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Review of literature on hyperuricemia.

Revisión de la literatura sobre hiperuricemia.

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ABSTRACT

Hyperuricemia has long been recognised as the primary cause of gout. A large body of evidence suggests that hyperuricemia may play a role in the development and pathogenesis of a variety of metabolic, hemodynamic, and systemic pathologic diseases, including metabolic syndrome, hypertension, stroke, and atherosclerosis, in recent years. Several epidemiologic studies have linked hyperuricemia to each of these conditions. Uric acid-lowering therapies have been shown in some studies to prevent or improve certain aspects of the metabolic syndrome.

Keywords- Hyperuricemia.

RESUMEN

Hyperuricemia has long been recognised as the primary cause of gout. A large body of evidence suggests that hyperuricemia may play a role in the development and pathogenesis of a variety of metabolic, hemodynamic, and systemic pathologic diseases, including metabolic syndrome, hypertension, stroke, and atherosclerosis, in recent years. Several epidemiologic studies have linked hyperuricemia to each of these conditions. Uric acid-lowering therapies have been shown in some studies to prevent or improve certain aspects of the metabolic syndrome.

Keywords- Hyperuricemia.

INTRODUCTION

Hyperuricemia and gout are pathological conditions characterised by excessive or insufficient excretion of uric acid, a by-product of purine catabolism normally eliminated through urine. Both conditions are frequently linked to chronic illnesses like cardiovascular, renal, and renal-related diseases, metabolic syndrome, diabetes mellitus, hypertension, and diabetes mellitus, controlling uricemia and monitoring uric acid levels over time are therefore essential [1,2].

A metabolic disorder known as hyperuricemia, which causes a rise in serum urate/uric acid levels to as high as 6.8 mg/dL, is widespread throughout the world. Underexcretion, excessive uric acid production, or a combination of the two may be to blame. Another name for it is "rich man disease". [3]

Uric acid crystallisation and deposition in joints and surrounding tissues accompany the most frequent clinical manifestations of hyperuricemia, but the precise mechanism underlying uric acid-induced tissue damage is still unknown.

URIC ACID PHYSIOLOGY AND PHARMACOLOGY

Physiology:

The final byproduct of purine nucleotide catabolism is uric acid, which can be affected by a variety of t hings like diet or medications used to treat multiple cardiovascular risk factors or cardiovascular co-morbidities.These catabolism intermediate products are xanthine and hypoxanthine. The final two reactions in the biochemical chain that results in the formation of uric acid are catalysed by xanthine oxidoreductase- the transformation of xanthine to uric acid and xanthine to hypoxanthine. At the glomerulus, urate is freely filtered, but up to 90% of that filtered urate is reabsorbed. URAT1 and GLUT9 are the primary transporters in charge of tubular reabsorption . Urate oxidase, a very efficient enzyme for lowering uric acid levels in most mammals, converts uric acid to allantoin, which is highly water-soluble and eliminated unchanged in the urine. Unfortunately, urate oxidase does not function in humans, and as a result, hyperuricemia can occur in people. As a result, uric acid crystals can accumulate in human tissues and the urinary tract, causing chronic hyperuricemia-related diseases. [12]

PHARMACOLOGY

XO and URAT1 are two key enzymes involved in the metabolism and excretion of urate, and their activity is currently being modulated as part of management of hyperuricemia[12]. They fall into two categories: uricosuric drugs (such as sulphinpyrazone, probeneceid, and benzbromarone), which increase urinary uric acid excretion by preventing renal tubular re-absorption of urate.

uricostatic drugs (such as allopurinol), which reduce uric acid production through competitive inhibition of xanthine oxidase.

A XO inhibitor like allopurinol or febuxostat is frequently used to start primary urate-lowering therapy. Generally allopurinol is a safe medication, but about 2% of patients experience hypersensitivity reactions, which can occasionally be severe and fatal with a mortality rate of about 20%. Furthermore, the side effects of allopurinol can be both dose-related (like gastrointestinal intolerance and rashes) and individualised. When the dose of allopurinol is not properly lowered in patients with renal impairment, this happens especially often. However, allopurinol used at lower doses in these patients does not sufficiently control gout.

TYPES OF HYPERURICEMIA

Two categories can be used to classify the condition[12]:

1. Hyperuricemia of metabolic origin: This category includes 10% of cases and is characterised by excessive uric acid production. There is a faster rate of purine biosynthesis or a higher rate of nucleic acid turnover.

2. Hyperuicemia of renal origin: About 90% of cases fall into this category as a result of decreased renal excretion of uric acid, which may be caused by decreased glomerular uric acid filtering, increased tubular reabsorption, or decreased secretion.

AYMPTOMATIC HYPERURICEMIA

Individuals with hyperuricemia, particularly those with higher serum uric acid levels, are susceptible to gouty arthritis. However, prophylactic treatment is not recommended because the majority of hyperuricemic individuals never develop gout. Furthermore, before the initial attack, neither tophi nor structural kidney damage are visible.

Considering that specific routine treatment of asymptomatic hyperuricemia cannot be justified other than for the avoidance of acute uric acid nephropathy due to the inconvenience, expense, and potential toxicity of antihyperuricemic medications. But if hyperuricemia is identified, the root cause needs to be identified. If the condition is secondary, it is important to treat any related issues like hypertension, diabetes mellitus, obesity, etc.

PREDISPOSING FACTORS

Gouty arthritis may develop in hyperuricemic people, especially those with higher serum urate levels . The risk factors for hyperuricemia include reduced physical activity, a higher intake of purine-rich foods and alcoholic beverages, as well as smoking. Compared to patients in the other age groups (30 yrs. and 31-50 yrs.), a higher percentage of subjects with an age >50 yrs. reported having elevated uric acid levels.

There is growing evidence that hyperuricemia itself may be an independent risk factor for cardiovascular disease. Hyperuricemia is intricately linked with the metabolic syndrome (hypertension, glucose intolerance, dyslipidaemia, obesity, and increased risk of cardiovascular disease).

Few studies have shown that patients with T2DM (25.35%), metabolic syndrome (47.1%), obesity (44.6%), and HTN (37.32%) have higher prevalences of hyperuricemia than do healthy people (14%), suggesting that hyperuricemia is a significant and independent risk factor for CVD, cerebrovascular diseases, HTN, and T2DM[4,7].

The need of the hour is early screening of Serum Uric Acid levels in these patients in light of the rising incidence and high mortality rates of HTN and DM in developing and developed countries, as well as a positive correlation between serum uric acid levels and impaired renal function, high cardiovascular risk, and other disease-related complications. Given that the prevalence of hyperuricemia has increased globally this would aid in the early detection, prevention, and management of T2DM & HTN.

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LITERATURE RELATED TO HYPERURICEMIA

Clinical Studies

In- Vivo Study On Hyperuricemia-

Three Common Models Used –

-potsssium oxonate induced hyperuricemia models

-diet- induced hyperuricemia models

-pyrazinamide induced models

S.no.	Article	Author	Clinical studies/ Animal used	Outcome
1.	The role of hyperuricemia and gout in kidney and cardiovascular disease	N. Lawrence edwards	Rats	Showed a potential mechanism of microvascular changes leading to endothelial dysfunction precursor to both cad, htn
2	High-protein diet induces hyperuricemia in a new animal model for studying human gout	Fan hong, aizjuan zheng	Chicken	Development of hyperuricemic model
3	Anti-gouty arthritis and anti hyperuricemia effects of sunflower head extract in gouty and hyperuricemia animal models	L li, mteng,y qu	Rats	Sunflower head ethanol extracts suppress the swelling of ankles in inflammation.
4	Hyperuricemia and risk of strokes: a systematic review and meta-analysis	Seo young kim, james p guevara	Clinical- prospective cohort study	Hyperuricemia may modestly increase the risks of bpth stroke incidence and mortality
5	Hyperuricemia and incident hypertension : a systematic review and meta-analysis	Peter c grayson, seo young kim	Clinical- cohort study	Hyperuricemia is associated with an increased risk for incident hypertension, independent of traditional hypertension risk factors, greater in younger individuals and women.
6	Community based epidemiological study on	Kuan- chia lin,	Survey study	Alcohol consumption and central obesity were independent predictors of

	hyperuricemia and gout in kin- hu			gout among hyperuricemic subjects irrespective of uric acid levels
7.	Clinical outcome of hyperuricemia in iga nephropathy : a retrospective cohort study and randomised controlled trial	Yongjun shi, wei chen, diana jalal.	Retrospective cohort study	Hyperuricemia predicts the progression of iga nephropathy independently of baseline estimated gfr
8	Relations of hyperuricemia with the various components of the insulin resistance syndrome in young black and white adults: the cardia study	Wolfgang rathman, Ellen funkhouser, alan r. Dyer , jeffrey m. Roseman	Cross- sectional study	-bmi , fasting insulin and triglycerides were higher and hdl was low. - bmi showed positive corelation with uric acid
9	Asymptomatic hyperuricemia without comorbidities predicts cardiometabolic diseases: five year japanese cohort study	Masanari kuwabara, koichiro niwa, thomas jensen	Cohort study	Asymptomatic hu
10.	Role of hyperuricemia in vascular disorders	N lawrence edwards	Review study	
11.	Effect and mechanism of total saponin of dioscorea on animal experimental hyperuricemia	Guang-liang chen, wei wei, shu- yun ju	Mouse and rats	There was significant lowering of sua .
12.	The association between purine – rich food intake and hyperuricemia : a cross-sectional study in chinese adult residents	Sumaiya aihemaitjiang, yaqin jhang,m li zhang , jiaoyang, chen ye, wei zhang, mairepati halimulati	Cross – sectional study	The intake of animal derived food and legumes are associate with occurrence of hyperuricemia . Controlling of diet is beneficial in controlling risk of hyperuricemia
13.	The establishment and characteristics rat model of atherosclerosis induced by hyperueicemia	Zhen liu, tong chen, haitao niu, wei ren, xindeli, lingling cui, changgui li	Wistar- kyoto rats	High uric acid levels may cause atherosclerosis in rats

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HOMOEOPATHIC LITERATURE REVIEW -

S.no.	Article	Writer	Type of study/ animals used	Outcome
	Case report on hyperuricemia presenting as one- sided disease	Renu bala , Amit srivastava	Case sudy	Anti- miasmatic treatment with symptomatic treatment improved the case
2.	Homoeopthic management of hyperuricemia in primary gout : a randomised single blind placebo controlled study	aman deep kumar, anupam kumar	Prospective randomised Single blind	Significant reduction in sua and vas score in medicinal group in contrast to placebo group
3	Individualised homoeopathic medicines in treatment of hyperuricemia: evalustion by double – blind, randomised ,placebo controlled trial	Priyanka ghosh, subhashish ganguly, shyamal kumar mukherjee, souvik dutta	Double–blind rct	Individualised Homoeopathic medicines showed better results than placebo group in reducing sua
4	Treatment of hyperuricemia with homoeopathic medicine lycopodium calvatum: case series	Dr. Sitara perveen, kr mansoor ali , bj maya, t nithya	Cases	Lycopodium clavatum caused success Ful alleviation of hu
5.	Anti- hyperuricemic potential of phododendron tomentosum harmaja syn. Ledum pal. 30c and 1m in potassium oxonate induced rats	Shifa shaffiqe, saeed ahmed, tayeba rehman, waheed mumtaz, haseeb anwar	In – vivo study- rats	There was much reduction in sua levels.
6.	Individualised homoeopathic medicines and urtica urens q in treatment of hu : an oen rndomised prgmatic trial	Chintamani nayak, nivedita pattnaik, abhijit chattopadhyay, pankhuri misra, kaushik bhar	Randomised trial	No definite conclusion
7	a case report of hyperuricemia with nephrolithiasis treated with homoeopathy	G. Pooja	Case study { modified naranjo criteria score}	Lycopodium was given which showed favourable results .

CONCLUSION

Globally, the prevalence of hyperuricemia is increasing and is now estimated to be 25.8%. The drugs that are currently available in modern medicine to lower uric acid levels have negative side effects like vomiting, adverse drug reactions, hepatatorenal toxicity, gastrointestinal bleeding, etc. Homoeopathy can function as an alternative medical system with few side effects in such a situation.

The list of drugs mentioned in the book includes Ledum Palustre, Urtica urens, Lycopodium, and many others. There are very few evidence-based studies on homoeopathic medicines, despite the fact that it is claimed that they are effective in treating hyperuricemia. Homoeopathic medications can be a good substitute for other hypouricemic drugs because they can normalise as well as reduce uric acid levels.[9,10]

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