1. INTRODUCTION

The medicinal and culinary values of various herbs have been recognized and appreciated by the people of almost every culture and every part of the globe since time immemorial. In the ancient cultures of India and China, the benefits of medicinal herbs were recognized and chronicled over thousands of years ago. In modern times, people in Western counties have grown appreciative of the healing properties of herbs. Antibiotic treatments as well as Indian herbal remedies are gaining popularity in Western countries. Today, more and more people in Western countries are opting for herbs and natural healing alternative medicine treatments as the side effects in such treatments are relatively negligible.\(^1\)

Research and development has been pivotal in the development of Indian herbs. Continuous high level research helped in developing many unique products particularly in those segments where modern system of medicine does not provide satisfactory solutions. Herbal medicine which may also be referred to as medicinal botany, herbology, botanical medicine or phytotherapy has been popular in ancient traditions from time immemorial. In India, ancient religious works of the Rigveda as well as books on the ancient art of ayurveda offer evidence of the fact that the powerful healing properties of herbs were well recognized by ancient Indian scholars. Today there are many herbs that contribute to healing and have been seen to possess great medicinal value and curative properties. For example there are specific herbs for treatment of depression, diabetes, anxiety and even weight loss. In modern times, herbs are also being used in applications like cosmetics. Ginseng (panax ginseng) activates the skin metabolism and blood flow, reduces keratinisation, moisturizes and softens, alleviates wrinkling and enhances skin whiteness. Grape seed extract (vitis vinifera) contains procyanidins with strong anti-oxidant and anti-degenerative activity useful in skin-lightening cosmetics. Chamomile is one of the most popular plant due to its soothing properties and the variety of uses. It has a soothing and anti-inflammatory effect on dry skin. Agrimony, Poplar, Privet, Elderberry, Horsetail, Everlasting, Beetlejuice/carmine, Running club moss, Marshmallow, Pennyroyal (mint plant), Rosemary, Thyme, Pigweed are some of the most commonly used plants to regenerate the skin, nourish it and soothe irritation.\(^2\)
The world health organization (WHO) estimated that more than 80% of the world population rely on traditional medical practices for primary health care needs. Modern medicines are not really useful to treat certain chronic diseases such as arthritis, leprosy, psoriasis, spondilytis and kidney stones. There is also a popular concern over toxicity and side effects of modern drugs. Herbal teas are also immensely popular and often such teas are believed to be infused with the healing properties of certain herbs. Green tea is useful for the treatment of cancer. Detox Tea is a blend of detox herbs including milk thistle, sarsparilla root, dandelion root, echinacea, and red clover that stimulate the liver, gallbladder and immune system. Chamomile Tea is helpful for people under stress. It also relieves bloating and indigestion. Ginger Tea soothes the digestive system. It is also used for nausea and has been used for arthritis due to its anti-inflammatory effects. Peppermint Tea is a digestive aid that helps to relieve bloating and indigestion. In the form of coated capsules, peppermint is also used to treat irritable bowel syndrome (IBS) and gallstones. Hawthor Berry Tea is a traditional heart tonic. It is an antioxidant that strengthens blood vessel walls and heart function and has a beneficial effect on cholesterol. One such plant is *Salvadora persica* (Salvadoraceae), a plant commonly used as a folklore medicine for Lithiasis, in the southern parts of India.

Lithiasis means formation of stones in the kidney. One of the major causes of acute and chronic renal failure is lithiasis (Stone formation) which includes both nephrolithiasis (stone formation in the kidney) and urolithiasis (stone formation in the ureter or bladder or both). Lithiasis is a male predominant disorder, with a recurrence rate of 70-80% in males and 47-60% in females. The mechanism of calcium oxalate renal calculi formation has attracted the attention of medical scientists because of its widespread clinical occurrence and the difficulty of treatment.
1.1. Physiochemical factors involved in stone formation

1.1.1. Supersaturation

The key process in the development of kidney stones is

- Supersaturation of salts including calcium oxalate, uric acid, cystine or xanthine carried in the urine.
- These salts can become extremely concentrated if the volume of urine is significantly reduced or if abnormally high amounts of crystal forming salts are present.
- When concentration levels reach the point at which the salts no longer dissolve, the salts form crystals.

1.1.2. Deficiencies in protective factors

Normally urine contains substances like magnesium, citrate, pyrophosphate, various proteins like nephrocalcin and enzymes like alkaline phosphatase and $N$-acetyl-$\beta$-glucosaminase that may protect against stone formation. These substances

- Allow salt in the urine to be at higher than normal concentrations without forming crystals.
- Prevent crystal formation.
- Coat the crystals and prevent them from sticking to the surface of kidney tubes.

1.1.3. Changes in the acidity of urine

Changes in the acid balance of the urine can affect stone formation.

- Uric acid and cystine stones thrive in acidic urine.
- Calcium phosphate and struvite stones (Ammonium Magnesium phosphate stones) thrive in alkaline.

1.1.4. Factors that bind crystals to the kidney tubules

Researchers are studying the cells lining the kidney tubules in order to understand how and why early crystals bind to the tubes long enough to form stones. Under investigation are elevated levels of substances that either cause crystals to stick to the tubes or deficiencies in those that prevent them from, sticking.
1.2. Types of kidney stones

The type of kidney stone depends on several factors that cause the precipitation of a crystal(s) which eventually either grows or aggregates into a stone. Urinary concentrations of calcium, oxalate, phosphate, uric acid and certain proteins are believed to promote the stone formation. Magnesium and citrate are believed to inhibit stone formation. Urinary protein nephrocalcin which is a glycoprotein is known to prevent stone formation. Other factors including urine pH (a measure of the urine acidity) and infections can also play critical roles.

The following is a list of the common types of stones

1. Calcium oxalate
2. Calcium phosphate
3. Uric acid stones
4. Struvite stones (ammonium magnesium phosphate stones)
5. Cystine stones
6. Crixivan (Indinavir) stones
7. Ammonium uricacid

1.2.1. Calcium oxalate stones

The most common types of kidney stones are composed predominantly of calcium oxalate. A smaller proportion is composed mainly of calcium phosphate. Many stones can be mixtures of calcium oxalate and phosphate, but usually oxalate will be predominant.

Many factors contribute to the development of calcium oxalate stones. Physicians perform urinary studies to analyze calcium, oxalate, uric acid, and citrate levels and for dietary levels of sodium and protein. There can be derangements in the concentrations of one or more of these urine components. These tests are necessary to guide treatment and dietary changes.

Calcium is absorbed from the gut and stored in bone. Body levels are in part regulated by the kidney. High urine concentrations of calcium can lead to stone formation. Causes of high urine calcium include a genetic disorder known as familial hypercalciuria, and also hyperparathyroidism, which is less common in young people.

The cause of hypercalciuria appears to be multifactorial. Typically, there is increased absorption of calcium from the diet and/or bones with subsequent increased
calcium excretion into the urine. Treatment is not directed at lowering calcium in the diet, or absorption from the gut. Instead, therapy is designed to decrease calcium concentrations in the urine.

High levels of oxalate in the urine (called hyperoxaluria) can result from genetic defects and also from diet. Certain oxalate containing foods such as chocolates (dark or milk) and berries contain large amounts of oxalate. People with moderate hyperoxaluria and without genetic defects are encouraged to avoid high oxalate containing foods and to eat foods containing calcium, such as dairy products. These people usually have dietary hyperoxaluria. It may be particularly beneficial to eat these calcium containing foods with meals. Since calcium binds oxalates contained in the food, it prevents the absorption of oxalate in the gut. The calcium-oxalate complex in the gut is non-absorbable and will be passed in the stool. In fact, stone formers on low calcium diets form more stones.

Another form of hyperoxaluria is enteric hyperoxaluria. People with this condition usually have digestive disorders such as Crohn's disease, ulcerative colitis, chronic pancreatitis, ileal bypass surgery for the treatment of obesity, or celiac sprue. These conditions are characterized by an inability to absorb fat (fat malabsorption) which leads to excess oxalate absorption. Treatment includes avoiding high oxalate foods and calcium with meals to bind oxalate in the gut to prevent its absorption.

A rare genetic disease is primary hyperoxaluria. This is an inherited disease characterized by kidney stone formation at a young age and very high urine oxalate levels. It is due to a liver gene defect. The only effective treatment is liver transplantation, so that affected people will receive a new liver with the normal gene. Dietary therapy is also encouraged but likely of little or no benefit. If liver transplant is delayed, the kidneys can be irreversibly damaged by massive crystal and stone deposition. If this happens, then a dual kidney and liver transplantation may be needed. Another rare cause of hyperoxaluria that must be considered is excessive ingestion of vitamin C. A typical calcium oxalate stone is given in Fig 1.1.
1.2.2. Calcium phosphate stones

Calcium phosphate stones are less common than calcium oxalate. It is common to find stones that contain both calcium oxalate and phosphate crystals. However, usually the stone is more than 50% calcium oxalate.

If a stone has a large content of calcium phosphate, then medical causes should be investigated. These include hyperparathyroidism and renal tubular acidosis (RTA). If the kidneys cannot eliminate acid produced by the body, then acidosis may ensue. Signs of an RTA include low serum bicarbonate, low urine citrate, and a urine pH above 5.5.

Patients with calcium phosphate stones are treated with citrate. They tend to have multiple recurrent stones and loss of bone density. Over time the kidneys can become calcified. This is known as nephrocalcinosis.

1.2.3. Uric acid stones

Uric acid stones form from high concentrations of uric acid in acidic (low pH) urine. At low pH uric acid precipitates, while at normal pH it dissolves. In contrast to calcium stones, this type of stone can be “dissolved” purely with medical treatment. The cause of the elevated urinary uric acid can be multifactorial including high protein diet and gout.

Treatment includes increasing the urine pH with potassium citrate (making the urine less acidic) in addition to increasing water intake. Citrate is converted to bicarbonate, which increases urine pH. Occasionally, allopurinol is used to decrease production of uric acid by the body.

High uric acid levels (Hyperuricosuria) in the urine can also lead to calcium stones. One reason may be that small uric acid crystals form, but this is not certain. These crystals would then form seeds on which calcium crystals can grow. Another possibility is that dissolved uric acid somehow causes calcium oxalate to precipitate into crystals and stones. In this setting (i.e. calcium stones with high urine uric acid levels) treatment can be with either allopurinol or occasionally potassium citrate. A
low purine diet is also an important aspect of prevention. A typical uric acid stone is given in Fig. 1.2.

1.2.4. Struvite (infectious) stones (ammonium magnesium phosphate stones)

Struvite stones can be one of the most troublesome for patient and physician. These are fast growing stones that grow to fill up the naturally occurring cavities in the kidney to take on a “staghorn” appearance. Staghorns most commonly, but not always, occur with urinary tract infections which lead to the precipitation of magnesium ammonium phosphate crystals. Hence these stones are also called as “infectious stones.” Non-infectious stones such as uric acid and cystine can also form large cavity filling staghorns.

These stones are difficult to remove and often require extensive surgery. The infection often gets deep into the stone where antibiotics cannot penetrate. Therefore, many people will have recurrent infections. Preventing recurrence involves not only full removal of the stone and infected fragments, but also effective sterilization of the urine and kidney with antibiotics. Unfortunately, the infection is often difficult to eradicate and stones often recur. Certain bacteria which cause an increase in urine pH are associated with these stones. A common cause is Proteus bacteria. These stones can cause significant damage to the involved kidney. Sometimes kidney removal prior to transplantation is necessary. A typical struvite stone is given in Fig. 1.3.
1.2.5. Cystine stones

Patients with cystinuria excrete abnormally high amounts of cystine into the urine. Cystine has poor solubility in urine and easily precipitates to form stones. This genetically caused disease leads to recurrent stones which can occur at a young age. Cystine is an amino acid which is used for making proteins. It contains sulfur which is the basis for preventive treatment. Drugs containing sulfur, penicillamine and captopril are used. These sulfur containing drugs will bind to the sulfur component of the cystine. As in other types of stones, raising urine volume is key to prevention of new stones or slowing growth of already existing stones. These stones are very hard and usually cannot be removed with lithotripsy. People with cystinuria can go on to develop chronic kidney infections and permanent kidney scarring. Like struvite or infectious stones, cystine stones can develop into large staghorn shaped stones which fill the cavities of the kidney. Although they are not made of calcium, they can be seen on X-ray, unlike pure uric acid stones which cannot be seen on X-ray. Stones can develop in children. Diagnosis is made by collecting urine for 24 h and measuring elevated urine cystine levels. Distinctive hexagonal crystals are classically seen on microscopic urine analysis.

1.2.6. Crixivan (indinavir) stones

Indinavir is an antiretroviral (protease inhibitor) medication used in the treatment of HIV infections. It easily precipitates to form crystals. People who take this drug are instructed to drink large amounts of water in order to create a large urine volume which helps to dissolve the medication in the urine. Diagnosis can be difficult. Many patients who come in with symptoms of kidney stones are likely to have a CT scan or an X-ray of the kidneys which may not show indinavir stones. Indinavir stones often are difficult to detect on these tests. However, they should be visible on an ultrasound. It is critical to differentiate these stones from uric acid stones which sometimes can also be difficult to detect. Uric acid stones are treated with citrate (alkali) therapy to raise urine pH (decrease acidity) while this same treatment could actually promote formation of crixivan stones. Physicians treating people with HIV and uric acid stones, should consider prescribing a medication other than indinavir if possible.
1.2.7. Ammonium uricacid

Ammonium uricacid stones are a very rary type of stones. These are usually seen in people with chronic intestinal disorders and frequent laxative users.

1.3. Symptoms

People with kidney stones experience a range of symptoms from none to severe pain. However, if it passes into the ureter, which is the tube connecting the kidney to the bladder, severe crippling pain can occur. As the stones moves from the kidney into the ureter towards the bladder, mild to severe pain can start in the back and move with the stone around to the side of the abdomen and then to the groin. Blood will appear in their urine. Passage of the stones in the urine leads to dramatic improvement in the pain. Large stones do not pass in the urine. Small stones will appear as gritty material in urine with blood.

1.4. Diagnosis

These tests include a urine studies to look for blood. In young otherwise healthy people with characteristic pain who should not otherwise have blood in their urine, this is often a good confirmatory test. More precise testing is done with either ultrasound, computerized tomographic (CT or CAT) scanning, and/or intravenous pyelogram (IVP). Physicians have multiple ways to look at kidney stones. The method often depends on the severity of the problem, the type of stone and the presence or absence of symptoms.

1.4.1. X-ray

Standard x-rays of the mid to lower abdomen will include the kidneys, ureter and bladder. X-rays work by passing low doses of radiation through the patient's body onto a photographic negative. When X-rays pass through soft body tissues, they hit the film and cause it to turn black (exposure). When a calcium stone or bone (which is also made of calcium) is present the X-ray cannot pass through and no exposure occurs. The stone(s) and bones will appear white. This technique can only detect stones which contain calcium. It will miss pure uricacid or indinavir stones. X-rays can be done quickly and cheaply and are a quick, inexpensive, and useful technique for monitoring growth of a kidney stone. X-ray of lower abdomen showing stones in the kidney are shown Fig 1.4.
1.4.2. Ultrasound

Ultrasound is performed by passing a probe over the kidneys, ureters and bladder. Sound waves are emitted from the probe and are integrated into an image of the urinary system that can be seen on a television screen. This test can detect both calcium and non-calcium types of stones. It often is not a good test to find a stone that is suddenly passing from the kidney through the ureter on its way to the bladder. This is the time when stones hurt the most and cause people to seek immediate emergency medical assistance. If the stone has been lodged in the ureter for some time, then it can detect obstruction. In this situation the ureter is abnormally dilated. It is used by some for routine monitoring of new stones or growth of old stones. It has the benefit of no radiation.
1.4.3. Computerized tomography (CT or CAT scan)

This is one of the best methods to detect kidney stones, especially when someone comes to the emergency room with severe pain (colic) due to a passing stone. It is more sensitive than ultrasound or X-ray. It is performed by placing the patient in an X-ray tube that creates several images of the kidneys, ureter and bladder. It can detect both calcium and non-calcium stone, although it may sometimes miss crixivan/indinavir stones. It is more expensive than an X-ray and requires more radiation. Since it scans many organs, it can sometimes detect non-stone causes of severe pain. Fig. 1.5 shows CT scan of kidney showing stone.

![CT scan of kidney showing stone](image)

1.4.4. Intravenous pyelogram (IVP)

This is one of the older techniques for detecting kidney stone and still sometimes used. A special dye is injected into a vein. Then X-rays are taken of mid to lower abdomen. If a stone is present, a filling defect will be seen on the X-ray images. It is very useful for detecting stones in the ureter, especially if not seen by CT scan. This sometimes happens when the ureter is dilated or obstructed but no stone is seen. One disadvantage is that the injected dye can cause allergic reactions, usually temporary kidney damage, and symptoms including nausea. If the patient has kidney disease other than stones, sometimes it is better to perform a retrograde
urogram. In this test, a catheter is placed into the bladder and the dye is injected to visualize the bladder and ureters for stones. This technique avoids absorption of the dye outside of the urinary tract.

Intravenous pyelogram of kidney is shown in Fig. 1.6.

![Intravenous pyelogram of kidney](image)

**Fig. 1.6** Intravenous pyelogram of kidney

### 1.4.5. CT urography

CT urography is a combination of CT and IVP. An injection of intravenous dye is given which outlines the parts of the kidney, ureters, and bladder where urine collects. The images are viewed with a CT scanner. Traditional CT images are also generated. This test is particularly helpful as a step in the evaluation for blood in the urine (hematuria). It can show causes other than just stones. It is particularly useful in the evaluation of a kidney (renal) diverticulum. This is a pouch that develops inside the kidney. Kidney stones and infections can form inside this pouch. It can also be associated with pain.
1.5. Treatment of kidney stones

Most small stones [less than 5 mm] move out of the body (pass) without the need for any treatment other than taking pain medicine and drinking enough fluids. The smaller a stone is, the more likely it is to pass on its own. About 9 out of every 10 stones smaller than 5 mm and about 5 out of every 10 stones 5 mm to 10 mm pass on their own. Only 1 or 2 out of every 10 kidney stones need more than home treatment.

The average time a stone takes to pass ranges between 1 and 3 weeks, and two-thirds of stones that pass on their own pass within 4 weeks of when the symptoms appeared. Not all kidney stones are diagnosed because of immediate symptoms. If the stone is not causing pain, it may be found out during a routine exam or an exam for another condition or disease.

1.5.1. Medical treatment

If the stone can pass on its own, the following treatments are prescribed.

1. Nonsteroidal anti-inflammatories (NSAIDS) to relieve the pain. Stronger pain medicines if needed.
2. Drinking enough fluids, about 8 to 10 glasses a day.
3. Alpha-blockers have been shown to help kidney stones pass more quickly with very few side effects.

If the pain is too severe, if the stones are blocking the urinary tract, or if there is an infection, the following treatments are suggested.
1.5.2. Extracorporeal shock wave lithotripsy (ESWL)

ESWL uses shock waves that pass easily through the body but are strong enough to break up a kidney stone. This is the most commonly used medical treatment for kidney stones. Extracorporeal shockwave lithotripsy is shown in Fig. 1.7.

![Fig. 1.7 Extracorporeal shock wave lithotripsy](image-url)
1.5.3. Ureteroscopy

The surgeon passes a very thin telescope tube (ureteroscope) up the urinary tract to the stone's location, where he or she uses instruments to remove the stone or break it up for easier removal. Occasionally, a small hollow tube (ureteral stent) placed in the ureter for a short time to keep it open and drain urine and any stone pieces. Ureteroscopy is often used for stones that have moved from the kidney to the ureter. Ureteroscopy is shown in Fig 1.8.

Fig. 1.8 Ureteroscopy

1.5.4. Percutaneous nephrolithotomy or nephrolithotripsy

The surgeon puts a narrow telescope into the kidney through a cut in the back. Then the stone is removed (lithotomy) or broken and removed (lithotripsy). This procedure may be used if ESWL does not work or the stone is a very large. Fig. 1.9 shows the percutaneous nephrolithotomy.
1.5.5. Open surgery

The surgeon makes a cut in the side or the belly to reach the kidneys and remove the stone. This treatment is rarely used.

The present-day medical management of lithiasis includes lithotripsy and surgical procedures. Unfortunately, these techniques do not correct the underlying risk factors. Therefore, continued medical supervision and therapy for preventing stone recurrence is mandatory. In addition, these techniques cause side effects such as hemorrhage, hypertension, tubular necrosis and subsequent fibrosis of the kidney. Hence, the search for antilithiatic drugs from natural sources has gained great potential. A large number of Indian medicinal plants have been used in the treatment of lithiasis and they were reported to be effective with no side effects. In this background an attempt has been made to evaluate the antilithiatic potential of *Salvadora persica*, a plant commonly used as a folklore medicine for lithiasis in southern parts of India. Relevant scientifically proven data supporting this claim were lacking, and hence a detailed study using the leaves of this plant was planned.

Despite the popularity of traditional medicines, scientific research on safety and efficacy is limited. However, documented fatalities and severe illness due to heavy metal poisoning are increasingly recognized to be associated with traditional medicine use. As society becomes more globalized, it is imperative for pharmacists and health
care providers to learn about the safety of traditional medical practices.\textsuperscript{15} It has been reported that heavy metals like cadmium, chromium, copper and zinc causing depletion in protein level in different tissues/organs of experimental animals. Intake of these heavy metals in human being could not be acceptable from any mode.\textsuperscript{16, 17}. Consequently, analysis of heavy metal contents in leaves of \textit{Salvadora persica} has been accomplished.

1.6. Plant profile

The \textit{Salvadora persica} (Salvadoraceae) tree derives its Persian name, \textit{Darakht-e-miswak} or tooth brush tree, from the fact that wood is much employed for the manufacturers of tooth brush. \textit{Salvadora persica} is a large, well-branched evergreen shrub or small tree having soft whitish yellow wood, bark is of old stems rugose, branches are numerous, drooping, glabrous, terete, finely striate, shining, and almost white. It is found in the dry and arid regions of India, and on saline lands and in coastal regions just above the high water mark. The trees readily regenerate from seeds and coppice well\textsuperscript{18}. The whole plant of \textit{Salvadora persica} is given as photograph in Fig. 1.10. Stems of \textit{Salvadora persica} used as toothbrush in North India and Africa is shown in Fig 1.11. Leaves and root of \textit{Salvadora persica} are shown in Fig 1.12.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Fig_1.10_Salvadora_persica_Whole_plant}
\caption{Fig. 1.10 \textit{Salvadora persica} (Whole plant)}
\end{figure}
Fig. 1.11 Stems of *Salvadora persica* used as toothbrush in North India and Africa.

Fig. 1.12 Leaves and root of *Salvadora persica*
1.6.1. Geographical distribution

It is widely distributed in the drier parts of India, Baluchistan, and Ceylon and in the dry regions of West Asia and Egypt.

1.6.2. Vernacular names of *Salvadora persica* (L) Garcin

<table>
<thead>
<tr>
<th>Arabic Name(s)</th>
<th>Arak, Miswak, Souwak, Seawak, Lushlush</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common Name(s)</td>
<td>Toothbrush tree</td>
</tr>
<tr>
<td>Tamil</td>
<td>Kalawa, Karkol, Perungoli, Ughaiputti</td>
</tr>
<tr>
<td>Hindi</td>
<td>Jhak, Kharjal</td>
</tr>
<tr>
<td>Telegu</td>
<td>Ghunia, Varagoga</td>
</tr>
<tr>
<td>Karnataka</td>
<td>Goni-mara</td>
</tr>
</tbody>
</table>

1.6.3. Scientific classification

- **Kingdom**: Plantae
- **Division**: Magnoliphyta
- **Class**: Magnoliopsida
- **Order**: Brassicales
- **Family**: Salvadoraceae
- **Genus**: Salvadora
- **Species**: Persica, oleoides

**Synonyms:**

*Salvadora wightiana* planchon ex thwaites

*Salvadora indica* wight

*Salvadora koenigiarn*

1.6.4. Macroscopical characters

Bark is dull grey or grey-white, deeply cracked, and leaves are variable in shape - elliptic-ovate or ovate-lanceolate - somewhat fleshy.

Leaves are somewhat fleshy, glaucous, 3.8-6.3 cm in size, elliptic-lanceolate or ovate, obtuse, and often mucronate at the apex, the base is usually acute, less commonly rounded, main nerves are in 5-6 pairs, and the petioles 1.3-2.2 cm long and glabrous. Leaves, opposite, oblong-elliptic to almost circular, 3 x 7 cm, light to
dark green, rather fleshy when young, brittle when mature, sometimes with wartlike glandular dots and dense, rather loose hairs; apex broadly tapering to rounded, sharp-tipped; base broadly tapering; margin entire. Leaves of *Salvadora persica* is shown in Fig 1.13.

![Fig. 1.13 Leaves of *Salvadora persica*](image)

The flowers are pedicellate greenish yellow in color, in axillary and terminal compound lax panicles 5-12.5 cm long, numerous in the upper axils, pedicels 1.5-3 mm long, bracts beneath the pedicels, ovate and very caduceus. *Salvadora persica* stem branches with flowers and fruits are shown in Fig. 1.14.
Fig. 1.14 *Salvadora persica* stem branches with flowers and fruits

Calyx is 1.25 mm long, glabrous, cleft half-way down, lobes rounded. Corolla is very thin, 3 mm long, deeply cleft, persistent, lobes are 2.5 mm long, oblong, obtuse, and much reflexed. Stamens are shorter than corolla, but exserted, owing to the corolla lobes being reflexed.
Fruit, a drupe, 2.5-5 mm in diameter. Drupe is 3 mm in diameter, globose, smooth and becomes red when ripe. Fruits of *Salvadora persica* are shown in Fig 1.15.

![Fruits of *Salvadora persica*](image)

**Fig. 1.15** Fruits of *Salvadora persica*