# ASSOCIATED PROBLEMS AMONG THE GOUTY PATIENTS IN KASHMIR

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ABSTRACT: The present study was aimed to study the associated problems among the Gouty Patients in Kashmir. To gather information from 50 gouty patients, Questionnaire was used. Respondents were selected purposively & randomly from the Kashmir valley. Data collection was done in the various district hospitals of Kashmir. It was found that pain in joints and redness of joints was found to be the most common associated problem present in majority of the studied sample.

KEYWORDS: uric acid, pain, inflammation, hyperuricemia, purine.

### INTRODUCTION:

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 crystals. Gout is a disease that results from an overload of uric acid in the body. This overload of uric acid leads to the formation of tiny crystals of urate that deposit in the body, especially the joints (**Christopher**, 2002).

Urate is a chemical in the blood. It is made in the body when certain foods are digested. It is usually in the form of a harmless liquid that passes out from the body through urine. But in some people, the amount of urate in the blood builds up. It starts to form tiny crystals which can collect in a joint. The crystals can cause inflammation and pain (**Lin**, 2006).

The joint inflammation is precipitated by deposits of uric acid crystals in the joint fluid (Synovial fluid) and joint lining (Synovial lining). Intense joint inflammation occurs as white blood cells engulf the uric acid crystals and chemical messengers of inflammation are released, causing pain, heat and redness of the joint tissue. Chronic gout leads to deposits of hard lump of uric acid in and around the joints and may cause joint destruction, decreased kidney function and kidney stones (**Keith**, 2007).

About one- third of the uric acid is derived from dietary sources and two- thirds from endogenous purine metabolism. Endogenous purines are made within human cells. Exogenous purines are obtained from food. The process of breaking down purine results in the formation of uric acid in the body. Most mammals have an enzyme called uricase, which breaks down uric acid so it can be easily removed from the body. Because humans lack uricase, uric acid is not easily removed, and builds up in body tissues (**Dehghan**, 2008).

The various factors that increase the risk of gout include: Gender, family history, obesity, medication, alcohol, lead exposure and other illnesses.

- **1**) Age:
- Middle- Aged Adults: Gout usually occurs in middle aged men, peaking in the mid 40's.
- Elderly: Gout can also develop in older people, where it occurs equally in men and women.
- Children: Except for rare inherited genetic disorders that cause hyperuricemia, gout in children is rare.
- 2) Family History:
  - A family history of gout is present in close to 20% of patients with condition.
- 3) Obesity:

Researchers report a clear link between body weight and uric acid. Overweight people had two or more than three times the rate of hyperuricemia as those who maintained a healthy weight.

- 4) Medications:
  - Thiazide diuretics are "water Pills" used to control hypertension. The drugs are strongly linked to the development of gout. Several other medications which can increase uric acid levels and raise the risk for gout include Aspirin, Niacin and Pyrazinamide (**Surendra**, 2008).
- 5) Alcohol:

Drinking excessive amounts of alcohol can raise risk of gout. Beer is a kind of alcohol most strongly linked with gout. Alcohol increases uric acid levels in three ways: providing an additional dietary source of purines, intensifying the body's production of uric acid and interfering with the kidney's ability to excrete uric acid.

**6)** Lead Exposure:

Chronic occupational exposure to lead is associated with build up of uric acid and a high

Gout is caused by uric acid crystallization in joints which causes an immune response. These uric acid crystals are seen as invaders by immune system which causes it to respond. This cause immune system to always be in a heightened state of alert and it can cause many deadly diseases (Linc et. Al. 2000).

Gout if left untreated can develop into a painful and disabling chronic disorder. Persistent gout can destroy cartilage and bone, causing irreversible joint deformities and loss of motion. Tophi are firm chalky, gritty clumps of uric acid that build up in tissue surrounding a joint. If gout is not treated, tophi can grow to the size of ball and can destroy bone and cartilage in the joints (**Chio et. Al. 2008**).

Low purine diets are recommended to the patients suffering from the gout. Low purine diets are low in vitamin c, vitamin B and other antioxidants. So, supplementation will be necessary to prevent damage from free radicals that can intensify gouty problems.

Researchers have found that Vitamin C appears to reduce levels of uric acid in the blood. Daily intake of 500mg of Vitamin C significantly reduces uric acid levels. Vitamin C intake may provide a useful option in the prevention of gout. Higher Vitamin C is associated with a lower risk of gout (**Huang et. al, 2005**).

### LITERATURE OF REVIEW:

According to Annil Mahajan et.al. (2007), gout is an important cause of arthritis and the prevalence is on the increase. The incidence of gout in India varies in population with an overall prevalence of less than 1 to 15.3%. Gout once called the "disease of kings", is also seen in women, especially after menopause. The male female ratio changes as estrogen status changes. Normally male: female ratio is 7:1 to 9:1, women before menopause is female> male, in the older age groups above 65 years- male: female- 3:1 ratio and after 80 years of age females are compared to males.

Thorough study carried out by Matsumoto Mifuji (2001), revealed that most of the epidemiological data showed that the long term prognosis of the patients with gout is related not to the symptoms of uric acid deposition but cardiovascular complications. The cardiovascular events occur at multiplicative frequency, higher than the additive frequency of each factor.

Atkinson et.al. (2003), stated that there is a strong association between the gout and Nephrolithiasis in men. The study shows approximately 10 million adults in the U.S have experienced the passage of a kidney stone and upto 5 million have been diagnosed with gout by a physician. A study carried by Alan R Erickson (2004), stated that the prevalence of hypothyroidism in the prospective group was significantly increased compared to controlled group. Overall 15% of these 54 patients, 25% of women and 12% of the men had hypothyroidism. These rates are higher in women as compared to men. Hypothyroidism is significantly increased in patients with gouty arthritis. Screening for hypothyroidism with an ultrasensitive thyroid stimulating hormone assay should be considered in all patients presenting with gouty arthritis.

Uric acid levels if remain raised for longer period of time, or there are frequent, recurrent attacks of gout, deposits of uric acid salts may appear around the affected joint. These are called tophi. Recurrent severe attacks of gout and the development of tophi can cause permanent damage to the joints. Surgery may be required to restore joint function (Jones P, 2005).

Study carried by Hyon K & Gary Curhan (2007), stated that men with gout have a high risk of death from all causes. Among en without pre-existing CHD, the increased mortality risk is primarily a result of an elevated risk of CVD death, particularly from CHD.

E. Krishnan et.al. (2008), found that men with a high cardiovascular risk profile suggest that men with gout are at higher future risk of Type 2 Diabetes independent of other known risk factors. These data expand on well established, cross-sectional association between hyperuricemia, gout and the metabolic syndrome and extend the link to the future risk of Type 2 Diabetes. Gout results from the deposition of monosodium urate crystals in different body tissues, including the skin and soft tissues. With increasing prosperity a rising prevalence of the disease can be observed, especially in highly industrialized countries. Gout can present with acute and painful attacks of arthritis that can affect several joints (Carleine A et.al, 2008).

Mary A Devera et.al. (2009), studied the association between gout and the risk of acute myocardial infarction among the elderly women, aged ≥ 65 years. A population based cohort study was conducted using the British Columbia linked health data base and compared incidence rates AMI between 9642. The population based data suggest that women with gout have an increased risk for AMI and the magnitude of excess risk is higher than in men.

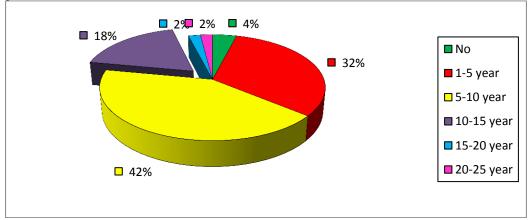
### **METHODOLOGY:**

The study was undertaken to assess the problems associated with the Gouty patients in kashmir. 50 Gouty patients of any age group were selected purposively and randomly for the present study. The sampling was conducted in OPD of various district hospitals. During the study, a structured questionnaire cum interview schedule was used to collect information from the Gouty patients. The purpose is to gather information from them. It is a quick and efficient way to gather information from target no. of people. After the required information was gathered, the data was carefully analyzed and interpreted.

# **RESULTS:**

### ASSOCIATED PROBLEMS

These tables revealed the information regarding various associated problems being experienced by the studied sample and the results are presented in fig 1.1 to fig 1.5.



According to Johnthas (2000), an acute arthritis is one of the most painful experiences reported throughout the medical history. Severe pain at and around joints is the common sign.

Fig. 1.1: Distribution of sample as per the presence of symptoms of disease (pain in joints).

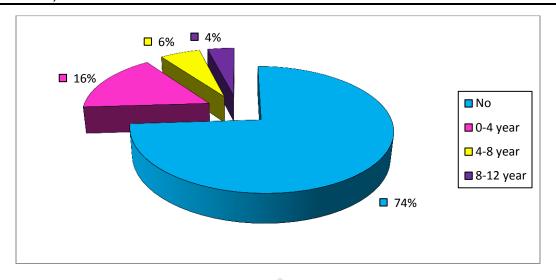


Fig. 1.2: Distribution of sample as per the presence of symptoms of disease (inflammation in arms & legs).

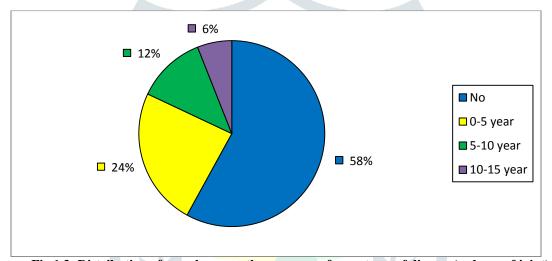
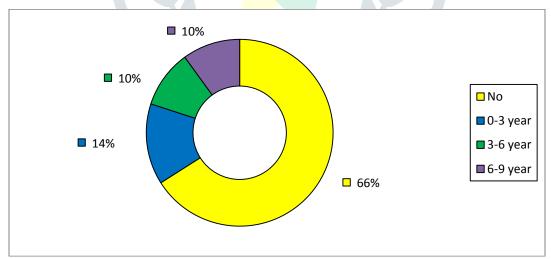


Fig.1.3: Distribution of sample as per the presence of symptoms of disease (redness of joint).



Swelling may also occur around the joints and there may be redness over the areas affected by gout (Johnthas, 2000).

Fig. 1.4: Distribution of sample as per the presence of symptoms of disease (tenderness of joint).

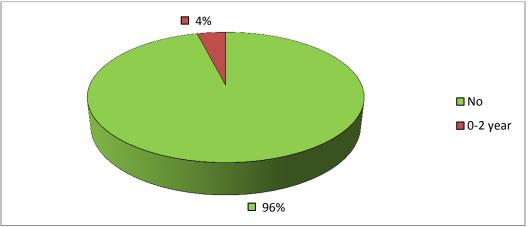


Fig. 1.5: Distribution of sample as per the presence of symptoms of disease (kidney stones).

Careful analysis of fig 1.1 to fig 1.5 showed that 96% of the studied sample were having pain in joints and the majority (42%) of them were having it from 5-10 years. Inflammation in legs and arms was present in 26% of the studied sample. 42% were having redness of joints and the majority (24%) of them were having it from 0-5 years. Tenderness of joints was present in 34% of the studied sample and only 4% were having kidney stones.

### **CONCLUSION:**

People with gout develop complications such as recurrent gout, advanced gout, kidney stones and kidney failure.

Pain in joints and redness of joints was found to be the most common associated problem present in majority of the studied sample.

# **ACKNOWLEDGEMENT**

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## REFERENCES

- [1]. Christopher, I., Haslett, K., Edwin, R., Chilvers, N. (2002). Principles and Practice of Medicine (19<sup>th</sup> Edition), India, Elservier Science, Pp 512-515.
- [2]. Lin, K. (2006). Urate in relation to gout. *Journal of Rheumatology*, 27(6): 1501-1505.
- [3]. Keith, M. P. (2007). Updates in the management of gout. American Journal of Medicine, 12(3): 221-224.
- [4]. Dehang, A. (2008). Endogenous and exogenous. *Lancet*, 372(965): 1958-1961.
- [5]. Surendra, P. S and Mohini, Teotia. (2000). Nutritional and metabolic bone diseases, New Delhi, CBS Publishers & Distributors.
- [6]. Lin, K. C., Taren, S and Chwayo, D. (2000). Overview on gout. Journal of Clinical Nutrition, 52(4): 184-189.
- [7]. Choi, H. K and Curhan, G. (2008). Coffee, tea and caffeine consumption and serum uric acid level. Arthritis Rheumatology Journal, 57(5): 816-818.
- [8]. Huang, H. Y and Chadha, D. (2005). The effects of vitamin C supplementation on serum concentrations of uric acid. *Journal of Arthritis Rheumatology*, 52(6): 1843-1845.
- [9]. Annil, Mahajan. (2007). Prevalence of major rheumatic Disorders in Jammu. JK Science, 9 (7): 49-51.
- [10]. Matsumoto, Mifuji. (2001). Gout. Journal of Current Therapy, 22(3): 181.
- [11]. Atkinson, K. (2003). Kidney stones & gout, JAMA, 20 (7): 17-22.
- [12]. Alan, R., Erickson, M., Wolf, S., Burney, P. (2004). The prevalence of hypothyroidism in gout. *The American Journal of Medicine*, 97 (3): 231-234.
- [13]. Jones, P. (2005). Modern management of gout. New Ethical Journal, 4(2): 29.
- [14]. Hyon, K and Gary, Curhan. (2007). Impact of gout on mortality and risk for coronary heart disease. *American Heart Association Journal*, 116: 894-900.
- [15]. Krishnan, E., Desilva, N., Fung, I. C., Gebre, S. (2008). Gout and risk of Type 2 diabetes among men with a high cardio vascular risk profile. *Oxford Journal*, 47(10): 1567-1570.
- [16]. Carleine, A and Lucker, P. H. (2008). Gout. Journal of the International Society of Dermatology, 41(3): 237-239.
- [17]. Mary, A., Devera, K and Maclachen, Z. (2009). Association of gout with myocardial disease. *Journal of Rheumatology*, 42(5): 633-635.