LITERATURE REVIEW

1. Knoll T, (2007) found that some surgical procedures have been widely adopted for removal of stone. The stones are failed to pass spontaneously in a reasonable time can be treated by minimally invasive surgical procedures such as extracorporeal shock wave lithotripsy (ESWL), ureteroscopy and percutaneous nephrolithotomy (PNL).

2. Hall PM, (2009) identified the mechanism of stone formation and outlined treatment for prevention of kidney stone recurrences. He also found factors that promote stone formation. Those factors include low daily volumes, saturation of the urine with calcium, oxalate, calcium phosphate, uric acid or cystine, acidic urine and bacterial infection. Most of stones are composed of calcium oxalate or calcium phosphate. Alkalinization of urine may help in the prevention of uric acid stones and cystine stones.

3. Wasserstein AG, (2005) explained the mechanism, promoters and inhibitors of nephrolithiasis. In the pathogenesis, the different stages are involved such as, saturation, crystallization, mode of stone growth and sites of crystal growth.

4. Sarica K et al., (2008) found that the effect of being overweight on stone-forming risk factors. In both sexes, being overweight might be associated with the risk factor of stone formation owing to alteration in urine composition.

5. Bailly GG et al., (2000) examined the effects of dietary fat on the urinary risk factors of calcium stone disease. Mean daily dietary fat intake was 105.6 g and 78.1 g in men and women respectively. There is no relationship between in total fat intake and urine volume, pH and excretion of calcium, citrate, oxalate, magnesium and uric acid in men and only weak association between fat intake and uric acid in women. They concluded that the dietary fat does not have any significant effect on the risk factors of calcium stone disease.

6. Goldfarb DS et al., (1999) explained the role of beverages, diet on kidney stones and prevention of kidney stones. Apple juice consumption was directly correlated with stone formation. The protective effects of coffee, tea and wine were found. They also found that the ingestion of grapefruit juice more once per week increased risk of kidney stones. Orange juice and dilute lemon juice are associated with either no change or an increase in urinary oxalate.
7. Joseph KC et al., (2005) reviewed that British isles, Scandinavian countries, Northern Australia, Central Europe, Northern India, Pakistan and Mediterranean countries. India has high incidence of calculi especially in Gujarat, Rajasthan, Punjab, and Madhya Pradesh. In India, 12% of the population is expected to have urinary stones, out of which 50% may end up with kidney or renal damage. Also, nearly 15% of the population of northern India suffers from kidney stones. Saurashtra region, Gujarat has higher prevalence of urolithiasis.

8. Bhatt PA et al., (2008) reviewed that Western India (Saurashtra region of Gujarat state), which comes under the region of low rain fall, hot climate and increased salinity of ground water, is a highly urinary stone disease prone area.

9. Pak CYC, (1998) found that kidney stones developed by various metabolic and environmental-nutritional factors including hypercalciuria, hyperoxaluria, hyperuricosuria, hypercitraturia, under urinary acidity, cystinuria and low urine volume. For the treatment of urolithiasis, There are different drugs are used such as thiazide diuretics, potassium citrate, low calcium diet for hypercalciuria, allopurinol for hyperuricosuria, magnesium citrate for hyperoxaluria, chelating agents for cystinuria and antibiotics for infection stones.

10. Basavaraj DR et al., (2007) explained the role of urinary kidney stone inhibitors and promoters in the pathogenesis of calcium containing renal stones. Calcium, sodium, oxalate, urate, cystine, low urinary pH, low urinary flow are promoting factors that promote urinary stones. Inhibiting factors include inorganic (citrate, magnesium, pyrophosphate) and organic (Tamm-Horsfall protein, urinary prothrombin fragment 1, inter α inhibitor, glycosaminoglycans, osteopontin, renal lithostathine, bikunin, calgranulin, high urine flow) that inhibit urinary stones.

11. Sas DJ et al., (2010) reported an increasing incidence of kidney stones in children evaluated in the emergency department. Between 1970 and 2000, a slight increase in the incidence of nephrolithiasis is reported in boys aged 0 to 19 years. An initial increase in nephrolithiasis incidence followed by a gradual decline is reported for girls in the same age range during the same period.

12. Curhan GC et al., (1997) found that high intake of dietary calcium decreases the risk for symptomatic kidney stones, whereas intake of supplemental calcium may increase risk of kidney stones. Dietary calcium reduces the absorption of oxalate. Both sucrose intake and sodium intake increased risk for kidney stones.
while potassium intake and fluid intake inversely related to risk of kidney stones.

13. **Tiselius HG, (2000)** explained stone incidence and prevalence. The average lifetime risk of stone formation has been reported to be in range of 5-10% with a considerable geographical variation.

14. **Choi EM et al., (2004)** revealed that methanolic extract of fruit of *Foeniculum vulgare* possesses antinociceptive effect after administration of 200 mg/kg orally, in hot plate method of antinociception and it blocked both the neurogenic and inflammatory pain by inhibiting the pain receptors at the inflammation site. It was indicated by significantly reduced hot plate thermal stimulation. *Foeniculum vulgare* extract at dose of 200 mg/kg caused a significant inhibition of paw edema (69%) as compared to the control group 3 h after carageenan injection. *Foeniculum vulgare* extract also inhibited by approximately 70% the ear-edema induced by arachidonic acid in mice. According to these results, they suggested that *Foeniculum vulgare* may act on both the cyclooxygenase and lipoxygenase pathways.

15. **Ozbek H, (2005)** also proved that the *Foeniculum vulgare* essential oil has anti-inflammatory activity by inhibition of carageenan (Phlogistic agent) induced paw edema in rats.

16. **Birdane FM et al., (2007)** found that the aqueous extract of *Foeniculum vulgae* fruit possessed better antiulcer properties by inhibiting ethanol induced gastric mucosal injury. At 300 mg/kg dose, it exerts better mucoprotective effect comparable with standard drugs famotidine. The preliminary phytochemical screening of *Foeniculum vulgare* showed the presence of various chemical constituents like, volatile oil, flavonoids (rutin, quercetin and kaempferol glycosides), coumarins sterols and sugars. Flavonoids, sterols, tannins, and coumarins of some plants have antiulcer activity. Therefore, the presence of flavonoid content and other bioactive compounds in *Foeniculum vulgare* may be associated with the ulcer preventing action.

17. **Ozbek H et al., (2004)** found out that *Foeniculum vulgare* essential oil at a dose of 0.3 ml/kg i.p. (all three times a week for seven weeks), significantly prevented Carbon tetrachloride induced liver fibrosis in rats. Biochemical parameters like serum aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase and bilirubin were within normal range or decreased as
compared to control group. *Foeniculum vulgare* essential oil prevents the development of chronic liver damage was also suggested by histopathological findings.

18. **Ozbek H et al., (2006)** proved the hepatoprotective activity of *Foeniculum vulgare* (fennel) fixed oil using a carbon tetrachloride-induced liver fibrosis model in rats. The hepatotoxicity produced by chronic carbon tetrachloride administration was not significantly inhibited by *Foeniculum vulgare* fixed oil with evidence of decreased levels of various biochemical parameters such as, serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), bilirubin and also histopathological findings.

19. **Ostad SN et al., (2001)** established the effects of fennel essential oil on the uterine contraction in rat. For assessment of pharmacological effects on the isolated rat uterus, prostaglandin E$_2$ (PGE$_2$) and oxytocin were given to induce muscle contraction. Administration of different doses of fennel essential oil reduced the intensity of PGE$_2$ and oxytocin induced contractions significantly (10 and 20 μg/ml for PGE$_2$ and 25 and 50 μg/ml for oxytocin, respectively). Fennel essential oil also reduced the frequency of contractions induced by PGE$_2$ but not with oxytocin.

20. **Tognolini M et al., (2007)** found that the essential oil of *Foeniculum vulgare* and anethole tested in rat aorta with or without endothelium, displayed comparable NO-independent vasorelaxant activity at antiplatelet concentrations. *In vivo*, both *F. vulgare* essential oil and anethole orally administered in a subacute treatment to mice (30 mg/kg/day for 5 days) showed significant antithrombotic activity preventing the paralysis induced by collagen-epinephrine intravenous injection (70% and 83% protection, respectively). At the antithrombotic dosage they were free from prohemorrhagic side effect at variance with acetylsalicylic acid (aspirin) used as reference drug. Anethole and Foeniculum vulgare did not cause cytotoxicity when incubated for 30 min upto 300 μg/ml in platelet viability test.

21. **Agarwal R et al., (2008)** established the intraocular pressure (IOP) lowering effect of single drop application of *Foeniculum vulgare* aqueous extract in rabbits with normal IOP as well as in rabbits with experimental increase in intraocular pressure. The experimental increase in IOP was achieved by water loading and topical steroid application. Single drop application of all three
concentrations of *Foeniculum vulgare* extract (0.3, 0.6 and 1.2%) resulted in a significant fall in intraocular pressure in rabbits with normal intraocular pressure. Both concentrations (0.6 and 1.2%) showed more significant action as compared to 0.3% concentration of *Foeniculum vulgare* extract. According to this result, they concluded that the *Foeniculum vulgare* aqueous extract showed significant oculohypotensive activity and the maximum intraocular pressure lowering effect as compared to timolol.

22. **Oka Y, (2000)** found that nematicidal activity under field conditions with other nematode species and in other types of soil. The essential oils may disrupt the cell membrane of the nematode and change its permeability.

23. **Pai MBH et al., (2010)** proved that the essential oil extract of *Foeniculum vulgare* elicited in vitro antifungal activity. This activity was carried out by disc diffusion method. *Foeniculum vulgare* showed antifungal activity at the end of 48 hours. So, they concluded that the essential oil of *Foeniculum vulgare* had antifungal activity against Candida.

24. **Shukla HS et al., (1989)** carried out the antiviral activity of the essential oil of *Foeniculum vulgare* against potato virus X (PVX), tobacco mosaic virus (TMV), tobacco ring spot virus (TRSV) on the hypersensitive host *chenopodium amaranticolor*.

25. **Ebeed NM et al., (2010)** found that fennel extract may have slight genotoxic effects on mice rather than Drosophila. In addition, the biochemical, chromosomal aberrations in mice bone marrow as well as aneuploidy and chromosomal aberration test in Drosophila male germ-lines confirmed the antimutagenic effects of fennel extract against Mitomycin C and colchicine induced mutations. However, the pre and post treatment analysis exhibited that hot water crude extract of fennel may contain some compounds that can act as dis-antimutagen and some compounds can act as bio-antimutagen. Random amplified polymorphism of DNA (RAPD) indicated the effect of fennel extract to induce DNA changes as confirmed by biochemical assays.

26. **Kaur GJ et al., (2009)** found out the aqueous (hot water) and organic (acetone) seed extracts of *Foeniculum vulgare* showed considerably good antibacterial activity against all the bacteria except Klebsiella pneumoniae and one strain of *Pseudomonas aeruginosa*. The antibacterial activity was determined by agar diffusion method. Seed extract showed antibacterial activity against different
bacterial strains viz. Enterococcus faecalis, Staphylococcus aureus, Escherichia coli, Pseudomonas aeruginosa 2, Salmonella typhi, Salmonella typhimurium 2 and Shigella flexneri. Aqueous and acetone *Foeniculum vulgare* seed extracts were better or equally effective against some of the bacteria as compared to standard antibiotics viz. ampicillin, cefixime, chloramphenicol, co-trimoxazole, gentamicin, imipenem, pipericillin/tazobactam and tobramycin.

27. **Pradhan M et al., (2008)** found cytoprotection activity of *Foeniculum vulgare* and *Helicteres isora* in cultured human blood lymphocytes and antitumor activity against B16F10 melanoma cell line.

28. **Javidnia K et al., (2003)** carried out a placebo controlled study of Fennel (fruits of *Foeniculum vulgare*) extract. They evaluated the clinical response of idiopathic hirsutism to topical Fennel extract. The efficacy of treatment with the cream containing 2% Fennel is better than the cream containing 1% Fennel and these two were more potent than placebo. The Fennel extract is safe with no adverse effect in topical application and can be re-introduced into modern medical usage in the treatment of hirsutism.

29. **Alexandrovich I et al., (2003)** carried out clinical study of the fennel seed oil emulsion in infantile colic patients. They proved that fennel seed oil emulsion was superior to placebo in decreasing intensity of infantile colic.

30. **Venkataranganna MV et al., (2002)** investigated the antispasmodic activity of SJ-200 (Himcospaz), an herbal preparation on gastrointestinal smooth muscles of rabbits, rats, mice and guinea pig. SJ-200, an herbal preparation contains *Zingiber officinale*, Zingiberaceae (rhizome), *Apium graveolens*, Apiaceae (fruit) and *Foeniculum vulgare*, Apiaceae (fruit). SJ-200 at a concentration of 40 μg/ml and 80 μg/ml dose-dependently inhibited spontaneous contraction of rat and rabbit colon. A dose of 80 μg/ml of bath concentration completely blocked spontaneous contraction. It also inhibited oxytocin induced contraction of rat uterus. Oral administration of SJ-200 dose-dependently reduced gastric emptying in rats and intestinal transit in mice. It also dose-dependently inhibited acetylcholine, barium chloride and histamine induced contraction of guinea pig ileum.

31. **Yasar F et al., (2006)** found that antioxidant activity of *Cucumis melo* varieties. The *Cucumis melo* seedling respond to salt induced oxidative stress by
increasing both their enzymatic and non-enzymatic anti-oxidant defence mechanism.

32. **Iqbal S et al., (1999)** found that the diuretic activity of an indigenous herbal preparation in goats. The herbal mixture containing *Foeniculum vulgare, Cinchorium intybus, Cucumis melo* and *Cucumis sativus* used as diuretic as well as hypotensive preparation.

33. **Knoll T, (2010)** reviewed that urolithiasis is a common disease with increasing incidence and prevalence worldwide. Renal stones and their predominant chemical composition are depended on age, gender, climate, lifestyle and dietary choices. Some recent evidence suggested a primary interstitial apatite crystal formation (Randall plaque) that secondarily leads to calcium oxalate stone formation.

34. **Sharma AP et al., (2010)** explained the epidemiology of pediatric urolithiasis. Calcium oxalate is the most common stone worldwide and accounts for 60-90% of pediatric urolithiasis. Calcium phosphate stones accounts for 10-20%. Struvite constituents 1-18% of the stones in developed countries. Uric acid constituents 5-10%, cystine 1-5% and mixed or miscellaneous 4% of the pediatric stones. Some metabolic risk factors such as hypercalciuria, hyperoxaluria and hypocitraturia increase the risk of stone recurrence. Idiopathic stone disease has been reported to be more frequent in white Caucasians than in Africa.

35. **Bihl G et al., (2001)** explained recurrence renal stone disease. Renal stones are common in industrialized nations. They described some drugs that associated with renal stone formation. Loop diuretics, antacids, acetazolamide, glucocorticoids, theophylline and vitamins C and D promote calcium stone formation. Thiazides, salicylates, probenecid and allopurinol promote uric acid stone formation. Triamterene, acyclovir and indinavir precipitate into stones.

36. **Pais VM et al., (2006)** reported that xanthine calculi may occur in association with inborn metabolic disorders (hereditary xanthinuria or Lesch-Nyhan syndrome) and hyperuricemia.

37. **Hughes P, (2007)** reported the risk factors for the urolithiasis. These include age, sex, racial differences, stone type, seasonal variation, diet, and some diseases.