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| Anternational | Correlation of Serum Uric Acid Levels in Gouty and Non Gouty Osteoarthritis | | | |
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| ABSTRACT Gout | ı kind of osteoarthritis caused by too much uric acid in blood. High level in blood of some population not get ut affected by arthritis. In our study we demonstrated determination of uric acid in 50 gouty osteoarthritis and | | | |

50 non gouty osteoarthritis and in 50 normal healthy subjects. In gouty osteoarthritis mean \pm S.D serum uric acid was 9.3 \pm 2.6, in gouty osteoarthritis 7.25 \pm 1.38 and in normal healthy control it was 5.97 \pm 1.38. Correlation between these subjects were found to be significant when compared between normal subject and gouty osteoarthritis (p < 0.001) and Non gouty osteoarthritis (p < 0.005). The prevalence of gouty arthritis and non gouty arthritis in present study indicated that a sincere efforts have been made to identify these disease and management too.

KEYWORDS : Uric Acid, Gout , Osteoartheritis

INTRODUCTION

Uric acid is the principal product of the catabolism of purine, adenosine and guanine nucleotides. Approximately 400 mg of uric acid are synthesized daily; food intake accounts for a further 300 mg, about 75% of the uric acid excreted is eliminated in the urine. Most of the uric acid is secreted in the gastrointestinal tract where it is degraded by bacterial enzymes into allantoin and other compounds. The amount of uric acid present is determined essentially by the life style adopted e.g. alimentary habbits, alcohol consumption, physical activity, assumption of pharmaceutical products may all influence hyperuricemia. Some of the most dangerous complications of uricemia include acute and chronic attacks of gout and pathological conditions related to renal function (Fossati et al 1980). Osteoarthritis is the most common type of arthritis. Its high prevalence, in the elderly and high rate of disability related to disease makes it leading cause of disability in the elderly. Osteoarthritis affects certain joints such as knee, hip, lumbosacral, spine and cervical. The distal and proximal interphalangeal joints and base of the thumb are most affected, which spared are the wrist, elbow and ankle (Davit T and Ferson 2011). Diagnosis of osteoarthritis seen as join space loss on x ray and osteophytes and elevated serum uric acid level. However uric acid level increased in osteoarthritis patients with history of gout and without history of gout. Osteoarthritis is a disease in which the amount of uric acid in one joint or both joints may increase, the most common form of arthritis worldwide (Science daily 2011). The determination of uric acid as diagnostic value in differentiating gout from non gouty arthritis (Schlesinger N 2007).In present study we determined level of uric acid in gouty and non gouty osteoarthritis and compare with normal healthy subjects.

MATERIAL AND METHODS

We have studied uric acid analysis in 150 subjects(Male and Female) between the age 35-65 years during years between November 2009 to December 2010. The present study were undertaken in the department of Orthopaedic and department of Biochemistry, Jhalawar Medical college, Jhalawar (Rajasthan) and study subjects coming to O.P.D and I.P.D department of Orthopaedic. Jhalawar Medical college,-Jhalawar (Rajasthan) .The study participants were devided into three groups- Group(1)= control, (2) gouty osteoarthritis and (3) non gouty osteoarthritis. Among of them 50 were healthy control subjects, 50 patients suffering from osteoarthritis with gout and 50 were without osteoarthritis. Uric acid was estimated by kit method of Trivedi 1975. subjects of our study had not taken any medication during study.

Subjects with history of diabetes mellitus, cardio vascular disease (Hypertension), alcoholism, renal disease and other diseases were excluded from the study. Data were analyzed using specific stastical tool, SPSS version 20.0.

RESULTS

In present study 50 patients (Male and Female) whose uric acid level was found to be higher than normal, with history of knee joint pain but had history of gout, 50 patients had no history of gout and 50 subjects whose uric acid level were within normal limits were evaluated. The age of our study cases were between 35-65 years.

In 50 healthy control subjects uric acid mean \pm S.D. were found 5.97 \pm 2.05 mg/dl, in 50 osteoarthritis patients level of uric acid without history of gout 7.25 \pm 1.38 (mean \pm S.D.) and in 50 osteoarthritis patients with history of gout uric acid value were found 9.37 \pm 2.61 (mean \pm S.D.) (Table 1). Uric acid value were compared among healthy control group with gouty osteoarthritis patients and without gouty osteoarthritis patient and significant results were obtained (Table 2).

TABLE-1: LEVEL OF SERUM URIC ACID(mg/dl) IN CONTROL, WITH GOUTY OSTEOARTHRITIS AND WITHOUT GOUTY OS-TEOARTHRITIS.

| GROUP | MALE | FEMALE | URIC ACID (mg/dl) |
|------------------------------------|------|--------|----------------------|
| Control | 28 | 22 | 5.97 ± 1.38 |
| Cases with Gouty osteoarthritis | 32 | 18 | 9.37 ± 2.61 |
| Cases without Gouty osteoarthritis | 36 | 14 | 7.25 ± 1.38 |

TABLE-2: CORRELATION OF SERUM URIC ACID (mg/dl) VALUE

| GROUPS | No. OF PATIENTS | P VALUE | SIGNIFICANCE |
|---------------------------------|--------------------|------------|--------------------|
| (1) Control | 50 | - | - |
| (2)Without gouty osteoarthritis | 50 | <0.001 | Highly significant |
| (3)Without gouty osteoarthritis | 50 | <0.005 | Significant |

correlation = gp1 Vs gp2 and gp1 Vs gp3

DISCUSSION

Gout is disease recognized since antiguity and has been termed "disease of Kings and King of disease" (Robert Terkeltaub 2013). In gout an elevation in the concentration of urate beyond its solubility threshold (77.0 mg/dl in vitro) promotes deposition of crystals of the mono sodium urate (MSU) in joint fluid and on the articular cartilage surface, and into larger structures containing aggregated crystals that are termed 'TOPHI' (Dalbeth et al, 2010). In our study uric acid mean level higher than normal level in gouty osteoarthritis is similar to reported by other workers (Lawrence R C et al, 2008, Wallace KL et al, 2004, Bhole et al 2008). Hyperuricemia in arthritis patients with gout is defined >7.0 mg/dl in adult males and >6.0 mg/dl in premenopausal females (Krishnan et al, 2012, D'Oherty M 2009).Patients with gout have co morbid conditions that predispose to renal dysfunction and increased urate reabsorption including hypertension and insulin resistance (metabolic syndrome) likely contribute to elevated risks of uric acid, urolithiasis in gout patients (Kramer H J et al 2003). Alcohol consumption promotes hyperuricemia in part by increasing uric acid production (Backer M and Koopman WJ 2001).

In our study mean uric acid level without gouty osteoarthritis patients increased up to 7.25 mg/dl. Our report matched with studied by other workers (Corrado et al 2006, Roddy E 2007, Choi H K 2010). Patients without gouty artheritis and patients with gouty arthritis shows , serum uric acid (Mean) concentration 8 mg/dl and they are excreting uric acid at a rate 0.9 mg/min and 0.5 mg/min respectively (Zhu y 2011). Increased gouty osteoarthritis and non gouty osteoarthritis have been seen all over the world population such as India, United States, Newzeland and Taiwan although apparently not in Britain (Roddy E et al 2007).

In present work we have observed arthritis with gout and non gout, uric acid level increased than normal and may be a risk factor for other diseases such as renal disease, hypertension and atherosclerosis, our findings similar to reported in literature (Feig Di et al 2008, Martinon 2005, Emersion BT 1996). Osteoarthritis and gout frequently coexist and osteoarthritis is a risk factor for development of gout in an affected osteoarthritic joints. In both gouty osteoarthritis and non gouty osteoarthritis level of uric acid is important compound for diagnosis and management of osteoarthritis.

CONCLUSION

Gout is not simply an acute illness, chronic inflammatory and erosive arthritis also can develop in gout. Gout and hyperuricemia are biomarkers for increased cardiovascular disease also manifests urolithiasis. Decreased uric acid elimination is the primary driver of hyperuricemia in gout in most of the cases. Overproduction of uric acid also observed in several acquired genetic disorder with excessive rate of cells (and purine) turnover. Relationship of hyperuricemia with gouty osteoarthritis and non gouty osteoarthritis in present study is important in diagnosis, prognosis of arthritis disease. Uric acid determination also economic test for diagnostic purpose. We conclude in our study that uric acid level should be monitored for osteoarthritis patients and hyperuricemia adequately control and ultimate permanent resolution of tophi with lifelong maintenance of a normal serum urate



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