Chapter No. VII

SUMMARY & CONCLUSION

Page No. 106 - 114
Chapter VII

SUMMARY AND CONCLUSION

Summary :-

The present study was carried out on total 200 subjects including normal healthy controls and nephrolithiasis patients having age 22-45 years of both genders. The diagnosis of nephrolithiasis patient was done by physicians based on the clinical history, ultrasonography and laboratory investigations.

The patients were free from diseases like diabetes mellitus, thyroid impairment, renal failure, liver diseases, heart diseases, hypertension, bone diseases etc. The controls selected for the study were free from diseases like nephrolithiasis, diabetes mellitus, thyroid impairment, renal failure, liver diseases, heart diseases, hypertension, bone diseases etc.

The subjects were consists of group I, which includes 100 normal healthy controls and group II consists of 100 Nephrolithiasis patients.

The venous blood samples were obtained from the subjects during fasting state. The serum was separated and analyzed for urea, creatinine, uric acid, calcium, inorganic phosphorus, sodium, potassium, chloride, total proteins, albumin etc. The 24 hours urine samples were collected and analyzed for urea, creatinine, uric acid, calcium, inorganic phosphorus, magnesium, sodium,
potassium, chloride, citrate and oxalate. The physical characteristics, microscopy and culture-sensitivity of urine was also assessed. The results of all parameters were compared between normal healthy controls and nephrolithiasis patients.

In the present study no significant change (P>0.05) was observed in serum creatinine and urea levels in nephrolithiasis patients as compared to normal healthy controls. Microscopic examination of urine does not showed any pus cells and urine culture was sterile. This indicates that there is no any abnormalities in the kidney functioning and no obstructions in the renal system.

However in the present study the serum uric acid level was increased by 20.3% in nephrolithiasis patients. The increased serum uric acid levels in nephrolithiasis patients may be due to purine rich diet.

The levels of serum calcium and inorganic phosphorus were elevated by 6.6 % and 43.3 % respectively in nephrolithiasis patients as compared to normal healthy controls.

The significantly elevated levels of serum calcium and inorganic phosphorus indicates the high dietary intake of calcium, phosphorus and use of bore well water (having more hardness).

The elevated levels of serum calcium may be due to increased serum albumin concentration. As 40% of total calcium is bound to albumin, which may increased total calcium level.
There is no significant change in mean levels of serum sodium, potassium and chloride in nephrolithiasis patients as compared to normal healthy controls.

This indicates that there may not be any acid base disturbances in nephrolithiasis patients. The serum albumin levels were significantly elevated by 9.8% in nephrolithiasis patients whereas no significant change was observed in serum total proteins of nephrolithiasis patients as compared to normal healthy controls.

The non-significant change of serum total proteins in nephrolithiasis patients indicates that the nephrolithiasis patients may have normal nutritional status and synthesis as well as utilization of protein could have been normal. However significantly elevated albumin in nephrolithiasis patients may be due to genetic variation.

The urinary urea does not show any change in nephrolithiasis patients as compared to normal healthy controls.

The levels of urinary creatinine were decreased by 7% in nephrolithiasis patients, which shows partial obstruction in the renal flow.

The urinary uric acid level was significantly elevated by 82.6% in nephrolithiasis patients as compared to normal healthy controls. The elevated urinary uric acid may be due to high intake of sodium and purine rich diet.
The urinary oxalate was 68.93% higher in nephrolithiasis patients as compared to normal healthy controls. The elevated levels of oxalate in urine may be due to high intake of oxalate rich food stuffs such as dark green leafy vegetables, and high intake of tomatoes and ground nuts which causes higher absorption and excretion of oxalate in urine.

The urinary citrate excretion was significantly reduced by 61.9% in nephrolithiasis patients as compared to normal healthy control group. These reduced levels of citrate excretion may be either due to decreased levels of urinary magnesium or there may be imbalanced excretion of divalent cations, such as Ca++, Mg++ in urine.

The urinary magnesium excretion was decreased by 24.6% in nephrolithiasis patients when compared to normal healthy controls. This decreased urinary magnesium level in nephrolithiasis patients may be due to the imbalanced in the normal excretion mechanism.

The urinary excretion of calcium was doubly increased in nephrolithiasis patients as compared to normal healthy control group. The increased level of urinary calcium in patients of nephrolithiasis may be due to high intake of animal protein or sodium rich diet, which causes higher excretion of calcium through renal mechanisms and increases mobilization of calcium from bones. The high intake of lactose may be the cause of higher intestinal absorption of calcium and their excretion in urine.
The urinary inorganic phosphorus level was lower by 37.7% in nephrolithiasis patients as compared to normal healthy control group.

Generally, when serum phosphorus concentration increases, there is decrease in renal phosphorus reabsorption thereby increase of urinary phosphorus excretion. But this mechanism may be hampered in nephrolithiasis patients, as in present study phosphorus excretion was significantly decreased even though increased serum phosphorus concentration was found in nephrolithiasis patients as compared to control group.

The urinary sodium, excretion was significantly elevated by 22.67% in nephrolithiasis patients as compared to control group. It may be due to more intake of dietary salt, processed foodstuffs and animal protein by the nephrolithiasis patients as compared to control group.

The urinary chloride and potassium levels were significantly decreased by 33% and 25% respectively in nephrolithiasis patients as compared to control group. However it may be due to compensatory mechanism, to maintain normal electrolyte balance in the body (Acid – base balance).

Urine analysis revealed that there was no any abnormal constituents in nephrolithiasis patients and not much difference in the specific gravity of urine was observed in nephrolithiasis patients. The slight change in specific gravity may be due to less urine excretion in nephrolithiasis patients. The reduced excretion of urine may be due to low intake of fluid by the patients.
as compared to control group. The urine culture and sensitivity was sterile. It indicates that cause of renal calculi formation in the Marathwada region is not the infection.

In the urine microscopy crystals of calcium – oxalate was observed in nephrolithiasis patients which indicates that presence of calcium oxalate calculi which was confirmed by Stone analysis. The urinary pH of nephrolithiasis patients was lower than the control group but urine was neither highly acidic nor alkaline in nephrolithiasis patients. This indicates that the available pH of urine was much favourable for calcium – oxalate stone formation.

Conclusion :-

The existance of "Stone belt" or geographical areas where cases of urinary calculi occur with unusual frequency has been recognized as an accepted fact. A relationship between geographical regions and the incidence of presence of urinary calculi exists, it may have impotent implications. However in the present study attempt was made to correlate the biochemical parameters in urine and serum of the patients having first episode of renal calculi and also the other factors which are known to be the risk factors such as climate, diet, water etc.

The specific dietarty therapy can be adjusted according to a metabolic evaluation is more effective than non specific dietary recommendatons in preventing the formation of renal stone in a future or second episode of renal stone formation.
In conclusion, our data reinforce the concept that hypercalciuria, hyperoxaluria, hyperuricosuria and hypernatraturia in nephrolithiasis patients can be prevented by suggesting the dietary modifications such as animal protein intake can be replace by plant protein. The adequate calcium and protein requirement is fulfilled by taking milk in a diet. Oxalate rich food can be avoided but dark green leafy vegetables are the rich sources of oxalate along with other minerals and vitamins, thus completely devoid of green leafy vegetables in diet, may leads to deficiencies of minerals and vitamins, which are important to maintain the health. Thus less consumption of green leafy vegetables like spinach, tomatoes and less salt and nuts are recommended. It maintains the health and prevents the excessive oxalate, calcium and uric acid excretion. Hypocitraturia, hypomagnesuria and hypophosphaturia are also found to be risk factos in nephrolithiasis patients. These are the inhibitors of the nephrolithiasis, thus to keep balance between inhibitors and stone forming salts excretion is important, which is achieved by reducing the intake of above said dietary component which are responsible for stone formation.

Intake of fluid should be adjusted season wise. In summer increase in fluid intake is useful and prevents the supersaturation of urine. As in summer fluid is lost through more perspiration along with insensible loss of fluid. Generally individuals have a habit to drink less water at night than the day time and hence there is more chances of supersaturation of urine during night, thus to create a habit of drinking water at bed time will be helpful to minimise the super saturation of urine and thereby crystal formation.
More exposure to sunlight is also risk for the nephrolithiasis. In hot climate region, synthesis of vitamin D increase and thus intestinal calcium absorption is enhanced which may lead to calculi formation. Hence more exposure to sunlight is a risk factor for nephrolithiasis. Thus more exposure to sunlight should be avoided.

Hardness of water is an important criteria for drinking water. The present study was carried out in Marathwada region, where the individuals with nephrolithiasis are using bore well (hard) water for drinking. Thus the hardness of water is one of the precipitating factor for nephrolithiasis along with above said factors in Marathwada region.

The multifactors come together and causes renal stone but it depends on individual to individual because the control group also selected from the same region, we should not ignore this fact, thus the biochemical changes occurs in nephrolithiasis patients not only due to dietary factors and environmental factors but it is also due to genetic variation (inheritance) and hence further study is required such as at the genetic level and at hormonal levels to find out the real cause.

Implications :-

The present study is clearly related to the use of preventive measures to reduce the risk of further development of nephrolithiasis and in individuals prone to nephrolithiasis. The simple preventive measures, which can
be advocated, are as follows:

- Substitution of animal proteins by plant proteins in the diet. Since animal proteins increases absorption of calcium, or limit the animal proteins in diet.

- Use of well-balanced nutrition or diet with consecutive high intake of fluids (> 2.5 lit/day) which decreases the risk of nephrolithiasis.

- Prevention of diet rich in oxalate, sodium, processed food stuffs, refined carbohydrates especially lactose, the bore-well water (water having high hardness) and excessive vitamin C.

- Prevent inadequate intake of calcium, as inadequate calcium intake causes high excretion of oxalate, which is risk factor for nephrolithiasis.

- Increase intake of dietary fiber, as dietary fibers are useful in prevention of excessive absorption of dietary constituents and minerals.

These preventive measures can help to a considerable extent in the regulation of minerals metabolism such as calcium, phosphorus, magnesium, sodium, potassium and chloride. This will certainly check the homeostasis of the mineral metabolism along with inhibitors and stimulators of nephrolithiasis and thereby reduces the risk of nephrolithiasis in future.
Untreated calculi caused kidney damage