- Sludge and Osteoporosis.

Signs and symptoms:

A kidney stone may or may not cause signs and symptoms until it has moved into theureter the tube conn ecting the kidney and bladder. At thatpoint, these signs and symptoms may occur:

Severe pain in the side and back, below the ribs

- Pain that spreads to the lower abdomen and groin
- Pain on urination
- Pink, red or brown urine
- Nausea and vomiting
- Fever and chills if an infection is present
- Urinary frequency/urgency, increased (persistent urge to urinate),
- Blood in the urine,
- Abdominal pain,
- Genitalia pain,
- Groin pain,
- Fever,
- Renal colic Musculoskeletal pain
- weakness, and loss of appetite
- Renal infarct
- Renal hemorrhage
- Pyelonephritis
- Duodenal ulcer
- Cholecystitis
- Gynecologic disorders [70,71]

THE PRESENTATION AND DIAGNOSTIC WORKUP OF NEPHROLITHIASIS

Kidney stones may present with symptoms or be found incidentally during investigation of other problem.

The differential diagnosis in a patient with symptoms suggesting renal colic includes:

1) Stone analysis

A) Serum and blood testing

Calcium, parathyroid hormone, vitamin D, electrolytes

B) Urine dipstick and microscopic examination

Urine Ph,urine sediment examination for crystals, leukocytes, erythrocytes bacteria

C) Urine culture (24-Hour urine collection)

Volume, creatinine, calcium oxalate, sodium, citrate, uric acid .

2) Diagnostic tests The diagnosis can be confirmed by

a) Ultrasonography has the advantage of not using radiation, but it is less sensitive for detecting stones and can image only the kidney and the proximal ureter. A retrospective study in 123 patients found that, compared with helical CT as the gold standard, ultrasonography had a sensitivity of 24% and a specificity of 90%. Ultrasonography may also miss stones smaller than 3 mm in diameter.

b) Helical CT (computed tomography) without contrast is the preferred imaging study in patients with suspected nephrolithiasis. It has several advantages over other imaging studies: it requires no radiocontrast material; it shows the distal ureters; it can detect radiolucent stones (i.e, uric acid stones), radio-opaque stones, and stones as small as 1 to 2 mm; and it can detect hydronephrosis and intra-abdominal and renal disorders other than stones that could be causing the patient's symptoms.

c) Conventional radiography (kidney-ureter bladder view) is inadequate for diagnosis as it may miss stones in the kidney or ureter (even small radio-opaque stones) and provides no information about possible obstruction.
d) Renal ultrasonography or Intravenous pyelography.
e) It has few advantages in renal lithiasis, exposes the patient to the risk of radiocontrast infusion and contrastmediated acute renal injury, and gives less information than noncontrast CT.
f) Plain abdominal radiography: Calculi that contain calcium are radiodense. Sulfur containing stones (cystine) are relatively radiolucent on plain radiography.[72]

SURGICAL MANAGEMENT OF PATIENTS WITH STONE DISEASE

Management of stone disease largely depends on the size and location of stones. Stones smaller than 5 mm that were more distal and on the rightside have a high probability of spontaneous passage. However, spontaneous stone passage may take up to 40 d. During this watchful waiting period, patients can be treated with hydration and with pain medications to control pain. In contrast, stones larger than 5 mm, stones in patients with a higher risk of developing renal insufficiency (eg, patients with a single kidney), or stones that fail to pass through should be treated by some interventional procedures including extracorporeal shock wave lithotripsy (SWL), ureteroscopy (URS), or percutaneous nephrolithotomy (PNL) [72]

1. **Extracorporeal shockwave lithotripsy (ESWL) removal of renal and ureteral Stones:**

Since the introduction of SWL for the removal of stones, this procedure has been optimised, and new instruments were developed to increase usability by the urologists and to improve tolerability for the patient this higher treatment comfort includes less pain, no need for general anaesthesia, and treatment on an outpatient basis. One study indicates that the original instrument for SWL, the HM3 lithotripter, yields the best outcome regarding the stone-free rate 1 d after treatment and may still be better than newer instruments such as the LSP and SLX lithotripters. Therefore, they concluded that newer instruments are not always better. The SWL procedure is the most widely used method for managing renal and ureteral stones. However, treatment success rates depend on stone composition, size, and location, as well as instrument type and shock frequency [72].

2. Percutaneous Nephrolithotomy:

Percutaneous nephrolithotomy or nephrolithotripsy uses a small incision in the person's back to remove kidney stones. The surgeon puts a hollow tube into the kidney and a probe through the tube. In nephrolithotomy, the surgeon removes the stone through the tube. In nephrolithotripsy, he or she breaks the stone up and then removes the fragments of the stone through the tube. Sometimes a procedure called percutaneous nephrolithotomy is recommended to remove a stone. This treatment is often used when the stone is quite large or in a location that does not allow effective use of ESWL.
One advantage of percutaneous nephrolithotomy over ESWL is that the surgeon removes the stone fragments instead of relying on their natural passage from the kidney.

3. Ureteroscopic (URS) for removal of ureteral stones:

The ureteroscope can now be used to extract stones as high up as the kidney. Catheters that contain lasers and lithotripsy devices can break up large stones.
In addition to SWL, other interventional procedures such as URS have been developed and improved for removal of ureteral stones. The new generation of ureteroscopes are flexible, smaller in diameter, stiffer, and more durable, and have an improved tip deflection. Of the many laser systems that have been used for stone fragmentation, the holmiumyttrium- aluminum-garnet laser is today the gold standard for intracorporeal lithotripsy. Furthermore, there are new tools for stone extraction such as the Nitinol baskets. New flexible and semirigid ureteroscopes that deflect up to 270° allow stones in the lower pole to be treated successfully.

4. Open and laparoscopic treatment:

This is the most invasive treatment and is rarely performed these days. There is no place for open ureterolithotomy in properly equipped endourological centres and the elimination of this form of treatment should be the aim of all urologists. In open surgery for ureteral stones, the doctor makes a surgical cut to expose the ureter where the stone is located. Another cut is made in the ureter itself, and the stone is directly removed. Open surgery is reserved for highly complicated. Difficult cases most patients need about six weeks to recover after the operation.[71]

The medical and preventative treatment of urolithiasis:

An increase in fluid intake to around 2.5 L per day to maintain a urine volume of >2 L is the cornerstone of urolithiasis management for the majority of patients. Maintaining dilute urine will prevent nucleation of supersaturated calcium oxalate and other poorly soluble salts (such as calcium phosphate and cystine) in

the urine. Some caution must be used with regard to low carbohydrate, ketogenic (''Atkins'') diets, as they may promote urolithiasis. More extreme measures to combat obesity such as bariatric surgery predispose to hyperoxaluria and calcium oxalate stones. For hypercalciuria, alongside increasing fluid intake, efforts to restrict dietary sodium intake and minimal protein should be made. Patients should not be advised to restrict calcium intake, as this may lead to hyperoxaluria; a normal calcium intake should be advocated. Pharmacological therapy with thiazide diuretics reduces urinary calcium excretion and is generally both well tolerated and effective. Importantly, thiazide diuretic have persisting effects on urinary calcium, with no diminution over time. Indapamide, a thiazide-like diuretic, is as effective as thiazide diuretics such as hydrochlorothiazide and may be better tolerated. These diuretics should be used in conjunction with a low sodium intake and a high potassium diet or potassium citrate to prevent thiazide-induced hypokalaemia. Hypokalaemia can cause an intracellular acidosis Within proximal tubular cells with consequent hypocitraturia, increasing stone risk.Finally, loop diuretics (such as furosemide) promote hypercalciuria and should be avoided. Hypocitraturia may be treated with a reduction of dietary animal protein and the addition of potassium citrate supplements, which have been shown to reduce recurrence in both calcium and uric acid-containing stones. Orange juice and lemonade have been shown to increase urinary citrate, and observational studies report fewer stone recurrences in lemonade drinkers. Screening for primary hyperoxaluria should be considered in all cases of hyperoxaluria, given the fact that pyridoxine treatment may be beneficial to some of these patients. Primary hyperoxaluria is an autosomal recessive disorder, diagnosed by finding a urine oxalate >0.7 mmol/24 h. In cases where renal failure is already established, plasma oxalate may be measured. Hyperuricosuria may lead to uric acid calculi and microscopic urate crystals can act as a nidus for calcium oxalate stone formation within the kidney. Therefore dietary advice to limit animal protein is recommended, however diet alone has not been shown to prevent uric acid stones. Lowering uric acid excretion using allopurinol therapy is beneficial in reducing stone episodes. Dietary modification with reduced animal protein and alkalinisation of urine using potassium citrate are recommended. For struvite calculi, treatment of the underlying infection combined with removal of residual infected stone fragments is the treatment of choice.

TRADITIONAL MEDICINE MUCH BETTER THAN ALLOPATHIC DRUGS

A lot of allopathic medicine are available for treatment of urolithiasis but because of cost effective, side effect and chances of recurrence of stone it is always better to prefer herbal medicine. Near about 80% of population is depend on plant based medicine. Automatically people are also using that medicine to cure from disease. Now a days kidney stone is the disease is the common disease. And after allopathic treatment there is possibility of recurrence of stone which will be reduce by herbal treatment. With no side effect. Easily available, cost is also less, no pain so, it is always preferred method for treatment of kidney stone. Herbal medicines are also playing role like improving renal function, by showing diuretic action , analgesic and anti-inflammatory action.[68]

Table 1: Diuretic and anti-urolithiatic plants. [9,10]

Sr. No.	Botanical name	Common name	Part Used
1	*Boerhaavia diffusa*	Punarnava	Roots
2	*Citrus limon*	Lemon tree	Fruit juice
3	*Crataeva nurvala*	Varuna	Bark
4	*Betula alba*	White birch	Infusion of leaves and buds
5	*Daucus carota*	Carrot	Root
6	*Fragaria vesca*	Wild strawberry	Leaves
7	*Physalls alkekengl*	Winter cherry	Fresh or dry berries
8	*Rubia tinctorum*	Madder	Decoction or root powder
9	*Tribulus terrestris*	Gokhru	Fruit
10	*Saxifrage granulate*	Saxifrage	Infusion or decoction of plant
11	*Spergulana rubra dietrich*	Sand spurry	Decoction of plant
12	*Filipendula ulmaria*	Meadosweet	Infusion of flowers
13	*Orthosiphon stamineus*	Javatea	Leaves
14	*Phyllanthus niruri*	Bahupatra	Whole plant
15	*Sonchus arvensis*	Sahadevi-bari	Leaves & roots, milky juice

India has great diversity of medicinal plants from ancient thousands of years .people are using plants to cure many diseases. Some are the medicinal plants with their local name are given which are found in Maharashtra region having activity against kidney stone disease are listed below.

Table 2: List of plants having Antiurolithiatic activity used in Maharashtra-

Scientific Name	Part used	Common name	Uses	Reference
Family – Acanthaceae				
Barleria prionitis	Roots	Koranti	DI	6,67
Adhatoda Vasica Medic	Whole part	Adulsa		67
Ecbolium viride	Leaf , Roots	Dhaktadulsa, Ranboli, Udajat	DI	5,7
Hygrophila auriculata	Roots	Talimkhana	DI	8,9,67
Family- Acoraceae				
Acorus calamus	Root	Vekhand, Sweet flag	DI	10
Family- Aizoaceae				
Trianthema portulacastrum	Leaf, Root	Pandhari –ghentuli,Sabuni	AG	11
Family- Amaranthaceae				
Amaranthus caudatus L.	Leaves	Rajgira	DI	9,67
Amaranthus viridis L.	All parts	Chawali	DI	9,67
Amaranthus spinosa L.	Stem	Kathe math	DI	9,67
Achyranthus aspera	Leaf, Root, Stem	Aghada,chichra,chirchira	DI, AG	5,67
Aerva javanica	Leaf, Root, Stem	Bui	DI	5
Aerva lanata	Leaf	Kapurmadhuri	DI,LL	9,67
Beta vulgaris	Root	Beet	AO	1,67
Celosia argentia	Root	Kurdu, morachendya, Garkha	AO	12,67
Chenopodium album	Leaf	Chakvat,bathuwa	AG	13,67
Nothosaerva brachiata	Root	Gael phal,madana	DI,LL,LT	14
Family- Amaryllidaceae				
Allium cepa	Bulb	Onion	DI,LT	15
Allium sativum	Bulb	Garlic	AG,AO	16
Family- Anacardiaceae				
Rhus succedanea	Fruit	Kakadshingi	AO	17
Spondias axillaris	Fruit	Ambada	NF	17
Family- Apiaceae				
Ammi visnaga	Bark,leaf,fruit	Khellin	DI,LL.LT	18
Ammi majus	Whole plant	Lace flower	DI	19
Apium graveolens	Root	Ajmod ajwain ka patta	DI,LL	20,21,22
Carum carvi	Fruit	Shahajire	DI	23
Centella asiatica	Leaf	Mandookparni	AO	24
Coriandrum sativum	Leaf	Dhane	DI	25
Dacus carota	Root	Gajar	NF	67
Foeniculum vulgare	Fruit	Kothimbir	DI	26,67
Pimpinella anisum	Fruit	Barisauf	DI,LL	27
Petroselinum sativum	Leaf ,Root	Ova	DI,LL	28,29
Family- Apocynaceae				
Holarrhena antidysenterica	Root, stem	Kuda	LL	30,31
Holarhena pubescens wall.	Stem ,Bark	Pandhara kuda	DI	67
Hemidesmus indicus	Leaf	Anantmul	AI	32,67,68
Ichnocarpus frutescens	Root	Kate bhovari	LL	33
Family- Arecaceae				
Borassus flabellifer	Bud	Tad	DI	34
Cocos nucifera	Fruit water	Coconut	AG,AO	35
Serenoarapens	Fruit	Palm	DI,AO	36
Family- Asparagaceae				
Asparagus racemosus	Root	Shatavari	LL,DI	37,67
Drimia indica	Bulb	Rankanda	DI	38
Family- Asteraceae				
Acmella oleracea	Whole plant	Akkalkara	DI	39
Ageratum conyzoides	Root	Ghanera	LL	40
Calendula officinalis	Leaf, flower	Zendu	DI	41
Tridax procumbens	Leaf	Kambarmodi	LL	9,67
Xanthium stramonium	Root	Shankeshwar	DI	11
Family- Bombaceae				
Bombax ceiba	Bark	Sayari	LL	1,42,67

Family- Boraginaceae				
Cordia grandis	Fruit	Bhokar	NF	68
Rotula aquitica	Root, stem	Pashanbhed	DI	43
Family- Brassicaceae				
Raphanus sativus	Bark,leaf ,root	Mula, raddish	LL,DI	44,45,67
Family- Bromeliaceae				
Ananas comosus	Fruit	Ananas	DI	46
Family- Burseraceae				
Commiphera mukul	Gum	Gugal	NF	67
Family- Cactaceae				
Opuntia ficus indica	Dry fluid	Nivdung	DI,LL	47
Family- Caesalpiniaceae				
Cassia auriculata	Leaf	Tarvad	AO	48
Cassia fistula	Fruit	Bahava	LL	48
Family- Cannabaceae				
Cannabis sativum	Fruit	Ganja	AG	49
Family- Combretaceae				
Terminalia arjuna	Bark	Arjun	AG	50,67
Family- Compisitae				
Taraxacum officinalis	Leaf, root	Tuki phool	DI,LL	51
Family- Chinopodiaceae				
Chinopodium album L.	Whole part	Chandan batwa	DI	67
Family- Convovulaceae				
Argyreia nervosa	Leaf	Samudrashok	AG	52
Family- Costaceae				
Costus igneus	Root	Insulin plant	DI,LL	53
Family- Crassulaceae				
Bryophyllum pinnatum	Leaf	Panfuti	DI,LL	54,67
Kalanchoe pinnatum	Leaf	Panphuti	DI,LL	55
Family- Cucurbitaceae				
Benincasa hispida	Fruit	Kohla	DI	54
Citrullus colocynthus	Whole plant	Bitter apple	AO	55
Citrullus lanatus	Whole plant	Watermelon	AO	56
Coccinia grandis	Root	Tondale	AO	57,58
Cucumis sativus L.	Seed	Kakadi	DI	59
Lagenaria siceraria	Fruit	Doodhi bhopla	DI,LL	5,67
Momordica dioica	Seed	Kartuli	AG	60,61,67
Family- Cyperaceae				
Cyperus rotundus	Rhizome	Nagarmotha	DI,LL	62
Family- Euphorbiaceae				
Acalypha indica	Leaf	Haritmanjiri	DI	63
Euphoria hirata	Whole plant	Doodhi	DI	64
Euphoria neriifolia	Whole plant	Sabarkand	DI	65
Phyllanthus amarus	Whole part	Bhui-awala	DI	66
Family-Fabaceae				
Abrus precatorius	Leaf, seed	Gunj	DI,LT	5
Butea monosperma	Bark,seed,leaf	Palas	DI	67
Tephrosia purpurea	Root, Leaf	Unhali	DI	68
Family-Lauraceae				
Cinnamonum verum	Leaf	Dalchini	AO	5
Family- Lytheraceae				
Lawsonia innermis	Bark, root	Mehandi	AG	67
Punica grantum	Fruit	Pomegranate	DI	67
Family- Malvaceae				
Abelmoschus moschatus	Leaf	Van bhendi	DI,LL	2
Family- Meliaceae				
Melia azadirachta	Leaf	Kadu-nimb	DI,LT	1,67
Family-Moraceae				
Ficus carica	Fruit,leaf	Anjeer	*LL*	*5*
Family- Moringaceae				

Moringa oleifera	Fluid, leaf, seed	Amba	*DI,LL*	*1,2,67*
Family- Myrtaceae				
Syzygium aromaticum	Flower bud	Lavang	*AG*	*2*
Family-Nyctaginaceae				
Boerhavia diffusa	Root	Punarnava	*DI,LL*	*5*
Family- Papaveraceae				
Fumaria officinalis	Whole plant	Pitpapada	DI,LL	5
Family- Papilionoideae				
Derris trifoliate	Aerial part	Karanj vel	DI	5
Family- Pedaliaceae				
Pedalium murex	Fruit	Gokharu	LT	2
Family- Phyllanthaceae				
Phyllanthus emblica	Fruit	Avala	AG	5
Phyllanthus fraternus	Whole plant	bhuiamala	DI	5
Family- Pinaceae				
Cedrus deodara	Wood	Devdar	DI,LT	5
Pinus brutica	Fruit	Pine	LL	5
Family- Piperaceae				
Piper longum	Fruit	Kali miri	DI	2
Family—Poeceae				
Bambusa nutana	Shoot	Bamboo	DI	2
Cynodon dactylon	Rhizome	Durva	DI,LL	2,67
Hordeum vulgarae	Seed	Jav, barley	DI	5
Zea mays L.	Tassel	Maka	DI	67
Family- Polygonaceae				
Emex spinosa	Leaf	Devil's thorn	DI	5
Rheum emodi	Root	Revachini	DI	5
Family- Ranunculaceae				
Nigella sativa	Fruit, seed	Kalunji	DI	2
Family- Rosaceae				
Agrimonia eupatoria	Seed	Visarpushpa	DI	5
Prunus avium	Fruit	Meethi cheri	DI	5
Rosa canina	Fruit, leaf	Dog rose	DI	5
Family- Rubiaceae				1
Coffea Arabica	Seed	Coffee	DI	5
Hamelia patens	Root	Fire bush	DI	5
Rubia cordifolia	Root	Manjishta	DI	5
Family- Rutaceae				
Aegle marmelos	Leaf, root	Bel	LT	67
Citrus aurantiifolia	Fruit	kagadilimbu	DI	5
Citrus laptipes	Fruit	Khasi papeda	NF	2
Citrus limon	Fruit	Limbu	DI	2
Citrus sinensis	Fruit	Santri	AO	5
Ruta graveolens	Leaf	Satapa	DI	5
Family- Santalaceae				
Santalum album	Sandalwood	Chandan	DI	5
Family- Sapotaceae				
Mimusops elegni	Bark	Bakul	DI,LL	1,5
Family- Saxifragaceae				
Bergenia ligulata	Rhizome	Pashanbhedi	DI,LT	1,2
Family-Solanaceae				
Withania somnifera	Whole plant	Ashwagandha	DI	5
Key words- AG-Analgesic; AO-Antioxoidant; LT- Lithotriptic; LL- Litholytic; DI- Diuretic ; NF – Not found				

CONCLUSION

The present study shows a clear direction & knowledge of the native and the use of medicinal plants. Also this study aims to gather information on traditional medicinal plants of Maharashtra region. There are many advanced and modern treatment are available in the market but tribal & rural people are still

dependent on the plants. Many of these plants are easily available in their vicinity and are cost effective. Hence, 80% of world population depends on traditional remedies to cure various diseases. Plant based medicines are effective herbal alternative & mean of discovery of novel drug molecule for curing urolithiatic disorders, researchers can surely focus on discovering their value for human use.

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