

A Clinical Study on the Efficiency of Homoeopathic Medicines in the Treatment of Gout with an Evaluation Based on Serum Uric Acid Level

Dr. Shibina K.

Department of Practice of Medicine, MNR Homoeopathic Medical College (KNRUHS),
Narasapur Road, Sangareddy, Hyderabad, Telangana

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ABSTRACT

Gout was first described by "Hippocrates". The term 'gout' is derived from Latin word 'gutta' and French word 'gote', both meaning a drop of liquid. Hundred years ago the gout was thought to be caused by drops of viscous humors that seeped from blood into the joints. It has been known as "the disease of kings" because of its association with the kind of over indulgence in food and wine, which only the rich and powerful could afford. Among the lengthy list of metabolic disease, gout is by far the most common one arising either from the enzymatic malfunctioning of purine synthesis or metabolism resulting in the over production of uric acid or else its poor clearance due to faulty performance of the kidneys. Gout is the most common inflammatory arthritis worldwide; gout and hyperuricemia are on the rise owing mainly to dietary changes, obesity, and metabolic syndrome and increased age. In the past two decades the prevalence of gout has doubled owing mostly to changes in diet.

Key words: Gout, Homoeopathy, Serum Uric Acid Level, Tophi, podagra

INTRODUCTION

Biochemical And Physiological

Implication Of Gout

The biochemical factors behind Gouty arthritis are primarily due to inherent defect in purine metabolic or catabolic pathway and for physiological reason the blame is offered to impaired clearance of uric acid or urate crystals by the kidney. The inheritance of Hypoxanthine phospho ribosyl

transferase deficiency increases uric acid level in plasma which is related to X-linked recessive gene of which females are the usual carriers. Therefore males are often affected indicating the early onset of gouty arthritis. The enzyme Hypoxanthine phosphoribosyl transferase encoded on the long arm of X-chromosomes. (1)

Definition

Gout is a true crystal deposition disease. It can be defined as the pathological reaction of the joint or periarticular tissues to the presence of monosodium urate monohydrate (MSUM) crystals. The monosodium urate monohydrate crystals commonly deposited on peripheral connective tissues in and around the synovial joints, initially favouring lower rather than upper limbs and especially targeting the first metatarsophalangeal and small joints of feet and hands. As the crystal deposits slowly increase and enlarge there is progressive involvement of more proximal sites and the potential for cartilage and bone damage, and development of 'secondary' OA. MSUM crystals take months or years to grow to a detectable size, implying a long asymptomatic phase. (2)

Gout is a metabolic disease that mostly affects middle-aged to elderly men and postmenopausal women. It is due to increased body pool of urate with hyperuricemia. It presents with episodic acute and chronic arthritis caused by

deposition of monosodium urate monohydrate crystals in joints and connective tissue tophi and the risk for deposition in kidney interstitium or uric acid nephrolithiasis.(3)

Gout is an inflammatory response to the monosodium urate monohydrate crystals formed secondary to hyperuricemia. The major clinical manifestations are acute synovitis, chronic erosive and deforming arthritis, tophi, nephrolithiasis, and interstitial nephritis. Hyperuricemia is defined as a serum or plasma urate concentration >7.0 mg/dL in males and 6.0 mg/dL in females.(4)

EPIDEMIOLOGY

The prevalence of gout varies between populations but is approximately 1-2%, with a strong male predominance (> 5:1). It is the most common inflammatory arthritis in men and in older women.(2)

The National Health and Nutrition Examination Survey (2007-2008) states a new prevalence for gout and hyperuricaemia. The gout rates reported as 5.9 % among male and 2 % among female. Whereas prevalence rate of hyperuricemia was noted as 21.2 % for men and 21.6 % for women. This difference is largely due to age, because oestrogen hormones have a mild uricosuric effect. It is rare in children and young adults but affects 1.3 % of elderly patients. The higher chance of occurrence of tophaceous gout in elderly persons may also reflect an increased prevalence of diabetes, high rates of diuretic treatment for hypertension.(5)

In general, both developed and developing countries presented with increasing prevalence and incidence of gout in recent years. (6)

RISK FACTORS

Gender: It plays a key role. It has been found that men are likely to fall victim of gout syndrome much earlier and in more numbers as compared to women. Triggering factors for acute attack remain same for both men and women.

Lifestyle change: Certain conditions related to nutrition and lifestyle includes obesity, emotional stress, frequent episodes of dehydration, injury to a joint, very low calorie diet and moderate to heavy alcohol ingestion (particularly beer, which is rich in purine). Alcohol can reduce the excretion of uric acid by the kidney into urine, causing an increase of uric acid level in the body. A higher intake of added sugars or sweetened drinks leads to higher blood level of uric acid. Meat or seafood consumption increase the risk of gout attacks.

Medication: Medicine that may increase uric acid concentration include regular use of aspirin or niacin, diuretics, chemotherapeutic agents, immunosuppressants such as cyclosporine and medicines that are used to treat tuberculosis.

Medical conditions: Major illness, infection or certain medical conditions like rapid weight loss, chronic kidney disease, high blood pressure, hypothyroidism and haemorrhage may enhance the risk of gout. Condition that cause an abnormal rapid turnover of cells, such as psoriasis, multiple myeloma, hemolytic anemia or tumor may leads to gout. 8 Genetics: Genetic study have found that variants of Glucose transporter 9 (GLUT9) and ATP-binding cassette sub-family G member 2 (ABCG2) were associated with urate level. (6)

AETIOLOGY

Primary gout: About 1/3 of the body uric acid pool is derived from dietary sources and two –third from endogenous purine metabolism. The concentration of uric acid in the body fluid depends on the balance between its synthesis and elimination by the kidneys and gut. In over 90% of patients with primary gout, hyperuricemia results from an inherited defect in fractional uric acid excretion which impairs their ability to increase urate excretion in response to a purine load.(2)

Diminished renal excretion(common)

- Idiopathic .(7)
- Familial juvenile gouty nephropathy. (8)

- Inherent isolated renal tubular defect.
- Renal failure.
- Chronic drug therapy.
- Lead toxicity.
- Lactic acidosis(alcohol). (2)

Increased production of uric acid (uncommon)

- Increased purine turnover. (2)
- ❖ Idiopathic. (8)
- ❖ Chronic myeloproliferative or lymphoproliferative disorders (polycythaemia, chronic lymphatic leukemia)
- Increased de novo synthesis (over producers)
- ❖ Unidentified abnormality (most common).
- ❖ Specific enzyme defect(rare)
Hypoxanthine-guanine phosphoribosyl transferase deficiency
Phosphoribosyl pyrophosphate synthetase over activity
Glucose -6-phosphate deficiency. (2)

Secondary gout: Secondary gout is results from chronic hyperuricemia due to renal impairment or chronic diuretic use. In diuretic-induced gout nodal generalised OA is a further risk factor, especially in women. This presumably relates to a non-specific predisposition to crystallisation in osteoarthritic cartilage, possibly due to reduced levels of proteoglycan and other inhibitors of crystal formation. Lead poisoning is a rare cause of hyperuricemia and secondary gout.

Gout in old age groups: Etiology: predominantly primary gout, but a higher proportion of secondary gout (chronic diuretic therapy or chronic kidney disease) than in middle-aged patients. Nodal generalised OA: an important additional risk factor for gout. Presentation: more often atypical (e.g. presentation with painful tophi and chronic symptoms, rather than as classic acute attacks; presentation in upper rather than lower limbs).(2).

CLINICAL FEATURES:

There are four clinical stages in the natural history of gout:

1. Asymptomatic hyperuricemia.
2. Acute gouty arthritis
3. Inter-critical gout
4. Chronic tophaceous gout .(9)

ASYMPTOMATIC

HYPERURICAEMIA - Hyperuricemia may be an incidental finding and it may never lead to gout in all times. Conversely, during acute gouty arthritis serum uric acid levels may not be increased. - This is because adrenocorticotrophic hormone (ACTH) released in response to stress is uricosuric. Generally, asymptomatic hyperuricemia needs close observation with no active treatment. - However, growing epidemiological and experimental evidence indicates that asymptomatic hyperuricemia is capable of directly promoting hypertension and vascular disease. - Severe increased production of urate, as may occur with cytotoxic chemotherapy, may linked with high risk of getting acute renal failure and requires intervention.(10)

ACUTE GOUT - It's commonly in men aged 30-60 years. (8) - In almost all first attacks a single distal joints affected. - The 1st Metatarsophalangeal joints is affected in over 50 % of the cases "podagra".(2) - Asymmetrical affection of big toe.(11) - In order of decreasing frequency other common sites are ankle, mid foot, and knee, small joints of hands, wrist and elbow. - Typical attacks have the following characteristics: - Extremely rapid onset, reaching maximum severity in just two to six hours, often waking the patient in the early morning - Severe pain often described as worst pain ever. - Extreme tenderness: the patient couldn't unable to wear a sock or to let bedding rest on the joint. - Marked swelling with overlying red, shiny skin. - Mild fever with chills and even confusion, especially if a large joint such as the knee is involved.(2) - Leukocytosis and raised ESR. - First attack is seldom associated with residual disability. - It can also present as tenosynovitis, bursitis, cellulitis. (11) - Self-limiting over five to fourteen days, with complete return to normality. - Many patients describe milder episodes lasting just

a few days (petite attacks). Some have attacks in more than one joint; sometimes one attack triggers attacks in other joints a few days later ('cluster attacks'). - Polyarticular attacks are rare. (2)

RECURRENT AND CHRONIC GOUT – After an acute attack some people never have a second episode, in others the next episode occurs after years. – In most, however, a second attack occurs within 1 year and the frequency of attacks gradually increases with time. – Later attacks are more likely to involve several joints and to be more severe. Eventually, continued MSUM deposition causes chronic pain and joint damage. – The interval between the first attack and the development of chronic symptoms is variable, but averages around 10 years. – The main determinant is the serum uric acid; the higher it is, the earlier and more extensive the development of joint damage and MSU deposits. – The joints most commonly involved are the same as those affected by acute attacks. – Occasionally there may be severe deformity and marked functional impairment, especially of feet and hands. As with tophi, asymmetry is characteristic.(2)

CHRONIC TOPHACEOUS GOUT: Initially the tissue around joints is red, swollen and edematous. Later ligaments and bone ends are infiltrated by chalky deposits which form tophi. (11) Development of chronic tophaceous gout depends on uncontrolled hyperuricemia of long duration, usually >10 years. But tophi or chronic polyarthritis may occur early as 3 years or as late as 40 years after the first acute attack. (10) – Tophi -Large MSUM crystal deposits produce irregular firm nodules. (2) Overlying skin generally thin and red. Tophi that are very near to skin may appear cream –coloured or yellow. (6) The usual sites for nodules around extensor surfaces of fingers, hands, forearm, elbows, Achilles tendons and sometimes the helix of the ear. (2) Rarely tophi may form in the eye, tongue, larynx or heart, and interference with cardiac conduction and Valvular function has been recorded. (11)

INVESTIGATIONS: Typical Clinical Picture: The presence of acute monoarthritis involving first metatarsophalangeal (Podagra), where inflammation peaks in 24 hours, and may involve mid tarsal, ankle, knee, wrist or elbow joints. There may be presence of tophi. A fast response to colchicine further establishes a diagnosis of gout. Typically gout is seen in elderly, shows oligo/Polyarticular subacute/persistent arthritis of unusual joints, also affecting bursae, tendon sheaths making distinction from chronic arthritides difficult. (12)

Serum Uric Acid Estimation: Serum uric acid (SUA) is usually elevated but may be normal in about 30 percentage of patients during the acute attack because of IL-6 and endogenous cortisol secretion, which are uricosuric so Serum uric acid should be repeated after 2 weeks. (11) Blood uric acid is measured, when fluid cannot be aspirate easily from the affected joints. (6).

Synovial Fluid (SF) Examination: Definitive diagnosis requires identification of MSU crystals in the aspirate from a joint, bursa or tophus. (2) A fresh sample synovial Fluid should be aspirated to demonstrate the presence of monosodium urate (MSU) crystals. Synovial Fluid if not examined immediately may be refrigerated for days to months. Monosodium urate crystal identification is considered the gold standard for diagnosis. (12)

Conventional computerised Tomography (CCT): It is able to detect erosion's better than Magnetic Resonance Imaging (MRI). These are described as well defined, punched out lytic bone lesions, with sclerotic overhanging edges.(13)

MATERIALS AND METHODS

Method of collection of Data Research design:

- A prospective experimental study design without control group.

Sample design:

- A purposive sampling done as per inclusion and exclusion criteria.

Data collection:

- Primary and secondary data collected through a predesigned case sheet.

Statistical analysis:

- Paired t-test

Inclusion criteria fixed for the study.

- Age group of 40 and above.
- Both male and female has been included.
- Subjects who have given consent for the study.
- Serum uric acid level greater than 7.0 mg / dL in males and 6.0 mg /dL in females.
- Medication will be on the basis of the constitutional homoeopathic totality.
- Diagnosed cases of Gout.

Exclusion criteria fixed for the study:

- Gout patients with other systematic diseases like HTN, DM and Rheumatoid arthritis on active treatment.

- Gout patients with complications like uric acid stones, uric acid nephropathy.

PROGNOSTIC CRITERIA:

- It is based on the symptomatic improvement and investigatory findings (serum uric acid level).

METHODOLOGY:

- Patients will be selected on the basis of inclusion and exclusion criteria. The cases will be followed for a period of one year, with a fortnightly O.P.D visit.
- The Medicines will be prescribed on the basis of constitution of patient and repeated accordingly.

STATISTICAL ANALYSIS

Table Statistical Analysis: In this study the value of Serum uric acid (SUA) is considered for the comparison of result.

Assessment criteria:

| SL.NO | S.URIC ACID VALUE BEFORE (MESURED IN Mg/dL) X VALUE | S.URIC ACID VALUE AFTER (MESURED IN Mg/dL) Y VALUE |
|-------|---|--|
| 1. | 7.8 | 6.50 |
| 2. | 8.6 | 7.10 |
| 3. | 7.50 | 7.0 |
| 4. | 8.90 | 6.80 |
| 5. | 8.10 | 7.80 |
| 6. | 10.50 | 8.30 |
| 7. | 13.20 | 9.80 |
| 8. | 11.80 | 7.0 |
| 9. | 8.60 | 6.0 |
| 10. | 8.60 | 6.90 |
| 11. | 6.70 | 5.90 |
| 12. | 11.50 | 9.80 |
| 13. | 6.90 | 5.80 |
| 14. | 6.50 | 6.0 |
| 15. | 8.0 | 6.30 |
| 16. | 8.70 | 7.20 |
| 17. | 8.90 | 6.30 |
| 18. | 8.0 | 6.50 |
| 19. | 9.20 | 7.50 |
| 20. | 7.40 | 6.50 |
| 21. | 8.0 | 5.90 |
| 22. | 8.0 | 7.10 |
| 23. | 8.10 | 6.70 |
| 24. | 8.40 | 6.0 |
| 25. | 10.20 | 7.0 |
| 26. | 7.90 | 5.60 |
| 27. | 7.40 | 5.80 |
| 28. | 10.30 | 6.70 |
| 29. | 8.10 | 6.80 |
| 30. | 10.20 | 6.70 |
| | 262 | 198.3 |

Question to be answered:

Is there any difference between the scores taken before the treatment and scores after the Homoeopathic treatment?

A. Null Hypothesis: There is no difference between the scores before and after the Homoeopathic treatment.

B. Alternate Hypothesis: There is a difference in the scores before and after the Homoeopathic treatment.

C. Standard score of the mean of difference

$n = 30$

$X =$ Scores before treatment

$Y =$ Scores after treatment

$Z =$ Mean difference

$\Sigma z = 55.69$

$\bar{Z} = \Sigma z / n$

$\bar{Z} = 55.69/30 \bar{Z} = 1.85$

Step 1: Find t value and degrees of freedom

To find t value and degrees of freedom we will use following formulas: The estimate of population standard deviation is given by

$$SD = \sqrt{\Sigma (z - \bar{z})^2 / n - 1}$$

$$SD = \sqrt{31.47 / 29}$$

$$SD = \sqrt{1.08}$$

$$SD = 1.039$$

The estimate of standard error of mean, SE

$$SE = SD / \sqrt{n}$$

$$SE = 1.03 / 5.47$$

$$SE = 0.18$$

Critical ratio 't'

$$t = z / SD \sqrt{n}$$

$$t = 1.85 / 1.03 / 5.47$$

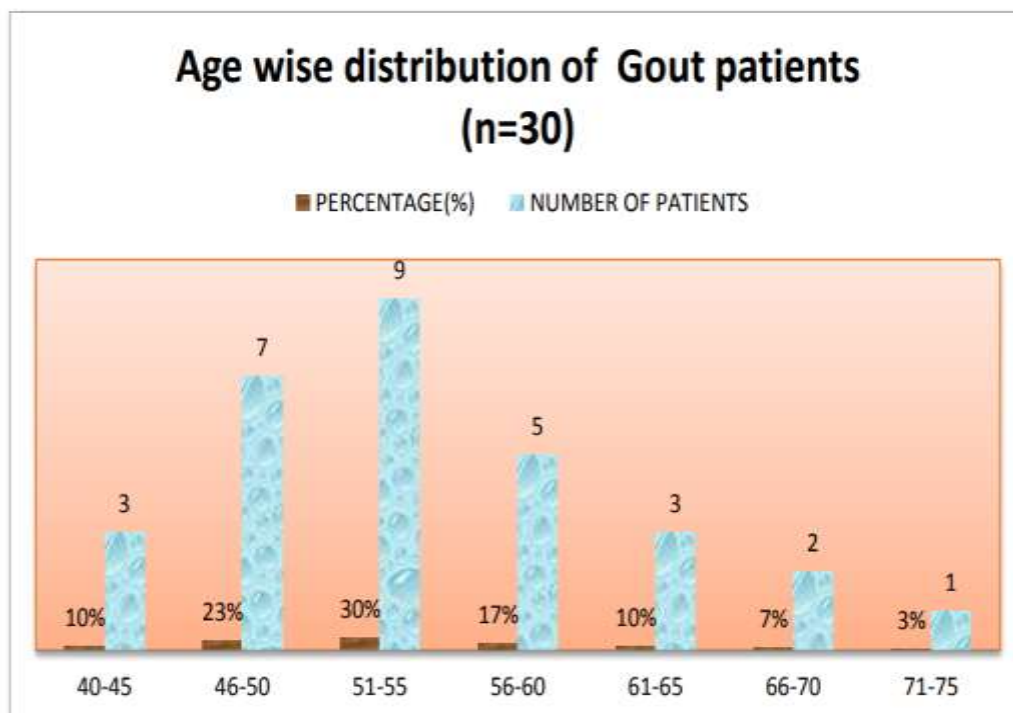
$$t = 10.2777$$

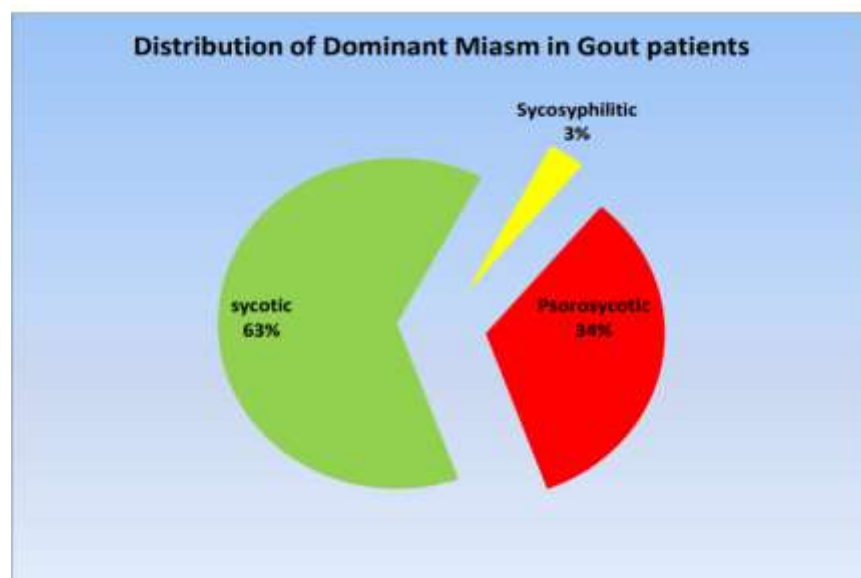
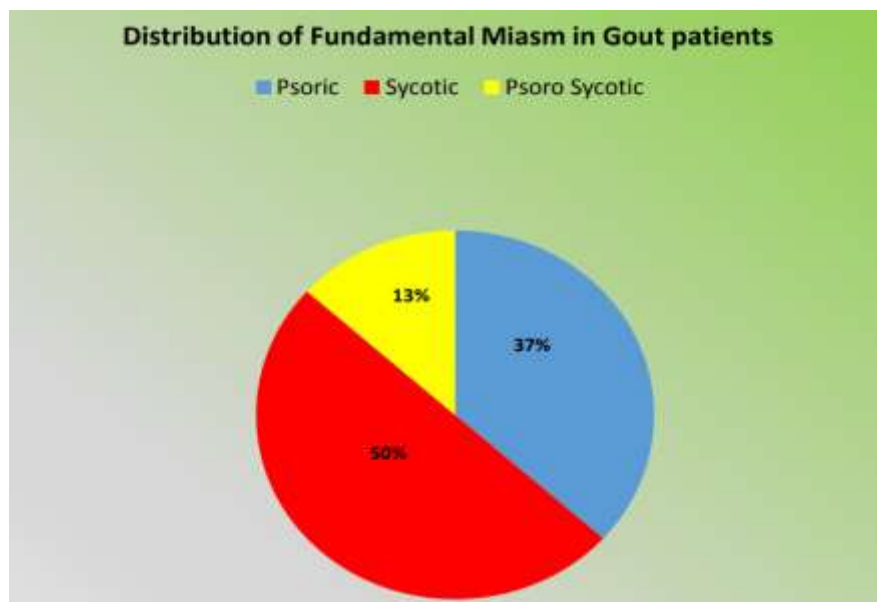
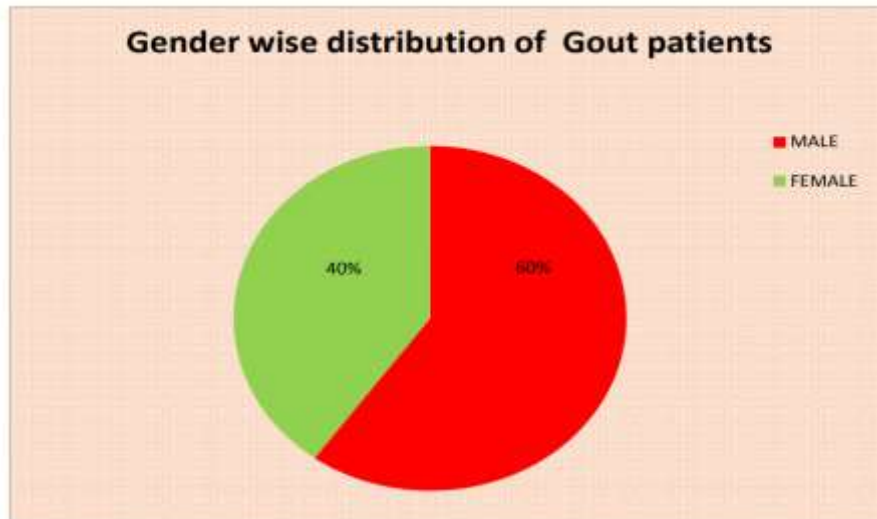
Comparison with tabled value:

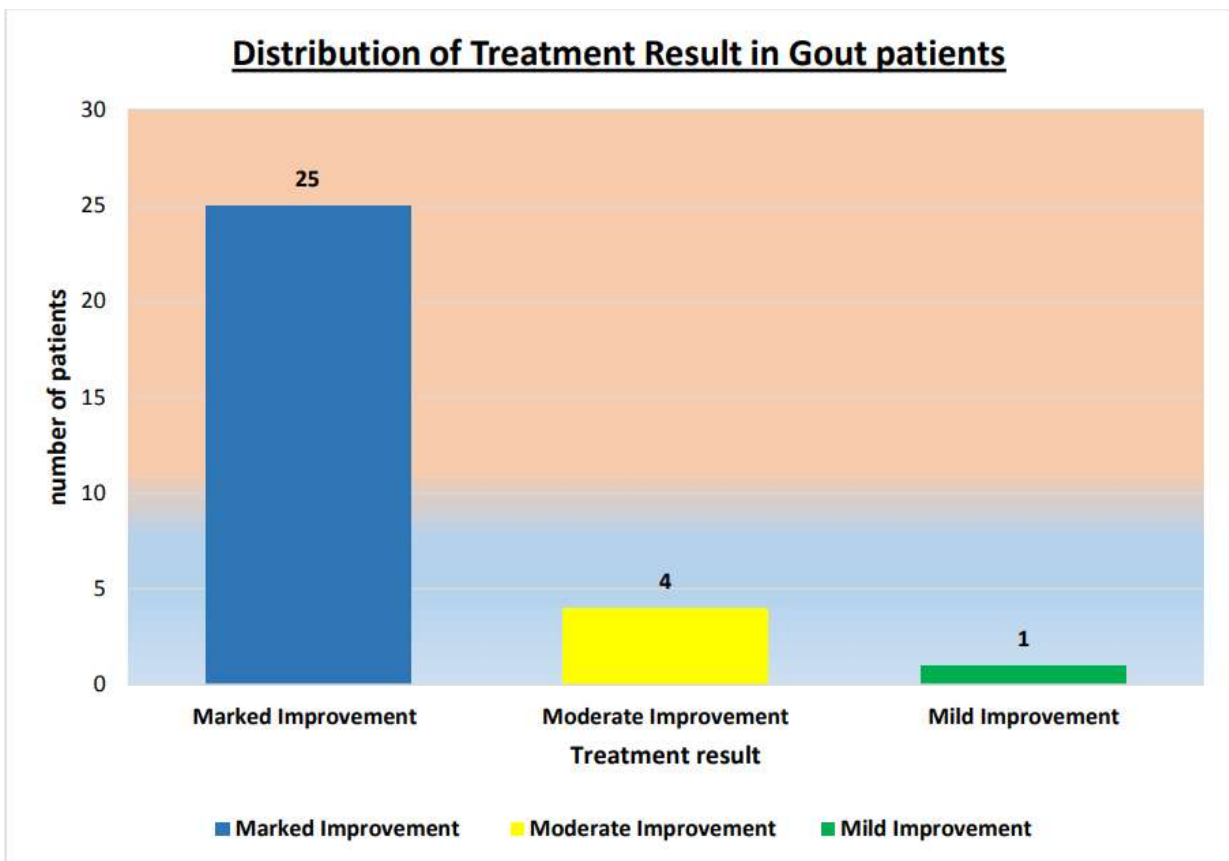
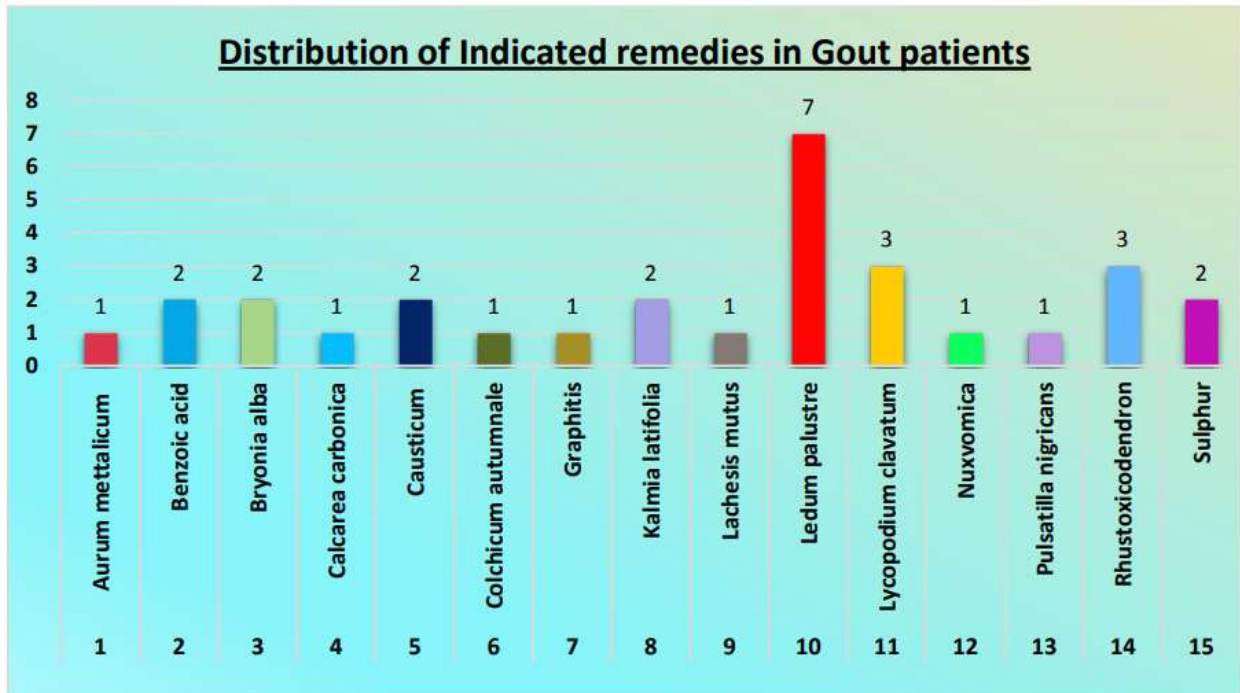
The test statistic 't' follows patient's t distribution with n- 1 (29) degrees of freedom. Here, tabled value of 't' at 5% level of significance is 2.045 for 29 degrees of freedom. Since the calculated value is 10.2777 which are greater than the table value at 5% we reject null hypothesis.

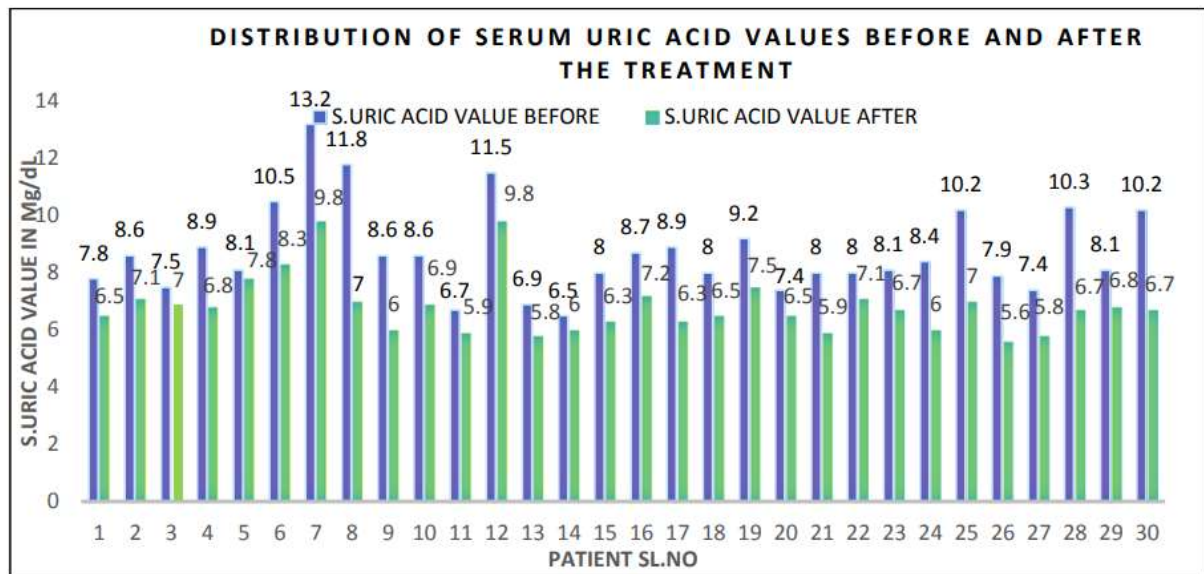
INFERENCE: This study provides evidence to say that, there is significant reduction in the serum uric acid levels after giving Homoeopathic treatment. Therefore, Homoeopathic treatment is effective in cases of Gout.

OBSERVATION AND RESULTS









DISCUSSION

Thirty clinically and diagnosed cases of Gout patients were taken into consideration for the study. The patients were the age group of 40 and above. Patients of both sexes were treated. A detailed case history with the proper clinical examination was done in all the patients. The protocol for the study was cases are followed for a period of 1 year and Investigations will be done before and after treatment. This study shows most affected patients where Age wise distribution of patient's shows that most affected patients where 51-55 years (30%), and age group of 46-50 years (23%) , age group of 56-66 years (17%), age group of 40-45 & 61-65 years 10% each , age group of 66-70 years (07)%, age group of 71-75 years (03%) respectively. Sex distribution of patients shows that of affected persons, 60% were male and 40 % of female were affected by Gout. According to literature, it suggests males are more affected than females.

The Predominant fundamental miasm in the study was Sycotic (50%) and Psoric which is seen in (37%) patients, rest were Psoro Sycotic (13%) patient. The major Dominant Miasm was Sycotic seen in 63% of patients, next to it was the Psorosycotic which seen in 34% of patients. Followed by Sycosyphilitic which is seen in 03 % of the patients. The remedies that are used most

frequently for managing the patients were 7 cases Ledum palustre, both Lycopodium clavatum and Rhus toxicodendron in 3 cases. The next grade remedies were Benzoic acid, Bryonia alba, Causticum, Kalmia latifolia, Sulphur in (2) cases. The other minimum used remedies were Aurum metallicum, Calcarea carbonica, Colchicum autumnale, Graphitis, Lachesis mutus, Nuxvomica, Pulsatilla nigricans, (1). The treatment result has shown that maximum number of patients (25) were Marked Improvement which is 83% of total patient, while Moderate Improvement was seen in 14% of (04) patients. Mild Improvement was seen in 3% of patients (01). The test statistic 't' follows patient's t distribution with n- 1 (29) degrees of freedom. Here, tabled value of 't' at 5% level of significance is 2.045 for 29 degrees of freedom. Since the calculated value is 10.2777 which are greater than the table at 5% we reject null hypothesis. This study provides evidence to say that, there is significant reduction in the serum uric acid levels after giving Homoeopathic treatment. Therefore, Homoeopathic treatment is effective in cases of Gout. Homoeopathy is known to control the uric acid diathesis and is also useful in acute attacks. Its helps in controlling the pain during the acute attack of gout as well as helps in preventing the recurrence of such episodes.

SUMMARY AND CONCLUSION

Thirty cases of Gout patients were taken into consideration for the study. The patients were between the age group of 40 and above, both males and females included for the study. A detailed case history with the proper clinical examination with serum uric acid was done in all the patients.

- The commonly affected age group according to my study were 51 to 55 years (30%).
- Males are most commonly affected, which was evidenced by this study which shows presence on 60% of cases.
- The most affected were house wives which were evidenced by this study which shows presence on 34% of cases.
- The most affected persons were BMI above normal level (63%).
- Both fundamental miasm and dominant miasm which covered mostly was sycotic in my study.
- Ledum palustre is found to be most frequently indicated constitutional remedies. General management in the form of diet and exercise guidelines was given to the patients.
- According to need of the cases some acute remedies were prescribed in between.
- Most of the patients got symptomatic relief, as well as general wellbeing.
- this study shows that gout condition can be treated with homoeopathic medicines.

Recommendations

- A Similar study should be done under large sample.
- Proper advertisement should be given patients regarding the treatment of Gout in Homoeopathy and it has no side effects in treatment.
- Proper awareness should be given to public about life style modification and nutritional supplements.
- Proper study of patients with suitable research design can also be done to investigate role of homoeopathic

medicines in comparison to non-steroidal anti-inflammatory drugs.

- Further study of Gout can be done based on synovial fluid examination. • Proper study of patients with abnormal Body mass index leads scope to investigate the role of homoeopathic medicines in overweight.

LIMITATIONS

- The effectiveness of homoeopathic treatment in case of Gout is less aware in public.
- There is a common misunderstanding in public that homoeopathic treatment will take more time to get relief. So we have to counsel them regarding treatment of Gout in homoeopathy.
- It difficulty to get correct picture of the disease while the patients came after other system of medication.
- After getting slight improvement in the condition, they will leave the treatment.
- There was no control group since the sample size was small.

Declaration by Author

Ethical clearance: Approved

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