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A REVIEW AND CASE STUDY TO DESIGN A PROGNOSTIC MODEL FOR GOUT IN PATIENTS WITH ROSACIC ACID

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ABSTRACT:

The intent of the present case study was to scrutinize the data of subjects in order to predict the onset or progression of gout. It is evident that Hyperuricemia on the long run will lead to complications such as gout in other words the condition where uric acid crystals are amassed in the joint. The data was collected for attributes such as Uric acid levels, Creatnine levels, alcohol intake, Protein consumption, Gender, Cholesterol levels, Bile content, etc in order to determine the effect of one feature on another and to understand their influence on Uric acid variations. An attempt was made to build a prognostic model which could predict the onset of Hyperuricemia or gout, in patients who had high levels of lithic acid or uric acid. The study also was based on the fact that hormones such as oestrogen influenced the stimulation or inhibition of uric acid.

KEYWORDS: Lithic acid, Prognostic Model, Gout, Purines, Hyperuricemia, Hypouricemia,

INTRODUCTION:

Lithic acid or Rosacic acid habitually called as Uric acid is a superfluous by-product that is spotted in blood. It is liberated by the disintegration of chemicals known as Purines. Under normal functioning of human body Rosacic acid is dissolved in blood, absorbed or filtered in the kidneys and expelled out through waste excretion whereas on the contrary when high levels are consumed through the diet the body fails to expel the excess quantities; which remains in the blood causing the condition called Hyperuricemia. Lithic acid or Rosacic acid, a heterocyclic compound with the formula $C_5H_4N_4O_3$ is predominantly found in all organisms on Earth in either soluble or insoluble form. In specific organisms, it may also be expelled out as ammonia. For obvious facts it is known that compounds with high levels of nitrogen compounds are designated under the category 'Nitrogenous compounds', so uric acid containing nitrogen falls into this bracket[1-2]. Table (1.1) shows the various organisms and the form of uric acid excreted out.

Table 1.1: Forms of Uric acid in different organisms

Organism	Uric Acid Form

Birds, Reptiles, and Insects	Nitrogenous waste product
Mammals	Final form of purine metabolism
Microorganisms (fungi, Enterobacteria, Bacillus substilis)	Ammonia

The UA is a weak acid that mostly exists as a urate anion at normal blood pH values. The last metabolites of chronic and external purine mononucleotide catabolism, hypoxanthine and xanthine, are stimulated by XOR [3]. Rosacic acid is mainly absorbed into the liver, intestines and other tissues such as muscle, renal and vascular endothelium as a final result of a unique pool of purines, found mainly in animal proteins. [4] There is a very close relationship with the outbreak of gout and hyperuricemia with increased concentration or excretion of uric acid from the body. Plants and microorganisms also have an enzyme that can degrade even though they are different. Inhibition of xanthine oxidase and xanthine oxido-reductase and in addition inhibition of oxidation of hypoxanthine to xanthine leading to uric acid depletion in purine metabolism is mediated by many metabolites produced in plants. Similarly, a group of enzymes that degrade uricase, allantoinase, allantoicase and urease synthesized by bacteria to help promote the depletion of lithic acid to ammonia [2].

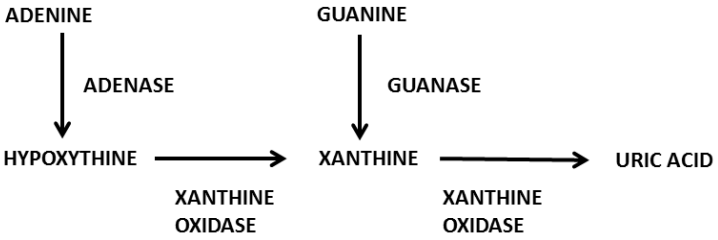

Figure 1.2: Uric acid formation

Hyperuricemia is being investigated for being associated with a higher risk of coronary heart disease, a rate of recurrence of coronary artery disease. Both hyperuricemia and asymptomatic gout are considered independent risk factors for the onset and progression of heart disease (CVD), CVD death, and all other underlying causes, in different populations [5].

Gout is a prevalent arthritic condition that has a significant impact on function and quality of life. Given the link between gout and dehydration, it's unclear if this disorder causes increasing paralysis or if gout is a factor. Functional disability, standard health-related quality of life (HRQoL), and higher mortality have all been found in gout patients. [5]. As a result, gout has become a huge public health issue. Gout, on the other hand, is linked to a number of heart disease risk factors, including high blood pressure, dyslipidaemia, insulin resistance, obesity, and renal failure. It's uncertain if the impact of gout is connected to the disease or stems primarily from hazards and medical conditions. [6] Gout is a great way to show if you have a uric acid problem. It's a well-understood and well-defined kind of arthritis. Its epidemic has resurfaced. New insights into the pathophysiology of hyperuricemia and gouty arthritis, which is complex and unending, have been discovered, allowing for a better knowledge of the condition. The importance of genetic predisposition is becoming clearer. Asymptomatic hyperuricemia, acute gouty arthritis, periodontal disease, and chronic gout are the different types of gout. [7]

i. Mechanisms for Hyperuricemia:

The fact that hyperuricemia frequently occurs before the onset of CKD shows that causes other than renal failure may play a role in the development of high uric acid levels. Different strategies have been shown to work in studies. CKD obesity and metabolic syndrome are two of the most harmful variables, both of which are strongly linked to hyperuricemia, which can be induced by insulin resistance and insulin effects on micturition. Renal vasoconstriction is linked to high filtration pressure, which leads to uric acid retention. Recent evidence, however, reveals that elevated serum uric acid precedes these disorders and may not be the root cause of hyperuricemia.

ii. Inflammatory Responses in Hyperuricemia:

The UA makes inflammatory responses. The inflammatory response caused by Hyperuricemia resolves kidney damage by altering blood vessels and kidneys. The UA has the ability to add monocyte chemo proteins (MCP-) 1 to smooth muscle cells, suggesting that they may contribute to metabolic changes associated with high blood pressure and cardiovascular disease. The UA also contributes to kidney damage by increased cell proliferation caused by 2 production and increased exposure to C-forming protein (CRP). Two-thirds of urate excretion occurs in the kidneys and the other is excreted through the intestinal tract. Decreased secretion activity of transporter ABCG2 leads to a decrease in uric acid secretion leading to an increase in serum uric acid levels and improved kidney excretion. Uric acid crystals are insoluble and therefore require specific membrane transmitters to cross the cell membrane. Of these transporting urate Transporter / channel (URAT) especially URAT1 and anion carriers (OAT1 and OAT3).

iii. Lithic acid regulation by Genes:

In the apical membrane of the renal tubules, the SLC22A12 gene encodes the transporter URAT1. Another gene implicated in the release of UA is SLC2A9. Transporter proteins are absorbed into the tubule lining of the kidney. Polymorphism in both genes causes a decrease in UA emissions, which leads to a rise in SUA levels.

iv. Physiological role in humans:

Lithic acid at high levels however harmful is a role for the body in humans compared to other mammals. Guessing has marked the longest human life span of all mammals as it restores the deprivation of the ability to produce ascorbic acid in primates that occurs through human birth. Genetic modification has led to the production of lithic acid as a circulating anti-oxidant compound after the genetic modification of L-gulonolactone oxidase (GULO) that supplied ascorbic acid from sugar.

Most of the serum uric acid is filtered through the kidneys of glomeruli, and about 90% of the filtered uric acid is returned to the body, meaning it plays a major role in the body. Uric acid provides more than half of the antioxidant capacity in human plasma. Uric acid is a peroxynitrite and active oxygen (ROS) scavenger and antioxidant. When they operate as antioxidants, high uric acid levels are readily available in the cytosol of normal human and maternal cells, particularly in the liver, vascular endothelial cells, and nasal passages. [4]

v. Metabolism:

Purines the mainstay of Deoxyribonucleic acid (DNA) on inculcation produces Lithic acid as its dominant outcome. Enzyme-catalysed reactions help reprocess the earlier results of purine digestion, although uric acid cannot. If and only if lithic acid is disintegrated into ammonia and carbon dioxide, can it be used for protein engendering inclusive of purine reunification. There comes a stage in an animal where the further breakdown of lithic acid is arrested and must be excreted out immediately. [3] Such criteria can be fulfilled by presence of uric acid-splitting bacteria. Studies have proven to indicate the presence of uric acid-splitting bacteria in hum gut

which maybe be absent or amended in gout patients. All the issues stated with reference to removal of uric acid is its meagre solubility in water.

vi. Micellar Electrokinetic Capillary Chromatography:

About 15 clinical birth control computers are found in the nonprotein nitrogen (NPN) component of the blood. The four principal NPN components are urea, uric acid, creatine, and creatinine, which are frequently used in clinical settings. They're used to keep track of how well your kidneys are working. In the blood, urea makes up around half of the NPN. Kidney transplantation, protein content in the diet, and the extent of protein catabolism all have an impact on plasma urea levels. Urea levels can be raised by a high-protein diet, a fever, a severe sickness, or stress. Urea levels can be reduced by low-protein diets or high amounts of protein synthesis throughout late pregnancy and youth.

FACTORS RELATED TO ROSACIC ACIDS:

a. Homeostasis:

Homeostasis is a condition in which the internal, physical, and chemical structures of a person can be stored through organs such as the kidneys. Uric acid and water and ions are extracted to maintain homeostasis or a balanced cycle. Under uncertain conditions, uric acid levels can suddenly vary due to two factors: Exogenous and Endogenous factors. The latter include high protein foods, alcohol use, urine use, etc. and the latter include genetics, iron supplementation, insulin resistance, etc. [1] Various facts have been found to resolve the role of Uric acid in maintaining homeostasis, however inconclusive studies have not yet been confirmed.

b. Gender:

Lithic acid can be seen in both men and women but in varying degrees. Numerous experiments were performed to classify and analyse the basics of these variations at levels. Significantly, the estrogen found in women was found to promote the release of lithic acid. It is very clear that this hormone is not present in men which allows why men have higher levels of Serum Uric acid than women. [1] In normal working men, the apparent amount of uric acid in the blood of men ranges from 3.74-6.2 mg / dL, while that in women ranges from 4-5.3 mg / dL.

c. Food:

Sustenance required for mankind contains various forms of carbohydrates, sugars, proteins, fibres and fats. Amidst these purine and protein rich foods are said to be avoided in those patients who suffer from hyperuricemia, [1] because these foods produce uric acid as an end product; and it is clear that excess quantities lead to crystal formation in joints. Table (1.2) shows the list of edibles that can be consumed and ones that must be avoided.

Table (1.2): List of food consumed and avoided

Avoided	Consumable
Meat- liver, kidney , Red meat, Seafood, sweetbreads	Vegetables such as beans, lentils, peas, legumes
Vegetables such as cauliflower, spinach, and mushrooms	Dairy products such milk, cheese, yogurt
Alcohol like Beer and grain	Beverages such as black tea,

liquors	coffee

The introduction of purine-rich foods, such as cooked or processed meats and shellfish, is a major role in the increase of uric acid precursors. Purine-rich vegetables such beans, lentils, mushrooms, peas, legumes, and dairy products are safe for hyperuriceamia and gout, but may not be permitted in gout patients. Furthermore, diets high in vitamin C, low-fat dairy products, and vegetable oils including olives, sunflowers, and beans have been linked to a lower incidence of hyperuricemia and gout. Vitamin C has been shown to stimulate renal uric acid release, suggesting that it could be utilised as a gout treatment adjuvant. [7]

d. Alcohol:

Ethanol-rich alcohol is said to be one of the first causes of gout flares. The reasons are that one-ethanol consumption produces uric acid as a product and two - the body prioritizes the removal of alcohol and not uric acid. [1] In some cases, wine has been shown to lower lithic acid levels but has led to the onset of gout, the cause remains a mystery.

Alcohol is a well-known gout risk factor. Alcohol consumption is linked to the amount consumed, according to studies. Furthermore, the risk of gout and hyperuricemia varies depending on the type of alcohol used. When compared to alcohol, it is a key contributor in increasing the risk of gout. Wine, on the other hand, posed the lowest danger of all the alcoholic beverages. [7]. The current study divided the participants into drinkers and non-drinkers, and discovered that only the drinking group had a positive relationship between uric acid levels and systolic/diastolic blood pressure. We confirmed that those who drank more had considerably higher uric acid levels than those who did not [8-9].

e. Genetics:

Hyperuricemia may be caused by genetic reasons in cases of chronic kidney disorders, diabetes, obesity etc nonetheless one may not suffer from hyperuricemia or gout if their parent or close relative suffered from it. Another genetic cause maybe due to the undermined functionality of the gene called Solute Carrier Family 2 Member 9 that engenders the protein glucose transporter 9 (GLUT9) which is responsible for the efflux of urates from the cells [1].

INTER-RELATED DISORDERS OR DISEASES:

A. Renal disorders - Contrast-induced nephropathy, Kidney injury:

Hyperuricemia may be caused by either high lithic acid quantity in blood, dysfunctional kidney uric acid removal or a combination of these two. Hyperuricemia increases the menace of severe kidney damage disrupting the contractile function of intraglomerular mesangial cells, and causes damage to mesangial cells and proximal tubules epithelial cell possibly with TLR 4-dependent regulation of NLRP3 and IL-1b. Hyperuricemia has also been shown to be an independent risk factor for chronic kidney disease type 2 diabetes by endothelial cell damage and the release of the HMGB1 alarm, which stimulates the TLR to produce cytokines that attack inflammation and chemotactic, muscle-to-muscle growth. [4] In addition, uric acid can accumulate in the kidneys, leading to the formation and formation of stones. Kidney stones and urinary tract infections are the most common urinary tract problems. Uric acid stones make up 10% of all kidney stones and are the second most common cause of urinary stones after calcium oxalate and calcium phosphate calculi. The most important factor in the risk of uric acid crystallization and stone formation is low pH (less than 5.5) due to urinary uric acid deficiency. The main causes of low pH next to high uric acid excretion are chronic diarrhoea, dehydration, and diabetic ketoacidosis.

Cardiovascular diseases, Hypertension, Hypertrophy and Myocardial infarction

Nitric oxide given due to endothelial dysfunction damages the heart, the whole process occurs due to lithic acid. It can also cause vascular sensitivity or apoptosis of cells that lead to congestive heart failure. The first evidence of a relationship with lithic acid and an increase in BP was seen in the 1870's by Frederick Akbar Mohamed, a British physician [10].

B. Respiratory disorders

Chronic obstructive pulmonary disease (COPD) is a complex and complex disease, with oxidative stress and inflammation involved in its development. Uric acid (UA) can produce anti-oxidative, pro-oxidative or pro-inflammatory effects, depending on the specific context.

Insulin resistance and diabetes type 2

Genetic factors indicate that diabetes is a major cause of hyperuricemia. In contrast, abnormal levels of uric acid can also be used as an indicator of type II diabetes and Insulin resistance because various studies show that 1/3 of diabetes cases can be reported with high serum uric acid (SUA) levels. [4] Over the next few centuries, increased consumption of sugary beverages has led to obesity, insulin resistance, diabetes, and increased SUA levels. This leads to low levels of ATP (adenosine triphosphate) and phosphates in the inner cell regions leading to catabolism of adenosine monophosphate (AMP) deaminize to inosine monophosphate eventually forming lithic acid. Inflammation of the endothelial cells, renal, vascular smooth muscle, Langerhans cells of the pancreas are experienced due to high SUA levels.

DIAGNOSIS OF LITHIC ACID:

a. Asymptomatic hyperuriceamia:

Asymptomatic hyperuriceamia is the first juncture amongst the four stages of gout. In certain cases patients tend to suffer from acute gouty attack which is the next stage due to the fact that asymptomatic hyperuriceamia is symptomless and cannot be detected easily unless the patient has frequent full body profiles conducted or the patient was scanned for other complications and this hyperuricemia was discovered [5].

b. Acute gouty attack:

These assaults are typically monoarthritic, lasting a few hours and causing substantial disruption of the basic symptoms of inflammation, such as redness, fever, soreness, swelling, and loss of function. Skin signs are uncommon in broad areas like the knees and ankles, but swelling and pain can be severe. The first MTP of gout is lower extremity preference, which is the most prevalent cause of acute gout, called as podagra. The tarsal and metatarsal joints, as well as the ankles, knees, wrists, MCPs, and middle hand joints, may affect other joints. The hip and shoulder joints are frequently affected. The involvement of the vertebral column is uncommon. [5] Olecranon bursitis and Achilles tendonitis can both cause soft tissue inflammation. Arthritis that affects more than one joint at the same time is uncommon. It's particularly common in people who haven't had their gout treated or in postmenopausal women. Fever, headaches, and malaise are common constitutional complaints. Until proven otherwise, the joint should be treated as if it had septic arthritis. When dealing with such instances, extreme caution is advised, as septic arthritis can develop when gouty arthritis is combined with the presence of MSU crystals. Gouty attacks, on the other hand, can be minor due to low-grade inflammation.

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c. Chronic tophaceous gout:

Untreated diseases continue to destroy joints through the formation of affected tissues. Tophus is a weight made up of large accumulated amounts from untreated gout. It can be near the joints of the ears, lower tissues or skin. Disease manifestations and disease control. According to macroscopically, the top contains a white chalky substance. Tophi can lead to the destruction associated with disability. Soil erosion is also possible as toxic growth reaches. Separating peaks from other tumours, such as rheumatoid nodules, osteoarthritic Heberden's and Bouchard's nodules, lipomas, or other tumours, is necessary for future therapy. This is easily accomplished by doing a simple needle biopsy, which will reveal MSU crystals, a gout symptom. Gout diagnostics in clinics is widely utilised around the world, particularly in underdeveloped nations with inadequate resources. Clinical diagnosis, on the other hand, appeared to be less sensitive and specific when compared to the availability of small amounts of substances. Head development might be a result of a previous clinical manifestation or it can occur at the start of a disease research. It can be an excellent sign of gout depending on where it is. However, before focusing on a specific diagnosis of gout, other arthritides linked with nodules must be checked out. [7]

d. Radiological diagnosis:

The significance of considering gouty arthritis cannot be overstated. It is critical to be diagnosed and monitored during clinical activities. Its utility as a clinical trial effect measure is also expanding. Technology has recently had an impact on the stage, including the type of viral name.

e. Conventional Radiography (CR)

It is a widely used method of medical practice; however in the early stages of the disease it does not work very well. Radiographic changes can be missed for at least 10 years after the first gouty attack. During the onset of gout, radiographic images are usually normal or may show inflammation of the soft tissues around the affected joints, but the first subtle lesions such as minor erosion and tophi are difficult to detect. [7]

f. Ultrasound:

Recently, advances in U.S. technology (Equipment, converters, techniques), has encouraged its use by orthopaedic surgeons in diagnosing and controlling gout. Nestrova and Foder noted crucial signs of US use in crystal-arthritis in their good review. [7] These include detecting joint and synovitis, distinguishing between active and inactive synovitis, cartilage learning, defining line erosion and osteophytes, tender testing, crystalline implant testing, US-directed procedures (diagnostics and/or treatment), monitoring the onset of the disease, and assisting in the differentiation of other arthritides.

PROGNOSTIC ROLE OF UA IN HYPERTENSION:

The combination of hyperuricemia and other cardiovascular risk factors has been one of the most difficult aspects of determining the link between UA and high blood pressure (CV). SUA levels, for example, frequently rise in persons who suffer from metabolic syndrome, a well-known risk factor for high blood pressure. Several epidemiologic studies have employed thorough analysis and correction to evaluate whether the UA was an independent risk factor for high blood pressure. Hyperuricemia is found in 25-60% of persons with untreated high blood pressure, according to several short-term studies, and SUA levels are linked to high blood pressure. Longitudinal investigations have verified an unexpected number of UAs in hypertension, indicating that greater SUA levels are linked to a higher risk of hypertension. Major meta-analyses have backed up these findings, however Mendelian Randomization studies have yet to demonstrate a link between SUA and high blood pressure. Surprisingly, genetic-based organisational study has revealed that XOR

genetic variants are linked to high blood pressure, but not large urate carriers. Because XOR is involved in the creation of UAH within cells, the findings of those genetic research could have a significant impact on the formation of internal UA rather than SUA in the development and progression of high blood pressure. Plasma XO activity is linked to CV outcomes, with the exception of SUA standards, which supports this notion. Furthermore, Boban et al. discovered that XOR is used in investigations with high blood pressure. [11]

POPULATION:

The subjects considered in the study were males and females aged between 25-50 years of age. Here we have taken into consideration 15 male and 15 females.

METHODS:

The entire conduction was carried out in such a manner that subjects who were on treatment for Uric acid were taken into consideration and factors such as age, alcohol intake, protein intake, medicine dosages were noted as the preliminary step [12-30].

Table 1.3: Data collected for 30 subjects

	Age	BP	Urea	Uric Acid (mg/dL)	Sex	Creatnine (mgs/dL)	Urine pH	T4 (mcg/dL)	T3 (ng/dL)	Total Cholesterol (mgs/dL)	Total liver protein (mg/dL)	Hgb (gms %)
S1	54	110/80	35	5.3	M	1.07	6	10.4	99.8	229	6.9	16
S2	45	115/80	21.2	5.3	F	0.97	4.8	12.1	100.3	189	7.1	12.8
S3	22	110/80	26.7	7.9	F	1.05	5.7	10.1	110.5	227	6.6	7
S4	30	120/70	29.5	6.3	F	0.88	5	11.6	115.1	147	7.06	7.5
S5	25	110/70	33.6	4.7	M	1.1	6.1	106	121	123	7.1	19
S6	46	115/80	28.4	6.2	M	0.96	5.7	12.3	112.6	163	6.3	17
S7	57	120/80	26.8	6	M	1.03	6.5	9.6	100.5	232	6.95	13.4
S8	60	110/80	30.5	6.7	M	0.89	7.5	10.2	98.6	226	7.3	14
S9	38	11	32.6	4.9	F	0.95	6.1	12.6	124	232	7.13	5

		5/80										
S10	53	110/80	22.4	5.7	M	1.01	6	10.4	100	193	7	14.9
S11	51	110/80	35.1	5.23	F	0.99	6	11.6	112.6	147	7.2	13
S12	42	110/80	27.2	7.3	F	0.87	5	12.4	100.2	205	6.4	8.8
S13	55	120/80	24.9	6.1	F	0.94	6	10.3	114.6	195	7.16	6.6
S14	33	110/70	23.9	5	F	0.97	5	11.4	115.2	232	6.5	11.5
S15	41	120/80	34.1	6.4	M	1.03	5	12.1	124.3	114	6.77	14
S16	56	120/90	28.6	5.3	M	0.9	5.5	12.4	101	197	6.91	16.5
S17	26	120/70	18.5	5.9	M	1.03	6	11.8	125.4	163	7.09	13.5
S18	38	110/70	16	5.8	F	1.06	5	12.6	112	156	6.32	8.6
S19	43	110/80	29.3	6.5	M	0.88	6	10.6	114.5	115	6.45	14.5
S20	60	120/80	26.7	5	M	0.92	6	12	99.7	178	7.6	12
S21	34	110/80	16	5.8	F	1.12	5	10.5	127	146	7.18	8.5
S22	50	110/80	19	4.8	M	0.8	6	11.38	114	126	7.1	15.3
S23	19	120/70	18.6	6.9	M	0.77	5	11.7	129	137	7.19	8.2
S24	55	110/70	26.1	6.7	F	0.97	6	12.2	126.4	115	11	9.5
S25	52	120/8	23.6	6.1	F	0.96	6	10.7	110	177	7.26	10.1

		0										
S26	40	120/70	16.8	5.6	F	0.84	5	12.5	112.6	200	7.8	12.6
S27	34	120/80	26	6.7	M	0.79	5	12.1	116	169	7.21	15
S28	37	110/80	23.2	5.7	F	1.06	5	11.1	126.3	143	6.1	8.1
S29	49	120/70	33.4	7.6	F	0.84	5	12.4	111.3	169	7.06	7.8
S30	55	110/80	17	6.3	M	1.06	6	10.1	130.6	179	6.6	16.3

Table 1.4: Consumption of Alcohol, Protein Diet, Medication

Sl.No	Uric Acid (mg/dL)	Alcohol	Protein Diet	Medication
S1	5.3	1	1	1
S2	5.3	0	0	1
S3	7.9	1	1	1
S4	6.3	0	1	1
S5	4.7	0	1	0
S6	6.2	0	1	0
S7	6	1	1	1
S8	6.7	1	0	0
S9	4.9	0	0	1
S10	5.7	0	0	0
S11	5.23	1	0	1
S12	7.3	1	1	1
S13	6.1	1	0	1
S14	5	1	0	1
S15	6.4	0	1	1
S16	5.3	1	0	1
S17	5.9	1	0	1
S18	5.8	1	0	1
S19	6.5	1	0	0
S20	5	0	0	0
S21	5.8	1	0	1
S22	4.8	0	0	1
S23	6.9	1	1	1

S24	6.7	0	0	0
S25	6.1	0	1	1
S26	5.6	1	0	1
S27	6.7	1	0	1
S28	5.7	1	0	0
S29	7.6	1	1	1
S30	6.3	1	1	0

These processes may not be responsible for the increased urate production found in people with primary gout [13]. The basic structure of renal histology has been demonstrated to be altered by high levels of uric acid, which contributes to severe renal failure. Despite the fact that uric acid levels have been linked to chronic kidney illness, it can be used as a symptom [14]. However, in persons with chronic kidney disease (CKD) and beyond, inconsistent impacts on kidney outcomes have been documented [23]. Renal arteriolopathy, tubulointerstitial fibrosis, and kidney inflammation are all linked to hyperuricemia. Several epidemiological studies have found that uric acid is a key risk factor for the development and progression of renal disease in diabetic and non-diabetic people [25]. Uric acid, through renal vasoconstriction and systemic high blood pressure, can modulate parts of the link between high blood pressure and kidney disease [24]. Although emerging data suggests that uric acid is an inflammatory molecule, the mechanisms of uric acid injury remain unknown. [17] Following surgery and hypotension, comparative nephropathy (CIN) has historically been a prevalent cause of acute kidney damage (AKI) in hospitalised patients, with an elevated risk of death during short and long-term follow-up. In the latter case, we wanted to see if acid, uric acid, serum bicarbonate, and serum uric acid levels may affect CIN growth in patients undergoing coronary angiography. [21]

RESULTS AND DISCUSSION:

Table 1.5: Model for Subject 1

Feature	Coefficient	Value (Y=1,N=0)	Coefficient X Value
Ln Urea (1 if urea<38)	0	3.555348061	0
Ln Uric Acid (mg/dL)	1	1.667706821	1.667706821
Gender (M=1)	1	1	1
Ln Creatnine (mgs/dL)	3	0.067658648	0.202975945
Ln Urine pH	1	1.791759469	1.791759469
Ln BP	2	0.318453731	0.636907462
Ln Cholesterol (mgs/dL)	2	5.433722004	10.86744401
Ln liver protein (mg/dL)	3	1.931521412	5.794564235
T4 (mcg/dL)	1	2.341805806	2.341805806
Ln Haemoglobin (gms %) [Male age <60 and female age > 50 then Hgb=1] else Hgb=2	1	2.772588722	2.772588722
Ln Age [30<Male age <70] =2 [45 < Female age] =2	2	3.988984047	7.977968093
Ln Age X Ln BP	4	1.270306853	5.081227412

Ln Age X Ln Haemoglobin	2	11.05981218	22.11962436
Sum			62.25457233
Avg			4.788813256
Round-off			4.79

Table 1.7: Model for Subject 24

Feature	Coefficient	Value (Y=1,N=0)	Coefficient X Value
Ln Urea (1 if urea<38)	0	3.261935	0
Ln Uric Acid (mg/dL)	1	1.902108	1.902108
Gender (M=1)	1	0	0
Ln Creatnine (mgs/dL)	3	-0.03046	-0.09138
Ln Urine pH	1	1.791759	1.791759
Ln BP	2	0.451985	0.90397
Ln Cholesterol (mgs/dL)	2	5.433722	10.86744
Ln liver protein (mg/dL)	3	1.931521	5.794564
T4 (mcg/dL)	1	2.501436	2.501436
Ln Haemoglobin (gms %) [Male age <60 and female age > 50 then Hgb=1] else Hgb=2	1	2.397895	2.397895
Ln Age [30<Male age <70] =2 [45 < Female age] =2	2	4.007333	8.014666
Ln Age X Ln BP	4	1.27615	5.104601
Ln Age X Ln Haemoglobin	2	11.11069	22.22137
Sum			61.40844
Avg			4.723726
Round-off			4.73

A comparison of male and female was taken to demonstrate the risk of lithic acid in both. Studies have proved that male are at a higher risk of suffering from gout attacks then females because of the presence of oestrogen which suppresses the effect of uric acid, on the contrary women in menopause i.e. above the age 50 suffer from gout attacks as much as men at the age 30-60. The factors such as haemoglobin are taken into account in order to validate the fact that anaemia acts as a factor which aggravates gout attacks.

Age and BP are inter-related due to the fact that BP increases with age so a coefficient of 2 is assigned for the two factors. Creatnine and Liver protein being related to the kidney and its removal play as an important factor in uric acid levels therefore; a coefficient of 3 is assigned. A higher coefficient indicates a higher risk from that factor whereas; a lower indicates a lower risk.

CONCLUSION:

It can be concluded that attributes such as age, history of disease, sex and genetic factors influence the abundance of constituents of uric acid in plasma. Due to distinguished physiological

circumstances of the body the features vary based on gender. It is known for a fact that oestrogen levels are moderate in women only till the premenopausal stage is attained post this stage there is no large divergence in the lithic acid levels in men and women.

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