Correlation between Uric Acid and Lipid Profile in Untreated Dyslipidemic Patients

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Abstract

Background: The effects of cholesterol and statin therapy on serum uric acid (SUA) concentration are poorly known, and the latter's effects are even less clear. A mean atorvastatin dosage of 24 mg/dl satisfies the American Cardiovascular Assessment Campaign management objectives and dramatically lowers prevalence of chronic in individuals with cardiovascular events, according to the Greek Drug and Cardiovascular Evaluation research. We compare the temporal evolution of SUA levels in patients receiving standard treatment who received insufficient statin therapy (12 percent received statins) to patients receiving formalised care who received atorvastatin therapy almost exclusively (98 percent).

Methods: In this study 160 individuals with abnormal lipid profiles in their blood were enrolled to investigate the connection between lipid profile and uric acid in dyslipidemic patients (dyslipidemia). It was a 5-month cross-sectional study conducted at Dr Abdul Sattar Lab Sialkot using a convenient sampling method. The uric acid, total cholesterol, triglycerides, LDL, and HDL cholesterol levels of enrolled participants were measured. In short, we performed uric acid and lipid profile tests on under-observation samples to investigate the association between uric acid and lipid profile parameters in the enrolled (dyslipidemic) individuals.

Result: This research looks at people between the ages of 20 and 60. The Graph shows that (15) patients are between the ages of 20 and 30, (46) patients are between the ages of 31 and 40, (74) patients are between the ages of 41 and 50, and (25) patients are between the ages of 51 and 60. Patients of both sexes are covered. It was found that there exist significant positive relationship between uric acid and lipid profile in dyslipidemic patients. This study shows a positive correlation between LDL, triglycerides, total cholesterol and uric acid whereas a negative correlation was observed between HDL and uric acid. According to the current results, when uric acid rises, Total Cholesterol, Triglycerides, and Low Density Lipoproteins (LDL) rise as well. But High Density Lipoproteins (HDL) falls with the increase in uric acid levels. As a result, this study may be useful in reducing the incidence of related cardiovascular morbidities, and we will be able to predict dyslipidemia more accurately, which may further leads to CVDs. As the rate of CVDs rises in Pakistan, it is becoming increasingly necessary to investigate the factors that are directly linked to the disease.

Conclusion: This article's objective was to investigate any connections between Uric Acid and Lipid Profile. Because dyslipidemia predicts the risk of coronary artery disease, so uric acid levels should be considered in these individuals for more complete risk factor prediction and treatment. Increased levels of lipid profile parameters can lead to serious heart diseases, and the only way to avoid this is to get a quick diagnosis of the disease.

Keywords: cholesterol, serum uric acid (SUA), lipid profiles, Low Density Lipoproteins.

INTRODUCTION

Dyslipidemia has long been recognized as a key metabolic event that increases the risk of atherosclerosis and cardiovascular disease (CVD). It shows up as an increase or decrease in lipoprotein concentrations in the blood [1]. Mean cholesterol, LDL cholesterol, and triglyceride levels are all raising in Pakistan. Dyslipidemia has long been recognized as a significant metabolic event linked to atherogenicity and cardiovascular disease (CVD). It manifests itself as an increase or decrease in lipoprotein plasma concentrations [2]. In humans, serum uric acid is produced as an oxidation result of purine catabolism. One of the most serious effects of hyperuricemia is an increase or decrease in uric acid production and excretion. CVD has become increasingly common in Pakistan during the last two decades, accounting for 24 percent of all fatalities among persons aged 35 to 80.
years [3]. Inflammatory mechanisms are increasingly being acknowledged as a factor in CVD pathophysiology. Several circulating inflammatory markers have been intensively examined as risk predictions. Uric acid was originally thought to be a harmless metabolic byproduct, but it has lately been linked to a range of cardio metabolic diseases, including hypertension, metabolic syndrome, cardiovascular disease, and diabetes [4].

The question of whether it is the cause or the effect of these inflammatory illnesses has revived recently. Uric acid (UA) has been demonstrated to produce amino carbonyl radicals in vascular smooth muscle cells, which have pro-inflammatory and antioxidant actions, leading to cardiovascular disease [5]. A high amount of lipids in the blood is known as dyslipidemia (e.g., triglycerides, fat phospholipids and cholesterol).

Plasma uric acid concentrations and have been linked across several observational studies. Adverse outcomes have been associated with coronary heart disease (CHD). However, when accounting for coexisting CHD risk factors, this link did not stay significant in certain studies, maybe it just had an impact on women [6]. According to other studies, men and women's cardiac and overall mortality are strongly and independently predicted by the SUA level in those with CHD10 or arterial hypertension. Additionally, those with CHD who are at low risk are affected [7]. One of the most important risk factors for diabetes and cardiovascular disease is dyslipidemia. Atherosclerosis is accelerated in both type 1 and type 2 diabetes, and dyslipidemia causes it in diabetic people [8]. Hypertriglyceridermia, low levels of high-density lipoprotein (HDL), and high levels of low-density lipoprotein (LDL), all indicate the existence of the lipid triad in diabetes individuals [9]. Hyperuricemia has become more common in the general population as a result of modern lifestyle changes. When uric acid levels rise beyond 7 mg/dl, super saturation occurs, and uric acid crystals begin to form on the arterial wall. Women, on the other hand, have a lesser value than males [10]. Plasma IgG binds to monosodium urate. The Fc receptor on platelets recognizes the monosodium urate-IgG combination, which causes platelets to become activated. Blood coagulation is stimulated as a result of this procedure. Atherosclerosis advances due to the secretion of cytokines [11].

Increased serum uric acid levels have been linked to high levels of Insulin, known as hyperinsulinemia, a decrease in physical activity or work, a high intake of alcoholic beverages, hypertension, and an increase in the values of the body mass index, or BMI, and a drop in HDL cholesterol levels [12]. All of these characteristics are risk factors for Coronary Artery Disease (CAD) and stroke. As a result, there is debate about whether uric acid is just a co-existing marker or a causative risk factor for the pathologic processes seen in these illnesses [13]. After accounting for traditional risk variables, a number of studies suggested that hyperuricemia might be used as an independent predictor of death in individuals with coronary artery disease (CAD) [14].

A high amount of the blood's lipids is called dyslipidemia. One of the most important risk factors for diabetes and heart disease is dyslipidemia [15]. Diabetics who have dyslipidemia develop atherosclerosis, which both type 1 and type 2 diabetics experience more quickly. The lipid triad is present in diabetics as evidenced by dyslipidemia, moderate doses of elevated lipoprotein, and increased concentrations of reduced lipoprotein [16]. Despite the danger of cardiovascular disease, several studies have found that diabetic people had normal or moderately raised total cholesterol levels [17].

Uric acid and lipid profile have such a strong relationship, that by analyzing the results of uric acid levels, several lipid profile test parameters such as total cholesterol, triglycerides, high density lipoproteins (HDL), and low density lipoproteins (LDL) can also be predicted, i.e. whether they are normal, elevated, or decreased, which can aid in the treatment of dyslipidemia [18]. It is also cost-effective and time-saving, and it does not necessitate the use of any particular or sophisticated device to determine the outcomes. As a consequence, the findings may be received in a short period of time, and the related risk factors can be anticipated as soon as feasible, and treatment can begin. This actually aims to explore the start of cardiovascular illnesses by measuring only one parameter, namely serum uric acid, so that the development of symptoms of Cardio Vascular Diseases may be easily predicted.

**MATERIAL AND METHODS**

In this study 160 individuals with abnormal lipid profiles in their blood were enrolled to look into the connection between the blood lipids and uric acid in dyslipidemic patients (dyslipidemia). This is a 5-month cross-sectional study conducted in Dr Abdul Sattar Lab Sialkot using a convenient sampling method. Following that, their uric acid, cholesterol, triglycerides, LDL, and HDL cholesterol levels were measured and the obtained results were correlated with each other. In summary, we perform uric acid and lipid profile tests on under-observation samples to look into the connection among uric acid and lipid profile in dyslipidemic individuals. We enrolled dyslipidemic patients, i.e. those with abnormal lipid profile parameters, and assess total cholesterol, triglycerides, LDL and HDL cholesterol, and uric acid levels in their blood to compare the connection of lipid profile with serum uric acid in such patients.

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Technique

After an overnight fast of around 10-12 hours, 3-4 ml of blood samples were taken in Clot tubes for testing, and their serum were separated so that the appropriate tests may be done. Vacutainers were be tagged with the patient’s ID and named and placed in the correct rack so that it do not get mixed up with other samples. The Cobas c311 (Automated) chemistry analyzer was used to perform the chosen tests on the samples.

After the tests are completed, the analyzer provided the results. Following that, the data was examined and interpreted. Dyslipidemia is characterized by an increase in total cholesterol, triglycerides, and low HDL cholesterol levels. After that, the uric acid readings were compared to the lipid profile parameters’ values to see if there exists any link or not. According to prior research, hyperuricemia is more commonly seen in dyslipidemic individuals than in healthy people and a strong correlation is also evident.

RESULTS

This research looks at people between the ages of 20 and 60. The (Graph 1) shows that (15) patients are between the ages of 20 and 30, (46) patients are between the ages of 31 and 40, (74) patients are between the ages of 41 and 50, and (25) patients are between the ages of 51 and 60. Patients of both sexes are covered. The (Table 1) shows the Regression relationship of Uric Acid with Lipid Profile.

According to the current results, the individual correlation of Uric acid with all parameters of the Lipid Profile shows that a very strong positive significant correlation exists between Uric acid and Total Cholesterol, Triglycerides, and Low Density Lipoproteins i.e. with the increase in uric acid levels, Total Cholesterol, Triglycerides, and Low Density Lipoproteins also increases. On the contrary, a very strong negative significant correlation exists between Uric acid and High Density Lipoproteins i.e. HDL falls with the rise in uric acid levels and vice versa, which is indicative of resulting in more effective dyslipidemia and indicating the development of cardiovascular diseases in the desired patients in the near future. As a result, this study may be useful in reducing the incidence of related cardiovascular morbidities, and will enable to correctly predict and treat dyslipidemia and assure the correct treatment option. As the rate of CVDs rises in Pakistan, it is becoming increasingly necessary to investigate the factors that are directly linked to the disease. Following table and graphs clearly shows the correlation of lipid profile parameters with the uric acid levels. Table 1 shows the Pearson correlation value, P-value Regression equation and $R^2$ value of the under-observed variables (mentioned in 1st column).

| Variable     | Pearson correlation value | P-value | Regression Equation                                      | $R^2$  |
|--------------|---------------------------|---------|----------------------------------------------------------|--------|
| Uric acid * HDL | -.929                     | .000    | HDL=109.458-9.259*(UA)                                   | 0.862  |
| Uric acid * LDL | .933                      | .000    | LDL=19.921+20.368*(UA)                                   | 0.870  |
| Uric acid * CHOL | .928                     | .000    | CHOL=129.398+13.168*(UA)                                 | 0.348  |
| Uric acid * TG  | .834                      | .000    | TG=27.045+23.503*(UA)                                   | 0.861  |
Graph 1

Graph shows the ratio of Patients in Different Age Group.

Graph 2

Graph Shows the Ratio of Male and Female patients in Complete Sample.

Graph 3

The Figures shows the regression prediction of HDL with Uric Acid (UA).
The Figure shows the regression prediction of LDL with Uric Acid (UA).

The Figure shows the regression prediction of CHOL with Uric Acid (UA).

The Figure shows the regression prediction of TG with Uric Acid (UA).
DISCUSSION

After adjusting for sex, age, and BMI, our research found a substantial relation among blood uric acid levels and the lipid profile of dyslipidemic patients. Several epidemiological studies have shown that dyslipidemia has a role in cardiovascular risk. Furthermore, for more than 55 years, it has been demonstrated that serum uric acid is closely important to the ability of cardiovascular disease. Cardiovascular diseases (CVDs) are atherosclerosis's side effects and are linked to oxidative stress. Uric acid is a marker indicating high levels of oxidative stress, which is linked to higher xanthine oxidase activity. It's a byproduct of purine nucleotide catabolism, which is catalyzed by the liver enzyme xanthine oxidoreductase (XOR), which allows hypoxanthine to be converted to xanthine, which may then be converted to uric acid. It catalyzes the synthesis of uric acid, as well as oxygen radicals and reactive nitrogen species, which can harm nucleic acids and proteins, as well as the conversion of polyunsaturated fatty acids to lipids. As a result of this catabolic process, reactive oxygen species (ROS) are produced as by-products, which play a crucial role in enhanced oxidative stress.

Chao-Feng LinYa-Hui ChangShih-ChiehChienYueh-Hung LinHung-Yi Yeh published an Article in 2018 on Epidemiology of Dyslipidemia in the Asia Pacific Region. Dyslipidemia is a key risk factor for atherosclerosis, which leads to a variety of cardiovascular disorders. It includes high levels of total cholesterol, low-density lipoprotein cholesterol, and triglycerides, as well as low levels of high-density lipoprotein cholesterol. Based on publicly available data from websites, this article analyzes the epidemiology of dyslipidemia in nations across the Asia Pacific region, including Australia, China, Indonesia, Japan, Korea, Malaysia, New Zealand, Singapore, Thailand, and Taiwan. The nations' sources of lipid management recommendations are also summarized. Readers should consider the influence of cholesterol testing methodologies, medication usage, the year of data gathering, the age range of the examinees, and the definition of dyslipidemia in each nation before comparing the data from each country. The frequency of dyslipidemia varies significantly across the Asia Pacific area, partially due to ethnic differences, as indicated by ethnic group differences within the same nation, and partly due to a broad variety of prescription rates for lipid-lowering medicines [19].

Another investigation on the epidemiological relationship between plasma uric acid content, cholesterol (a), and the conventional lipid profile was conducted in 2010 by Lippi -G et al. cardiology in practice, The outcomes of a serum lipids, which would include cardiovascular risk, reduced lipid saturated fat (LDL-C), increased lipoprotein cholesterol (HDL-C), free fatty acids, cholesterol and triglycerides to HDL-C ratio (TC/HDL-C), the thrombotic index of plasma (AIP), and lipoprotein(a), were retrospectively evaluated in this study in a sizable cohort of unassigned adult inpatients. No admission or purposive samplings were applied while stratifying the entire outpatient population. Venous blood was frequently drawn in the morning from participants who were fasting. On a Roche Modular System P, PUA concentrations were measured using the uricase/peroxidase enzymatic method. Serum lipids and lipoproteins were also examined using the Novartis Flexible System P (Roche Diagnostics GmbH). PUA testing could be helpful in determining who needs more triage and care because of their greater risk of heart disease (CVD) [20].

Kappas, Martin; Erasmi, Stefan; Boloorani, Ali DarvishiKappas, Martin; Erasmi, Stefan; Boloorani, Ali Darvishi studied on Uric Acid and Oxidative Stress in 2005 in this study the evidence for uric acid's antioxidant activity implies that it plays a significant role as an oxidative stress marker and may have a therapeutic purpose as an antioxidant. To clarify the possible utility of uric acid (or uric acid precursors) in disorders related with oxidative stress, further well-designed clinical trials are needed. In conclusion, there is mounting evidence suggesting UA's antioxidant effect in vivo and injection of UA increases plasma antioxidant capacity. However, it is important to keep in mind that the generation of UA is linked to the conversion of XDH to XO, which results in the synthesis of free radicals. Allopurinol, an XOR inhibitor that decreases UA levels, is protective in this situation. There is growing evidence that allopurinol's protective benefits are due to additional features of the molecule, such as direct radical scavenging or mitochondrial integrity protection [21].

Amount of uric acid were discovered to be significantly higher in dyslipidemic patients in our investigation. Because dyslipidemia predicts the risk of coronary artery disease, uric acid levels should be considered in these individuals for more complete risk factor treatment. In addition to typical lipid profile measurements, we looked examined lipid fractions in patients. In the other direction, clinical suspicion of coexisting dyslipidemia should be considered when diagnosing hyperuricemia.

CONCLUSION

The Aim of this study was to see if there was a link between Uric Acid and Lipid Profile. Because dyslipidemia predicts the risk of coronary artery disease, uric acid levels should be considered in these individuals for more complete risk factor treatment. In the other direction, clinical suspicion of coexisting dyslipidemia should be considered when diagnosing hyperuricemia. Increased levels of lipid profile parameters can lead to serious heart disease, and the only way to avoid this is to get a quick diagnosis of the
disease. This also helps to predict dyslipidemia more accurately by measuring the uric acid levels of the patients.

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