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Correlation study between serum bilirubin level and Risk factor of cardiac disease in patients admitted in hospital

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ABSTRACT:

Background: Bilirubin is a metabolic byproduct of the breakdown of hemoglobin degradation which itself must be metabolized for appropriate excretion. High levels of bilirubin are associated with decreased risk of coronary heart disease (CHD) and cardiovascular disease (CVD).

Objectives: To study the level is to see correlation between serum bilirubin level and Risk factor of cardiac disease in patients admitted in hospital.

Methodology: This study includes 100 Male Indian subjects between 35 to 60 years of age who visited medicine OPD of our institute. Biochemical test like Lipid profile, Serum bilirubin and blood sugar(Fasting & Post Prandial) were measured on a fully automated analyzer along with quality control sera. Obtained Results were analyzed statistically to calculate p value and to see the difference of significance.

Results: HDL level between OPD and IPD (31.92 ± 4.25 mg/dl and 49.92 ± 7.23 mg/dl) subjects respectively. In the same manner this table also showed a higher level of FBS 122.23±4.2 mg/dl and PP2BS 132.23±8.0 mg/dl in OPD subjects as compared to IPD patients but the difference among them was not significant. It was represented that the OPD subjects were more prone to risk of CVD because the level of S.choletserol was 220.92 ± 40.21 as compared to 209.45 ± 29.90 in IPD subjects.(p value:<0.001 significant)Regarding serum Triglyceride level , there is no such significant difference found among two group. While comparing the level of serum LDL it was 164.64±15.29 and 134.83±10.39 in Control and case Group Respectively.(p <0.001). The Level of Total bilirubin was 0.84±0.41 mg/dl in control group and 4.42±3.1 in case Group and difference among them was highly significant.

Conclusions: From my study it will be conclude that there is a significant negative relationship was demonstrated between baseline bilirubin levels and incident CHD and CVD death and the level of serum bilirubin is important parameter for defining risk of cardiovascular disease.

Key words: Blilirubin, HDL, Cardiac disease

INTRODUCTION:

Bilirubin is viewed as a solid lessening specialist and a potential physiological cancer prevention agent [1]. There's new expectation for the battle against malignant growth and cardiovascular infection, following advancement research recognizing a color in our bile.

The cancer prevention agent limit of bilirubin and its intense capacity to search peroxyl extremists have prompted the idea that somewhat expanded circulatory bilirubin might have a physiologic capacity to safeguard against sickness processes that include oxygen and peroxyl radicals. Indeed, reverse relationships between's the presence of CAD and absolute bilirubin focuses in the course were accounted for as of late in a few autonomous investigations. [2,3]
Moreover, plasma bilirubin relates contrarily with a few laid out risk factors for CAD, including smoking, expanded LDL-cholesterol, diabetes, and corpulence, yet is straightforwardly corresponding to the defensive component HDL-cholesterol. [4,5] The impact of bilirubin on the gamble of cardiovascular illness is obvious in men however is less clear in ladies.
The defensive impact of bilirubin connects with the cell reinforcement property of bilirubin, which forestalls lipid oxidation, particularly Low-thickness lipoprotein (LDL), and restrains free revolutionary prompted harms. Lower serum bilirubin level has been shown to be related with endothelium and microvascular malfunction. [6,7]

MATERIALS AND METHODS:

This Cross sectional study was conducted at the Parul sevashram hospital <vadodara,Gujarat from Jan 2020-Jan 2021 on 100 male [50 healthy OPD and 50 IPD admitted in the hospital for clinically different complaints instead of CVD/CHD] subjects of 35-60 yr age Group which were tested for Blood sugar(FBS & PPBS), lipid profile, and serum bilirubin, were considered for our study.

Patients with symptoms of congestive cardiac failure, chronic kidney disease, chronic liver disease, autoimmune diseases, COPD and malignancy were excluded from the study. Controls were selected matched with age, gender and other co-morbid conditions.
Female were excluded from our study.

Collection of blood sample: 5 ml venous blood was collected from all participants in a fasting condition. From them blood was distributed in Flouride and plain vaccutainer for estimation of various biochemical parameter.
Blood samples were centrifugated at 3000 RPM for a period of 10 minutes after giving uniq ID to all participants and same ID was mentioned on eppendorf cup to hide identity of participants.

Blood glucose was estimated by Hexokinase method from fluoride sample in fully automated biochemistry analyser. Cholesterol was measured by CHOD-PAP method, TG was estimated by GPO-PAP method ,HDL was estimated by Phospho tung state precipitation method and bilirubin was measured by diazotized sulfanilic acid method in biochemistry analyser with using Randox quality control material.

Serum LDL and VLDL was calculated by frieldwalds formula.

All obtained data were analysed stastatically by calculating p-value by using online student t-test calculator.

RESULTS:

In our study Table representing the biochemical and lipid parameters level of both Group.(Table 1)

Table 1 showed a great difference in HDL level between OPD and IPD (31.92 ± 4.25, 49.92 ± 7.23) subjects respectively. In the same manner this table also showed a higher level of FBS 122.23±4.2 mg/dl and PP2BS 132.23±8.0 mg/dl in OPD subjects as compared to IPD patients but the difference among them was not significant.

It was represented that the OPD subjects were more prone to risk of CVD because the level of S.choletserol was 220.92 ± 40.21 as compared to 209.45 ± 29.90 in IPD subjects.(p value:<0.001 significant)(Table 1)

Regarding serum Triglyceride level , there is no such significant difference found among two group.

While comparing the level of serum LDL it was 164.64±15.29 and 134.83±10.39 in Control and case Group Respectively. The difference among them was highly significant and LDL having atherogenesis property so it leads to increase risk of cardiovascular disease.(Table 1)

Table 1: Comparison of Lipid profile and Blood sugar level between case and Control grou

parameter	Subject Group		P-value
	OPD(50)(Control)	IPD(50)(case)	
S.Cholesterol(mg/dl)	220.92 ± 40.21	209.45 ± 29.90	<0.001(S)
S.Triglyceride(mg/dl)	121.83 ± 25.90	123.52 ± 31.23	0.7475(NS)
S.HDL(mg/dl)	31.92±4.25	49.92±7.23	<0.001(S)
S.LDL(mg/dl)	164.64±15.29	134.83±10.39	<0.001(S)
S.VLDL(mg/dl)	24.36±10.19	24.70±12.29	0.7350
FBS(mg/dl)	122.23±4.2	118.12±4.5	0.0001(S)
PP2BS(mg/dl)	132.23±8.0	122.23±5.2	0.0001(S)

The level of Direct bilirubin was 0.33±0.21 mg/dl in control group and 3.41±1.72 mg/dl in case group. and difference among them was highly significant. (Table 2)

The Level of Total bilirubin was 0.84±0.41 mg/dl in control group and 4.42±3.1 in case Group and difference among them was highly significant. (Table 2)

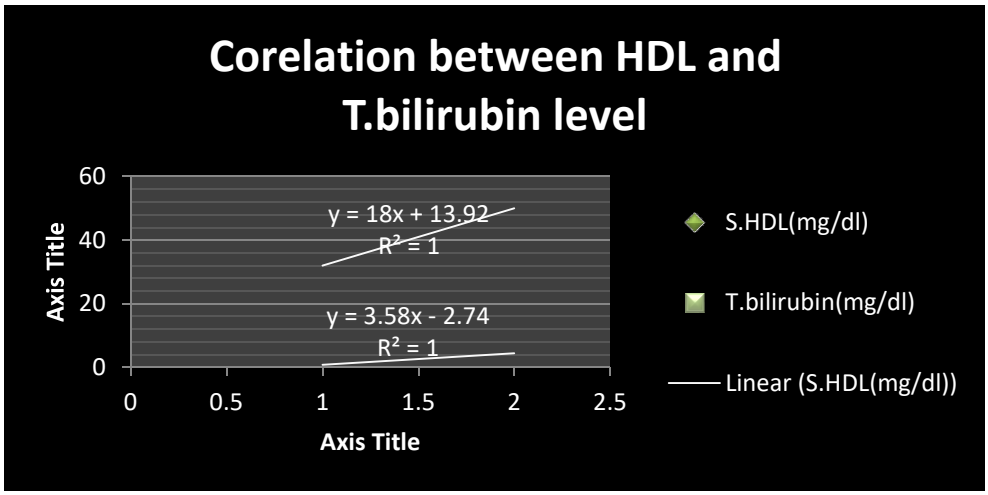
Table 2: Showing the Bilirubin Level in case and control group

(P <0.001 significant)

parameter	Subject Group		P-value
	OPD(50)(Control)	IPD(50)(case)	
D.bilirubin(mg/dl)	0.33±0.21	3.41±1.72	0.0001(S)
T.bilirubin(mg/dl)	0.84±0.41	4.42±3.1	0.0001(S)

Graph 1: Showing correlation between bilirubin level and HDL cholesterol level.

Graph 1: Showing correlation between HDL and T .Bilirubin Level



DISCUSSION:

The present longitudinal review shows a critical negative connection between baseline bilirubin levels inside the physiologic reach and incident CHD/CVD death, consequently recommending that most elevated tertile of serum bilirubin might assume a defensive part against episode CHD and CVD death. Furthermore, serum bilirubin is adversely connected with MetS and MetS parts. [8]These discoveries propose that height of bilirubin levels within the ordinary reach are a good condition from a metabolic viewpoint

Oxidative pressure assumes a significant part in atherosclerosis, which is a chronic inflammatory reaction to vascular endothelial injury brought about by an assortment of elements advancing incendiary cell section and initiation [9,10]. Therecognition of bilirubin as a significant endogenous mitigating and cell reinforcement atom has expanded in late many years. Bilirubin affects atherosclerosis by a few restraining systems, including low-thickness lipoprotein oxidation, vascular smooth muscle cell multiplication, and endothelial brokenness [11]. Somewhat raised circling bilirubin levels appears to address a promising objective for counteraction and decrease of the prevalence of CVD and other oxidative-stress issues, including type 2 diabetes mellitus (T2DM) and disease [12]. As needs be, the job of bilirubinas a natural indicator in the gamble evaluation of persistent issues, with expanding overall predominance, is of impressive clinical economic importance. Without a doubt, CVD address a primary driver of mortality and weight of infection.

Atherosclerosis is viewed as the most well-known fundamental reason for the coronary vein illness (CAD), which is the significant reason for mortality overall both in created and agricultural nations. [13]Whereas then again cell reinforcements are the overwhelming versatile

reactions by the blood vessel vasculature in light of the oxidative pressure accordingly forestalling the atherosclerosis.3 Bilirubin, being a harmful material item shaped during heme catabolism is as a matter of fact a strong physiological cancer prevention agent that gives significant insurance against atherosclerosis and aggravation. The results of the catabolic response, for example bilirubin, carbon monoxide and iron play a defensive part. The other significant job of bilirubin, the regular cell reinforcements are the hindrance of vascular cell attachment atom VCAM-1 forestalling the multiplication of the smooth muscle cells and the transendothelial relocation of the leucocytes.

More modest examinations on bilirubin and CVD occasions have revealed extents of affiliation like those found in the current review. A meta-investigation of 11 examinations led in men and distributed up to 2001 announced a 6.5% decline in atherosclerotic infection rates per 1-µmol/L expansion in bilirubin level. 9The Framingham Offspring Cohort Study (n=1780) detailed a measurably huge 10% decrease in CVD occasions per 1.7-µmol/L (0.1-mg/dL) expansion in bilirubin, a 13% decrease in CHD, and a 13% decrease in MI more than a 24-year follow-up after change for traditional gamble factors10. These discoveries are comparative in greatness to our consequences of a ≈3% to 5% abatement per 1-µmol/L increment at bilirubin levels <10 to 15µmol/L. The marginally more fragile relationship in THIN information might be because of changes for extra gamble factors, for example, social hardship or conceivably the presence of some opposite causation in which bilirubin was estimated nearer to the occasion date than in different accomplices, and any feeling of heme oxygenase in light of progressing yet undiscovered sickness might weaken the noticed affiliations [14].

The absence of absolute bilirubin fractionation (backhanded and direct) has confined the capacity to assess whether immediate or aberrant hyperbilirubinemia isassociated with CVD risk. Be that as it may, there is a solid connection between's all out bilirubin and unconjugated bilirubin, as well as between all out bilirubinand formed direct bilirubin in sound subjects [15]. We don't have definite information on pathologic jaundice (for example hepatitis, liver cirrhosis, spices oralternative prescriptions, and so forth) Consequently, we attempted to wipe out these possibilities by barring those with serum TB levels of ≥ 2.0. By and by, thisstudy tentatively showed the impacts of bilirubin levels on CHD risk and CVD mortality in an enormous local area based accomplice, which has beenpreviously led in just an exceptionally predetermined number of studies. Low bilirubin levels can be demonstrative of diminished heme oxygenase action (apowerful cancer prevention agent) or could be characteristic of high oxidative pressure in patients prompting utilization of the regular cell reinforcements including bilirubin.Hence, there is plausibility that lower levels of bilirubin are maybe not the causal variable for CVD but rather may show patients at an expanded gamble ofd eveloping CVD [16]. Also, further enormous scope long haul study will be expected to affirm our outcomes in regards to irritation and IR in the future.

CONCLUSION

From my review it will be infer that there is a huge negative relationship was showed between benchmark bilirubin levels and occurrence CHD & CVD and the degree of serum bilirubin is significant boundary for characterizing chance of cardiovascular sickness.

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