Journal of Cardiovascular Disease Research

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

THE RELATIONSHIP BETWEEN A COMBINATION OF VITAMIN D DEFICIENCY AND HYPERURICEMIA WITH SEVERITY OF CORONARY ARTERY DISEASE IN STEMI PATIENTS: NOVEL APPROACH

[1]Dr. Suresh v patted,Dr. Sanjay c PorwaL , , Dr. Sameer AmbarDr. Prasad M R , Dr. Vijay metgudmath , Dr. Vishwanath. H,Dr.Suhasini. A., Department of Cardiology, J.N. Medical college, Belagavi-590010

[2] Dr. RAVIKANTH RAMADHENU [1](author)ɒoctorate of Medicine (D.M) in Cardiology dept. of cardiology, J.N Medical college Nehru Nagar, BELAGAVI –590010.

DR. RAVKANTH RAMADHENU
DOCTORATE OF MEDICINE (D.M) N CARDIOLGY
J.N MEDCAL COLLEGE
NEHRU NAGAR, BELAGAVI,

I. ABSTRACT

OBJECTIVE:To study the correlation between Vitamin-D level with deficiency and hyperuricemia with severity of coronary artery disease in stemi patients: novel approach.

Background: Coronary ARTERY sickness is one of the significant reason for death around the world. Relationship between lack of vitamin D and hyperuricemia with CAD had been less contemplated. Proof proposes there is connection between hyperuricemia, vitamin D lack, and the seriousness of CAD independently. The fundamental goal of the current review was to research the relationship be tween's lack of vitamin D and hyperuricemia with the seriousness of coronary supply route sickness in STEMI patients.

Methodology: The current cross-sectional review was led in the Department of Cardiology, KAHER's Jawaharlal Nehru clinical school, Belagavi, Karnataka, India, during the time of January 2020 - December 2020. Complete of 100 patients were remembered for the review. Venous blood tests were examined to gather biochemical boundaries like Serum Vit D levels and lipid profile. Renal capacity tests like Serum Creatinine, blood urea, and serum uric corrosive have been finished. Coronary angiography was performed to survey the recurrence and seriousness of CAD. Gathered information were examined utilizing a factual device different relapse investigation on the SPSS programming v24.0. A p-esteem underneath 0.05 was viewed as huge.

Results: The larger part (68%) had hyperuricemia or lack of vitamin D or hyperuricemia + lack of vitamin D. Most of patients had triple-vessel sickness (TVD) (46%). Hyperuricemia and serum lack of vitamin D showed huge relationship with CAD (OR= 2.1, 0.8; $p < 0.001$, 0.01).

Conclusion: In the present The review tracked down direct relationship among hyperuricemia and lack of vitamin D autonomously and blend of hyperuricemia + lack of vitamin D with seriousness of CAD. Likewise, in the review, patients with TVD had a fundamentally higher worth of hyperuricemia, vitamin D lack, and hyperuricemia + lack of vitamin D in contrast with patients with SVD or DVD. Hyperuricemia and lack of vitamin D were viewed as free and huge indicators of CAD.

Keywords ***Vitamin D deficiency, hyperuricemia, CAD, MI, STEMI.***

II. Introduction

vitamin D is launched out by a keratinocyte plasma film. The chylomicrons assimilate the vitamin D that is ingested through diet. Vitamin D enters the blood flow through the two different ways, ties to Vitamin

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

D restricting protein (DBP), and is moved tothe liver.7 In the hepatic parenchyma, vitamin D is hydroxyl lated by CYP2R1 (fundamental catalyst), CYP2D11, and CYP2D25 chemicals and structures 25-hydroxyvitamin D (25(OH)D), which is the major circling type of vitamin D and is seen as more in

serum.15 Hydroxylation of 25(OH)D by 25(OH)D-1α-hydroxylase (CYP27B1) in the proximal renal tubule frames a functioning type of vitamin D which is, 1,25-dihydroxy vitamin D (1,25(OH)2D) and is liable for significant capacities. This (1,25(OH)2D) is viewed as in just 0.1% of 25(OH)D in serum. The combination of 1,25(OH)2D is invigorated by parathyroid chemicals (PTH) and it is restrained by coursing fibroblast development factor 23 (FGF23) made by osteocytes.14,7,16 The calcitriol or 1,25-dihydroxyvitamin D (1,25(OH)2D3) restrains vascular smooth muscle cell expansion, manages the renin-angiotensin framework, diminishes coagulation, and shows mitigatingproperties.9 In the objective tissues, the Vitamin D receptor (VDR), and (1,25(OH)2D)

have a high-fondness ligand for VDR.16 VDRs are tracked down in vascular tissues of the myocardium, smooth muscle, cerebrum, heart, stomach, pancreas, enacted T and Blymphocytes, skin, and gonads.13,17 The kidney creates an idle metabolite 24,25-dihydroxy nutrient D3 (24,25(OH)2D). When calcitriol limits, hydroxylation through 25-hydroxyvitamin D3 24-hydroxylase (CYP24), in target tissues, the catalystcatabolizes 1,25(OH)2D and produces calcitroic corrosive or 24,25(OH)2D.14,7.

Nutrients are a gathering of fundamental mixtures of diet that are expected for typical wellbeing upkeep and metabolic integrity.1-3 Vitamins are of two sorts, to be specific, fat-solvent (A, D, E K) and water-dissolvable (thiamin (B1), riboflavin (B2), niacin (B3), pantothenic corrosive (B5), pyridoxine (B6), cyanocobalamin (B12), biotin, and

folate/folic acid).4,5 The fat-solvent Vitamin D exists in two structures: Ergocalciferol (D2) which is made by plants, organisms, and fish, and cholecalciferol (D3) which is blended in the skin when presented to sunlight.6,7 Cod liver oils, the tissue of fish, mackerel, trout and salmon, hamburger liver, egg yolks, cheddar, mushrooms, vitamin D sustained milk, and prepared to eat cereal, broccoli and almonds are the rich wellsprings of nutrient D.8 The suggested dietary recompenses for vitamin D shift for all time which are 400 IU/day (new-brought into the world to one year), 600 IU/the very first moment (year to 70 years age),

Vitamin D deficiency

Serum 25(OH)D is considered a marker of vitamin D levels.[18] Levels of 25OHD ranging from 50-75 nmol/liter or 20-30 ng/ml signify vitamin D insufficiency andlevels of 25OHD < 50 nmol/liter or < 20 ng/ml signify vitamin D deficiency.[19,16]Maintaining normal levels of 25OHD, which is >30 ng/ml is important.[20] Prevalence of coronary artery disease, heart failure, and peripheral artery disease will be morewhen 25OHD levels will be < 30 ng/ml.[21,22,16] In the skeletal system decrease in 25OHD levels leads to secondary hyperparathyroidism, increased osteoclast genesis,and increased bone resorption rather than bone formation.[16]

Less exposure to sunlight and less dietary intake of vitamin D results in low. levels of 25(OH)D.[23] Apart from this, confounding factors, such as obesity (caused by deposition primarily in adipose tissue), physical activity, and age result in low 25(OH)D levels.[9] The populations that are at risk for deficiency of vitamin D include infants, elders, dark-skinned individuals, overweight, pregnant

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

women, and peopleliving at high altitudes.[24,25] Vitamin D deficiency mainly causes rickets in children and osteoporosis in adults. Fortification of milk helped to reduce such diseases in the1930s.[20] The absence of this vitamin also leads to increased risk of hypertension, multiple sclerosis, cancers of the colon, prostate, breast, and ovary, Type1 diabetes,depression, pregnancy-related complications, autoimmunity-related diseases.[26,27] Table 1.1. It also results in impaired glucose tolerance, reduced insulin turnover, andless insulin resistance.[28,29] Moreover, vitamin D balances the renin-angiotensin system and thereby causes positive effects on smooth vascular muscle cells, endothelium, and cardio myocytes. Deficiency of vitamin D imbalances the renin-angiotensin system and causes hypertension and changes in cardio myocytes and the cardiovascularsystem.[30,16.]

Coronary course illness (CAD) in STEMI patients

Changes in diet and way of life cause an epidemiological progress from transmittable to non-transferable illnesses, wherein cardiovascular sickness (CVD) is one of the major threat.55,56 CVD is a gathering of problems that influences the cardiovascular framework, which comprises of the heart and veins. Different types of entanglements are cerebrovascular sickness that incorporates stroke and transient ischemic assault, fringe conduit illness, and aortic atherosclerosis.57 Coronary Artery Disease (CAD) causes the restricting of the coronary corridor through atherosclerosis. Thus, blood provided to the heart becomes inadequate.45,58 The modifiable gamble variables of CAD are hypertension, unusual blood lipids, liquor, and tobacco use, weight, actual inertia, undesirable eating regimens. The non-modifiable gamble variables of CAD are age, sex, race, and family ancestry. Controlling the modifiable gamble factors assists with disposing of CAD.59 CVD is one of the main sources of mortality and dismalness internationally, that is 32% of demise in 2019 (WHO, 2019), among that CAD represents one-half.57,60,61 Owing to CVD, "computer aided design will be the main source of death in dev eloping nations continuously 2020" and more than 4.5 million passings would happen worldwide.62,56,63,64,65. In Iran, the DALY assessment of CAD tracked down an increment of 2 overlap by 2025 (from 847,309DALYs in 2005 to 1,728,836 DALYs in 2025).[66] The death due to CAD is highest in India (22.6% in males and 3% in females) and a systematic review of 31 studies found that the prevalence of CAD is higher in urban areas than rural areas in India (Men 35–90/1000 vs. 17–45/1000; Women 28–93/1000 vs. 13–43/1000).[67]

Impaired nutrition and a decrease in vitamin D levels cause inflammation, higher coronary artery calcium scores, impaired endothelial function, and increased arterial stiffness, and eventually lead to CAD.[68,69] Studies have proved the supplementation ofvitamin D aids in preventing and managing CAD.[70,69] Vitamin D suppresses inflammation by inhibiting prostaglandin and cyclooxygenase 2 pathways, reducing matrix metalloproteinase-9 (MMP-9), and upregulating the anti-inflammatory cytokine interleukin (IL)-10. A decrease in the level of 25-OHD would cause an increase of matrix metalloproteinase-9 (MMP-9) after 72 h of ST-elevationmyocardial infarction (STEMI).[69.]

III. Problem specification

Vitamin D deficiency, hyperuricemia in CVDs like CADare some of the least investigated and major causes of diseases that lead to death worldwide.[71,20,41] The current global and Indian scenarios show an alarming rate of mortality and morbidity due to such issues. Globally, vitamin D deficiency is a public health issue as 1 billion people and more than 50% of the global population are deficient in this essentialvitamin.[72] Factors, such as decreased physical activity, less intake of food, and less exposure to the sun result in reduced bioavailability of Vitamin D among all.[73,74] Theincreased prevalence of low vitamin D in all is an important public health problem.[9] The prevalence of vitamin D deficiency is 47% among US infants, 56% among African American infants and Caucasian infants, and 90% among Iranian,

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

Turkish, and Indian infants.[20,75] In adults, the prevalence of vitamin D deficiency is 35% in theUS and 80% in Pakistan, India, and Bangladesh.[20] The prevalence of vitamin D deficiency among the elderly population is 61% in the US, 90% in Turkey, 96% in India, 72% in Pakistan, and 67% in Iran.[72,76]Hyperuricemia is increasing worldwide, and its growth was recorded by 8.5% in 2005and by 11.9% in 2009.[77,78] Such an increasing trend indicates an epidemic problem.

This is mainly because of overweight, obesity, and increased consumption of meat alcohol, and soft drinks.[79,80] The prevalence of hyperuricemia in western countries like the US is 21% followed by China with 13%,[78], in India it is 25.8% in 2018.[81] I India, the prevalence of hyperuricemia with CAD is 42.68%.[82]More than 3 million people are affected with acute ST-elevation myocardial infarction(STEMI).[83] Elderly people are at risk because of the poor prognosis.[84] According to the Global Registry of Acute Coronary Events (GRACE) and Jakarta Acute Coronary Syndrome (JAC) registry database 36% and 37% of acute coronary syndromes,respectively, are of STEMI.[45]

The association of vitamin D abnormality and hyperuricemia can substantially enhance the chances of CVDs like MI. As these diseases are creating global issues, maintaining a normal level of vitamin D and serum uric acid is important as it helps to decline the progression of such diseases. Therefore, the issue needs immediate action.

IV. Aim & Objectives:

- The objective of the present study is to investigate the correlation between vitamin D deficiency and hyperuricemia with the severity of coronary artery disease in STEMI patients.

V. REVIEW OF LITERATURE: ROL

A systematic literature review (SLR) identifies, selects and critically appraises research in order to answer a clearly formulated question **(Dewey, A. & Drahota, A. 2016).** The systematic review should follow a clearly defined protocol or plan where the criteria is clearly stated before the review is conducted. PRISMA stands for Preferred Reporting Items for Systematic Reviews and Meta-Analyses. It is an evidence-based minimum set of items for reporting in systematic reviews and meta-analyses.

Considering the cardiovascular diseases, coronary artery disease (CAD) has been found to be the prevalent reason behind the occurrence of disabilities, morbidities andmortalities across the world, even in developed nations.[86,87] In fact, CAD has been known to contribute towards millions of deaths.[88]. The critical risk factors concerned with CAD include increasing age, dark complexion, elevated levels of homocysteine, vitamin D deficiency, renal or liver disorders, overweight, smoking addiction, etc. Enhanced uric acid has been reported to be an “inde pendent risk factor” for cardiovascular and kidney-related medical ailments.[89–91]

- **Vitamin D deficiency and coronary artery diseases**

The association of vitamin D deficiency with coronary artery disease has been reported by many researchers. Such studies have been described elaborately below in a decreasing chronological order.

Recently in 2021, Siddiki et al. [88] conducted a cross-sectional observational study to understand the correlation between serum Vitamin D and its severity in terms of coronary angiography among patients suffering from acute coronary syndrome (ACS). It was observed that levels of vitamin D

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

were statistically and significantly associated with the severity of coronary atherosclerosis. A majority (64.7%) of patients with moderate to severe levels of coronary atherosclerosis displayed mild to moderate vitamin D deficiency, followed by 33.3% of them suffering from vitamin D insufficiency. The reverse was observed in case of patients with absent or mild coronary atherosclerosis, where a majority (40%) of them presented vitamin D insufficiency, followed by 35% of them falling under the sufficiency category. It was also concluded that a majority of the patients displayed low levels of vitamin D.

Raslan et al. 99concentrated on the relationship of vitamin D with persistent stable angina (CSA) as a cardiovascular gamble factor among patients from Cairo. The outcomes uncovered pervasiveness of lack in Vitamin D was seen in patients with CSA in contrast with the benchmark group. It was gotten from the calculated relapse test that hypertension alongside Vitamin D anticipated CSA fundamentally. While the likelihood of getting CSA in individuals having hypertension is multiple times more, there is a 0.7 times chance that an individual with lower vitamin D levels will get CSA in the long run. Seriousness of CSA is subject to the presence of diabetes among patients. No association was tracked down concerning orientation, home, level of active work, measure of sun openness, hypertension and level of lack of vitamin D.

Morgan et al. 101 analyzed the immediate relationship of coronary course luminal stenosis with vitamin D-inadequate ladies. The patients were encouraged to embrace elective coronary angiography. A larger part (31%) of them introduced the indications of left foremost plummeting vessel illness (LAD), trailed by 22% of them with left circumflex (LCX) coronary vessel sicknesses and 26% of them with right coronary supply route (RCA) infections. The outcomes showed that 25-hydroxyvitamin D was adversely yet essentially related with the level of luminal stenosis in any vessel.

Shor et al. [102], in their observational study of patients who were supposed to undertake coronary catheterization, studied the implications of 25-Hydroxyvitamin D as a diagnostic factor of CAD

- **Hyperuricemia and Coronary Artery Diseases**

The association of hyperuricemia with cardiovascular diseases has beenlong established by researchers over several past decades 89.

The essential **point of the concentrate by Tatli et al. 104** was to lay out the relationship of coronary vein sickness with the gamble factors, clinical attributes and serum uric corrosive among youthful grown-up patients (matured around 35 years) with intense myocardial disease. The patients who had visited the clinic for coronary angiography were picked and partitioned into Group I with basic CAD (n=36) and Group II with typical coronary supply routes (n=44).

Duran et al. [105] explained the association of serum uric acid with the severity of CAD in patients who were detected with acute coronary syndrome (ACS). The severity of CAD was also evaluated through the Gensini score. The baseline clinical characteristics significantly varied among the groups categorised according to angiographic findings in terms of uric acid, Gensini score, age and LDL.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

De Luca et al. 106 examined the relationship of hyperuricemia with CAD in a patient populace going through coronary angiography. The correlation of pattern qualities relying fair and square of uric corrosive showed contrasts in the gatherings.

Raikou et al. [90] examined the relationship of serum uric acid with CAD in patients suffering from chronic kidney diseases (CKD). A significant positive correlation was found between serum uric acid and age, BMI, waist circumference, triglycerides, systolic blood pressure (SBP), estimated pulse wave velocity (ePWV), pulse pressure (PP), phosphorus levels and intact parathormone (i-PTH), while serum uric acid was negatively correlated with 1, 25-dihydroxy vitamin D, HDL-cholesterol and estimated glomerular filtration rate (e-GFR).

- **Correlation of Vitamin D deficiency, Hyperuricemia with Coronary ArteryDiseases**

Somuncu et al.113 researched the relationship of two significant gamble factors, specifically, vitamin D and hyperuricemia, on patients with CAD. Patients undertaking coronary angiography were remembered for the review. Among the 502 review subjects structure Turkey, 44% of the patients were lacking in vitamin D (n = 221), while 11.3% of them had hyperuricemia alone (n = 57). Just 16.5% of the patients gave lack of vitamin D and more elevated levels of serum uric corrosive (n =83). No distinctions were found among the review populace concerning benchmark qualities, for example, age, orientation, hyperlipidemia, diabetes mellitus, hypertension, smoking propensities, family background of CAD, BMI, levels of creatinine, systolic and merciless circulatory strain, pulse, levels of calcium, LDH-cholesterol, HDL-cholesterol and fatty substances, other than the uric corrosive levels and 25-hydroxyvitamin D levels.

No distinctions were found among the control (n = 141), patients with hyperuricemia, vitamin D lack and a mix of both lack of vitamin D and hyperuricemia regarding the sort of myocardial localized necrosis, infarct related supply route and the therapy including percutaneous coronary mediation, clinical intercession and coronary vein sidestep unite (CABG).

Satilmis et al. [112] studied the link between vitamin D and coronary atherosclerosis among young patients from Turkey. There were hardly any differences in the clinicalfurthermore, research facility boundaries of patients with coronary atherosclerosis and controls concerning orientation, age, hypertension, LDL-cholesterol and BMI, aside from diabetes, smoking, glucose levels, complete cholesterol, HDL-cholesterol, fatty oils, vitamin D levels, uric corrosive levels and creatinine levels. The discoveries showed that vitamin D was adversely connected to calcific plaque arrangement. Besides, multivariate relapse investigation for atherosclerosis showed a likelihood of 0.69 seasons of getting atherosclerosis with low degrees of vitamin D, while the likelihood expanded to 3.67 times when the degrees of uric corrosive were high.

- **Research gaps & issues**

Therefore, there is an urgent need to conduct this study in the Indian context. The findings of this study will be helpful in understanding the treatment of CAD, prioritising the risk factors (which can then be explained to patients) and increasing awareness towards such critical cardiac issues. The present study intends to bridge the gap in research that was well realised and will be directly useful to the millions of patients suffering from CAD.

VI. RESEARCH METHODLOGY:

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

A convenient sampling method was used. Patients admitted at KAHER's Jawaharlal Nehru medical college, Belagavi. with a diagnosis of STEMI and who underwent coronary angiography, and patients fulfilling the inclusion criteria and willing to participate were included in the study. Informed consent was obtained from each patient. A detailed history and clinical features were recorded for each patient. All patients are clinically evaluated and vitals are recorded on admission Data will be analyzed and tabulated for an etiological profile of the patients.

- The collected data will be quantitatively analyzed and expressed in mean and standard deviation.
- Statistical analysis will be done by multiple regression analysis and performed using Statistical Package for the Social Sciences (SPSS) version.
- Serum vitamin D levels, Serum Uric Acid
- CBC
- Renal function tests (serum creatinine, serum urea)
- Fasting blood sugar, postprandial blood sugar, HbA1c level.
- Lipid profile: triglycerides, LDL cholesterol and HDL cholesterol, Total Cholesterol.
- Serum thyroid stimulating hormone (TSH

VII. RESULT ANALYSIS:

The current review was completed to explore the relationship between hyperuricemia alone, vitamin D lack alone and hyperuricemia + lack of vitamin D, and the seriousness of coronary course illness (CAD) in ST-rise myocardial localized necrosis (STEMI) patients. The previously mentioned boundaries have been chosen for the concentrate after an intensive survey of surviving writing.
We further compared the prevalence of hyperuricemia, vitamin D deficiency and hyperuricemia + vitamin D deficiency based on their demographics and baseline characteristics. As presented in Table 4.6, there were significant differences in the prevalence of CAD among the groups with respect to sex ($p < 0.001$) (Figure 4.14), type 2 diabetes mellitus ($p < 0.01$) (Figure 4.15), and hypertension ($p < 0.001$) (Figure 4.16).

Males presented significantly higher levels of vitamin D deficiency while females presented significantly higher levels of hyperuricemia and hyperuricemia + vitamin D deficiency ($p < 0.001$). Out of 100 participants, hyperuricemia, vitamin D deficiency, hyperuricemia + vitamin D deficiency and normal uric acid and vitamin D levels were present in 20 (28.2%), 11 (15.5%), 9 (12.7%), and 31 (43.7%) of the males while females with hyperuricemia was 12 (41.4%), vitamin D deficiency was 2 (6.9%), hyperuricemia + vitamin D deficiency was 14 (48.3%), and normal levels of uric acid and vitamin D was 1 (3.4%) ($p = 0.000$).Also, patients with type 2 diabetes mellitus and hypertension presented significantly higher hyperuricemia and hyperuricemia + vitamin D deficiency values than patients without these disorders ($p < 0.01, 0.001$). Out of the 40 diabetes mellitus patients, hyperuricemia was present in 18 (45.0%), vitamin D deficiency in 5 (12.5%) and hyperuricemia + vitamin D deficiency in 12 (30.0%) patients.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

Similarly, out of 39 hypertensive patients, hyperuricemia was present in 17 (43.6%), vitamin D deficiency in 5 (12.8%) and hyperuricemia + vitamin D deficiency in 14 (35.9%) patients (p = 0.000). However, we did not find a significant difference in CAD prevalence in participants with and without hyperuricemia, vitamin D deficiency and a hyperuricemia + vitamin D deficiency having thyroid disorder (Figure 4.17).

Correlation of coronary angiogram with serum vitamin D and hyperuricemia + vitamin D deficiency

As shown in Table 4.8, correlation between CAD and serum vitamin D, and hyperuricemia + vitamin D deficiency was evaluated among the four groups. There was an inverse correlation of coronary angiogram with serum vitamin D (r = -.382, p<0.01) and with hyperuricemia + vitamin D deficiency (r = -.366, p < 0.01).. Besides, hyperuricemia combined + vitamin D deficiency showed significant correlation with serum vitamin D (r = .655, p < 0.01).

Table 4.8: Correlation ofcoronary angiogram with serum vitamin D and hyperuricemia + vitamin D deficiency

	1	**2**	**3**
Serum vitamin D (ng/mL) (1)	1		
HU + Vit D deficiency (2)	0.655**	1	
Coronary angiogram (SVD, DVD, TVD) (3)	-0.382**	-0.366**	1

****Correlation is significant at the 0.01 level (2-tailed)**

4.8Association between coronary angiogram profile with serum uric aciddeficiency and serum vitamin D deficiency

The coronary angiogram profile was found to be significantly associated with Hyperuricemia (Chi square = 25.812, p < 0.001) (Table 4.11). Among 44 patients with mild Hyperuricemia 14 patients (31.8%) were reported to have DVD and among 55 patients with severe Hyperuricemia 37 patients (67.3%) were reported to have TVD. Thus, it is show that severe Hyperuricemia is highly related to the severity of CAD.

Table 4.9: Association between Hyperuricemia and coronary angiogram

Serum					**Chi**	

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

uric acid	SVD	DVD	TVD	Total	square	p value
Mild	21 (47.7%)	14 (31.8%)	9 (20.5%)	44		
Moderate	1 (100%)	0 (0%)	0 (0%)	1	25.812	0.001
Severe	7 (12.7%)	11 (20.0%)	37 (67.3%)	55		

Serum vitamin D deficiency was also found to have significant association with coronary angiogram profile (Chi square = 18.163, $p < 0.01$) (Table 4.12). Among 22 patients with mild vitamin D deficiency 11 patients (50%) were reported to have SVD and among 42 patients with moderate vitamin D deficiency 14 patients (33.3%) were reported to have DVD. Among 36 patients with severe vitamin D deficiency 26 patients (72.2%) were reported to have TVD.

Table 4.10: Association between serum vitamin D deficiency and coronaryangiogram

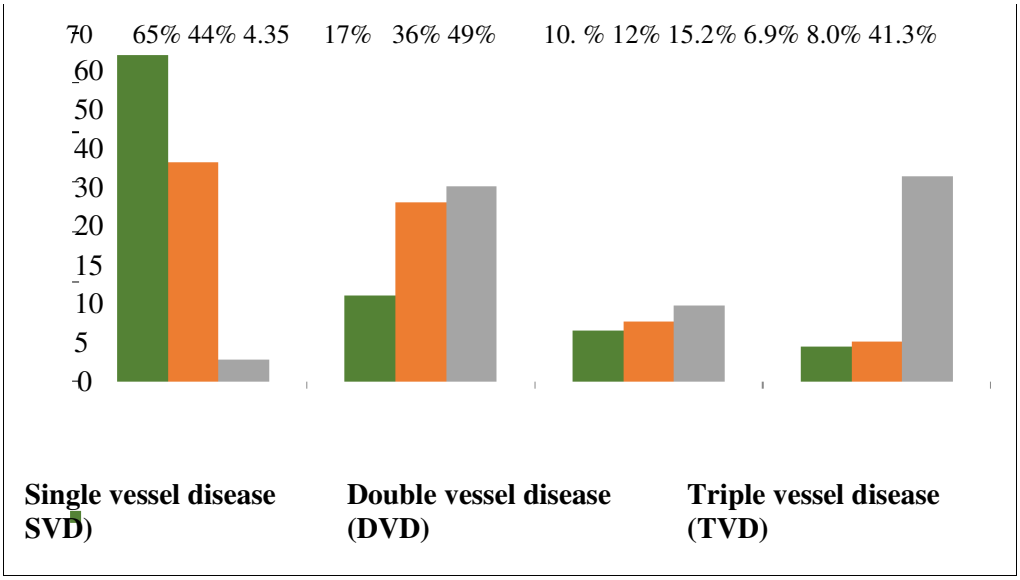
Serum vitamin D	SVD	DVD	TVD	Total	Chi square	p value
Mild	11 (50.0%)	6 (27.3%)	5 (22.7%)	22		
Moderate	13 (31.0%)	14 (33.3%)	15 (35.7%)	42	18.163	0.001
Severe	5 (13.9%)	5 (13.9%)	26 (72.2%)	36		

The current discoveries along with the outcomes examined above propose that there is a relationship between hyperuricemia alone, vitamin D lack alone, and a blend of hyperuricemia + lack of vitamin D and seriousness of CAD in STEMI patients, especially in patients with a background marked by hypertension and Dyslipidemia. Taking into account this reality, further examinations should make sense of the job of vitamin D and uric corrosive in the improvement of CAD and its complexities in patients with metabolic issues. Likewise, clinicians ought to know about this affiliation and give experiences toa dietary intervention to increase vitamin D levels. Besides, future randomized controlled trials assessing the effects of vitamin D supplements could help to understand if such intervention is effective for STEMI patients with metabolic disorders.

Among STEMI patients, hyperuricemia alone was present in the majority of the study population, followed by both hyperuricemia + vitamin D deficiency, while vitamin D deficiency was present in the lowest number of the population. This is unlike a recent study where the majority of the study population (44.0%) had vitamin D deficiency alone, followed by a combination of hyperuricemia and vitamin D deficiency (16.5%)and hyperuricemia alone (11.3%).[113] This difference may be due to the small sample size of the present study and different geographical location. Also, the mean value

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

of serum vitamin D in the entire population was 23.61 ng/ml while the mean value of serum vitamin D in the deficient population was 17.28 ng/ml.



VIII. DISCUSSION:

The present study has been undertaken to investigate the correlation of hyperuricemia and vitamin D deficiency with the severity of coronary artery disease (CAD) in ST-elevation myocardial infarction (STEMI) patients. In doing so, the study investigated the association between hyperuricemia alone, vitamin D deficiency alone, a combination of both hyperuricemia + vitamin D deficiency and the levels of CAD (SVD, double vessel disease (DVD) and triple vessel disease (TVD)) in STEMI patients, classified as per age, sex and status of hypertension, diabetes mellitus, thyroid and other metabolic risk factors.

This is unlike a recent study where the majority of the study population (44.0%) had vitamin D deficiency alone, followed by a combination of hyperuricemia and vitamin D deficiency (16.5%)and hyperuricemia alone (11.3%).[113] This difference may be due to the small sample size of the present study and different geographical location. Also, the mean value of serum vitamin D in the entire population was 23.61 ng/ml while the mean value of serum vitamin D in the deficient population was 17.28 ng/ml.

This variation in serum vitamin D levels in our study in comparison to other studies could be due to the method of measuring vitamin D concentration. Nevertheless, the values obtained from this study are consistent with the value reported from the epidemiological study assessing the vitamin D status (mean value = 18.0 ng/ml) in the Polish population despite the difference in themethod of assessment [118] Also, in the present study, serum vitamin D level was significantly lower in the patient groups (hyperuricemia, vitamin D deficiency, hyperuricemia + vitamin D deficiency) compared with the normal group (26.66 ± 4.69, 17.28 ± 1.68, 15.39 ± 2.49 vs 29.03 ± 4.15 ng /ml).

Besides, in this study, vitamin D deficiency was present in 36% of patients in the entire population, which is different from a previous finding, where vitamin D deficiency was present in the majority (80%) of the population whileonly 7% had optimal levels.[120] Moreover, this study observed that vitamin D levels decrease with age, that is, the higher the age, the more the vitamin D deficiency.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

In this study, a significant correlation between serum vitamin D and serum uric acid was found in STEMI patients. We observed that the serum uric acid levels were inversely and significantly associated with serum vitamin D levels. This finding is supported by previous studies that reported an inverse correlation between serum uricacid and serum vitamin D levels.[122]

This finding is somewhat similarto a study by Peng et al.[122] where the association between vitamin D insufficiency and hyperuricemia was significant in postmenopausal Chinese women without both diabetes and hypertension. However, the relationship between vitamin D deficiency and hyperuricemia, which as a significant metabolic risk factor for CAD, has not beenwell-documented.[122]

The main objective of our study was to assess the influence of hyperuricemia, vitamin D deficiency and hyperuricemia + vitamin D deficiency on the severity of CAD reflected by the number of diseased vessels in STEMI patients. To our knowledge, it is one among the first few studies to assess the relationship of these variables among the group of patients. In this study, the association between hyperuricemia and the severity of CAD was observed after adjusting for the potential confounders. Patients with TVD presented a significantly higher value of hyperuricemia compared to patients with DVD.

These findings suggest the clinical significance of monitoring hyperuricemia and intervention based on the diagnosis. However, whether hyperuricemia could be a potential target to prevent CVD is unknown.

the association between vitamin D deficiency and the severity of CAD (TVD) was independent and significant after adjusting for the confounders; that is, we showed that hypertensive STEMI patients with TVD presented a significantly highervitamin D deficiency in comparison to patients with DVD.

This study utilized different boundaries to decide the degree of CAD seriousness and various discoveries were acquired. TVD was more normal in patients with hyperuricemia alone, vitamin D lack alone and in patients with a mix of hyperuricemia + lack of vitamin D. At the point when the seriousness of CAD was examined, an essentially high worth was gotten for TVD and DVD patients with both Hyperuricemia and Vit D lack and TVD is more corresponded in patients with either Vit D or Hyperuricemia inadequacy. It isn't is to be expected for see problematic feelings in examinations researching the relationship between hyperuricemia or vitamin D

lack and CAD.113 The distinction in the outcomes could be because of the distinction in age of the review populaces and various areas. Albeit clashing discoveries have been gotten in regards with the impact of hyperuricemia and lack of vitamin D on the level of CAD, the current outcomes showed that the mix of hyperuricemia + lack of vitamin D altogether anticipated the seriousness of CAD in all definitions.

IX. CONCLUSON AND FUTURE SCOPE:

The current review was led to explore the connection among hyperuricemia and lack of vitamin D and hyperuricemia + lack of vitamin D, and the seriousness of coronary conduit sickness (CAD). Significant discoveries of the review are:

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

As STEMI patients with more extreme CAD have more successive unfavorable results, clinicians should accentuate on serum uric corrosive and serum vitamin D levels, and especially the mix, in all STEMI patients and consider more thorough control of metabolic gamble elements and treatment of these problems. Further examinations are expected to comprehend the unexpected impact of hyperuricemia + lack of vitamin D on the seriousness of CAD.

- STEMI patients with typical serum uric corrosive and vitamin D levels (ordinary gathering) had essentially better fasting and postprandial glucose and HbA1c values in examination with patients having hyperuricemia, vitamin D lack and hyperuricemia + lack of vitamin D.
- In our review, patients with hyperuricemia + lack of vitamin D had the fundamentally higher benefits of fasting glucose and HbA1c ($p < 0.001$).
- ·In our review patients with two gamble factors like diabetes mellitus and hypertension had fundamentally higher Uric corrosive levels and hyperuricemia + lack of vitamin D qualities than patients without these issues ($p < 0.01$, 0.001).
- ·Then again, greater part of the STEMI patients without risk factors like diabetes and hypertension had critical lack of vitamin D.
- ·There was a converse connection of coronary conduit sickness with serum vitamin D ($r = - .382$, $p < 0.01$) and with hyperuricemia + lack of vitamin D ($r =-.366$, $p < 0.01$).
- ·Our discoveries recommended that the serum uric corrosive levels were conversely and altogether corresponded to serum vitamin D levels in STEMI patients.
- Hyperuricemia and vitamin D deficiency were found to be independent and significant predictors of CAD severity among STEMI patients after adjusting for confounders highlighting that serum uric acid and vitamin D can be possible risk factors.
- Also, we found a significant synergistic effect of hyperuricemia + vitamin D deficiency on the severity of CAD, as evident by the number of diseased vessels.
- In the present study, patients with TVD had a significantly higher value of serum uric acid, vitamin D deficiency and hyperuricemia + vitamin D deficiency in comparison to patients with DVD.
- In our study we also found that the more severe vitamin D deficiency patients had more severe CAD

X. LMITATONS:

One of the significant qualities of this study is that one among the main concentrate straightforwardly researches the connection between hyperuricemia, vitamin D inadequacy and hyperuricemia + lack of vitamin D, and the seriousness of CAD.Nevertheless, there are a few constraints to this study that should be tended to. The single-focus nature of the review might bring about determination predisposition. Likewise, this study incorporated a little populace consequently restricting the force of certain examinations, and further examinations with a bigger example size are expected to affirm our discoveries and to survey measurable contrasts saw in gauge and clinical qualities among the gatherings. Besides, this

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

study included information from a gathering of patients from only one geological locale. Subsequently, the current review ought to be reached out to different locales of the country to extrapolate the discoveries into the whole Indian populace. Likewise, our review doesn't had control gathering to look at which is a significant limit.

XI. REFERENCES

1. Suzuki T. Regulation of the intestinal barrier by nutrients: The role of tight junctions. Anim Sci J. 2020;91(1):e13357.

2. Godswill AG, Somtochukwu IV, Ikechukwu AO, Kate EC. Health Benefits of Micronutrients (Vitamins and Minerals) and their Associated Deficiency Diseases: A Systematic Review. Int J Food Sci. 2020 Jan 7;3(1):1–32.

3. Bender DA. Nutritional Biochemistry of the Vitamins [Internet]. 2nd ed. Cambridge: Cambridge University Press; 2003 [cited 2022 Apr 18]. Available from: https://www.cambridge.org/core/books/nutritional-biochemistry-of-the-vitamins/10B31039A2B0F4B4DC58A89C523FAE97

4. Akram M, Munir N, Daniyal M, Egbuna C, Găman MA, Onyekere PF, et al. Vitamins and Minerals: Types, Sources and their Functions. In: Egbuna C, Dable Tupas G, editors. Functional Foods and Nutraceuticals: Bioactive Components, Formulations and Innovations [Internet]. Cham: Springer International Publishing; 2020 [cited 2022 Apr 18]. p. 149–72. Available from: https://doi.org/10.1007/978-3-030-42319-3_9

5. Ball GFM. Vitamins: Their Role in the Human Body. John Wiley & Sons; 2008. 451 p.

6. Freeman J, Wilson K, Spears R, Shalhoub V, Sibley P. Performance evaluation of four 25-hydroxyvitamin D assays to measure 25-hydroxyvitamin D2. Clin Biochem. 2015;48(16–17):1097–104.

7. Hossein-nezhad A, Holick MF. Vitamin D for Health: A Global Perspective. Mayo Clin Proc Mayo Clin. 2013 Jul;88(7):720–55.

8. National Institutes of Health | USAGov [Internet]. [cited 2022 Jan 27]. Available from: https://www.usa.gov/federal-agencies/national-institutes-of-health

9. Danik JS, Manson JE. Vitamin D and Cardiovascular Disease. Curr Treat Options Cardiovasc Med. 2012 Aug;14(4):414–24.

10. Jorde R, Grimnes G. Vitamin D: no cure for depression. Am J Clin Nutr. 2019;110(5):1043–4.

11. Abrams GD, Feldman D, Safran MR. Effects of Vitamin D on Skeletal Muscle and Athletic Performance. J Am Acad Orthop Surg. 2018 Apr 15;26(8):278– 85.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

12. Fleet JC. The role of vitamin D in the endocrinology controlling calcium homeostasis. Mol Cell Endocrinol. 2017 Sep 15;453:36–45.

13. Holick MF. Vitamin D: A millenium perspective. J Cell Biochem. 2003;88(2):296–307.

14. Christakos S, Ajibade DV, Dhawan P, Fechner AJ, Mady LJ. Vitamin D: Metabolism. Rheum Dis Clin N Am. 2012 Feb;38(1):1–1 1.

15. Arrangoiz R, Cordera F, Caba D, Juárez MM, Moreno E, Luque E. Parathyroid embryology, anatomy, and pathophysiology of primary hyperparathyroidism. Int J Otolaryngol Head Neck Surg. 2017;6(4):39–58.

16. Adams JS, Hewison M. Update in Vitamin D. J Clin Endocrinol Metab. 2010 Feb 1;95(2):471–8.

17. Barbarawi M, Kheiri B, Zayed Y, Barbarawi O, Dhillon H, Swaid B, et al. Vitamin D Supplementation and Cardiovascular Disease Risks in More Than 83 000 Individuals in 21 Randomized Clinical Trials: A Meta-analysis. JAMA Cardiol. 2019 Aug 1;4(8):765–76.

18. Jukic AMZ, Baird DD, Weinberg CR, Wilcox AJ, McConnaughey DR, Steiner AZ. Pre-conception 25-hydroxyvitamin D (25(OH)D) and fecundability. Hum Reprod. 2019 Nov 1;34(11):2163–7 2.

19. Mazahery H, Von Hurst PR. Factors Affecting 25-Hydroxyvitamin D Concentration in Response to Vitamin D Supplementation. Nutrients. 2015 Jul;7(7):5111–42.

20. Sizar O, Khare S, Goyal A, Givler A. Vitamin D Deficiency. In: StatPearls

[Internet]. Treasure Island (FL): StatPearls Publishing; 2022 [cited 2022 Apr

18]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK532266/

21. Rahman A, Karmakar P, Jabeen S, Nabi S, Khan A, Shahriar MS, et al. Association of Vitamin D Level with Severity of Angiographically Documented Coronary Artery Disease: Observations from Bangladeshi Patients. J Cardiovasc Dis Res. 2019 Jul 8;10:52–7.

22. Wu Z, Wang T, Zhu S, Li L. Effects of vitamin D supplementation as an adjuvant therapy in coronary artery disease patients. Scand Cardiovasc J. 2016 Jan 2;50(1):9–16.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

23. Baggerly CA, Cuomo RE, French CB, Garland CF, Gorham ED, Grant WB, et al. Sunlight and vitamin D: necessary for public health. J Am Coll Nutr. 2015;34(4):359–65.

24. Martin CA, Gowda U, Renzaho AMN. The prevalence of vitamin D deficiency among dark-skinned populations according to their stage of migration and region of birth: A meta-analysis. Nutrition. 2016 Jan 1;32(1):21–32.

25. Targeted 25-hydroxyvitamin D concentration measurements and vitamin D3 supplementation can have important patient and public health benefits | European Journal of Clinical Nutrition [Internet]. [cited 2022 Apr 18]. Available from: https://www.nature.com/articles/s41430-020-0564-0

26. Wimalawansa SJ. Non-musculoskeletal benefits of vitamin D. J Steroid Biochem Mol Biol. 2018 Jan 1;175:60–81.

27. Podd D. Hypovitaminosis D: A common deficiency with pervasive consequences. J Am Acad PAs. 2015;28(2):20–6.

28. Pramono A, Jocken JWE, Blaak EE. Vitamin D deficiency in the aetiology of obesity-related insulin resistance. Diabetes Metab Res Rev. 2019;35(5):e3146.

29. Mirhosseini N, Vatanparast H, Mazidi M, Kimball SM. Vitamin D Supplementation, Glycemic Control, and Insulin Resistance in Prediabetics: A Meta-Analysis. J Endocr Soc. 2018 Jul 1;2(7):687–70 9.

31. Yu W, Cheng JD. Uric acid and cardiovascular disease: an update from molecular mechanism to clinical perspective. Front Pharmacol. 2020;11.

32. Kumar DS, Mahajan DP, Badyal DA. Serum uric acid levels and its association with type 2 diabetes mellitus. World J Pharm Sci. 2019 Mar 30;38–42.

33. Desideri G, Castaldo G, Lombardi A, Mussap M, Testa A, Pontremoli R, et al. Is it time to revise the normal range of serum uric acid levels? Eur Rev Med Pharmacol Sci. 2014;18(9):1295–306.

34. George C, Minter DA. Hyperuricemia. In: StatPearls [Internet]. Treasure

Island (FL): StatPearls Publishing; 2022 [cited 2022 Apr 18]. Available from: http://www.ncbi.nlm.nih.gov/books/NBK459218/

35. Vargas-Santos AB, Neogi T. Management of Gout and Hyperuricemia in CKD. Am J Kidney Dis. 2017 Sep 1;70(3):422–39.

36. Liang J, Jiang Y, Huang Y, Song W, Li X, Huang Y, et al. The comparison of dyslipidemia and serum uric acid in patients with gout and asymptomatic hyperuricemia: a cross-sectional study. Lipids Health Dis. 2020 Mar 3;19(1):31.

ISSN: 0975-3583,0976-2833 VOL13,ISSUE05,2022

37. Choi HY, Kim S hyung, Choi AR, Kim SG, Kim H, Lee JE, et al. Hyperuricemia and risk of increased arterial stiffness in healthy women based on health screening in Korean population. PLOS ONE. 2017 Jun 30;12(6):e0180406.

38. Wu AH, Gladden JD, Ahmed M, Ahmed A, Filippatos G. Relation of serum uric acid to cardiovascular disease. Int J Cardiol. 2016 Jun 15;213:4–7.

39. Luo Q, Xia X, Li B, Lin Z, Yu X, Huang F. Serum uric acid and cardiovascular mortality in chronic kidney disease: a meta-analysis. BMC Nephrol. 2019 Dec;20(1):1–12.

40. Zoccali C, Mallamaci F. Uric acid in chronic kidney disease: the quest for causality continues. Nephrol Dial Transplant. 2018 Feb 1;33(2):193–5.

****
****