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## Role of serum bilirubin as an antioxidant and lipid profile in acute myocardial infarction

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### ABSTRACT

The present study was carried out to evaluate lipid profile and bilirubin as a risk factor associated in AMI patients admitted to ICCU.

Changes in serum lipid profile and serum bilirubin levels after AMI were analyzed in patients within 24 hours of admittance in ICCU. Blood samples were collected in patients with AMI (n=20), within 24 hours after the infarct and compared with age, sex matched normal healthy controls (n=20).

Highly significant increase in Non HDL-C and bilirubin level was observed. Significant increase was observed in total cholesterol and LDL-C. All lipid ratios were highly significantly increased in AMI. Serum bilirubin level was significantly lower in AMI smokers as compared to AMI non smokers

Endogenous antioxidant serum bilirubin was elevated in AMI in 24 hours which may suggest that there is excess formation of free radicals. Smoking status may also affect serum bilirubin levels. Dyslipidemia was observed in AMI patients which may suggest that these patients may be prone to AMI due to increased free radical generation. Serum LDL-C levels are the main target for lipid management in the majority of guidelines. In our study non-HDL-C is highly significantly increased than LDL-C. The NCEP-ATP III recently recommended that non-HDL-C be used as a secondary target of therapy. Our study also recommends the same in patients with AMI as it is less expensive and simpler to use.

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### INTRODUCTION

Coronary artery disease (CAD) is associated with multiple factors including hereditary, hyperlipidemia, obesity, hypertension, environmental factors and life style variables such as stress, smoking and alcohol consumption. Lipids have been investigated extensively in recent years<sup>[1]</sup>. Asians show a mixed picture of dyslipidemia. Dyslipidemic patients are more prone to myocardial infarction due to increased free radical generation and ischemia<sup>[2]</sup>.

Lipid oxidation and formation of oxygen radicals are important elements of arterial plaque formation and atherosclerosis and involved in pathophysiology of coro-

nary artery disease. The endogenous antioxidant system includes albumin, uric acid, and total bilirubin. Imbalance of this system, either due to excess free radical formation or insufficient removal by antioxidants, leads to oxidative stress. As bilirubin has antioxidant properties, it has been suggested that it may have a protective role in atherosclerotic process.

The antioxidant capacity of bilirubin and its ability to provide potent scavenging of peroxyl radicals have led to suggestion that mildly increased circulatory bilirubin may have a physiologic function to protect against disease processes that involve oxygen and peroxyl radicals<sup>[3]</sup>.

The present study was carried out to evaluate lipid profile and bilirubin as a risk factor associated in AMI

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patients admitted to ICCU of GMCH, Aurangabad.

### MATERIALS AND METHODS

In this prospective case-control study changes in serum lipid profile and serum bilirubin levels after AMI were analysed in 20 patients within 24-hours of admittance in ICCU. The diagnosis of AMI was established according to diagnostic criteria: chest pain lasting for d'3 hours & ECG changes. The patient group included 9 smokers, and 11 hypertensives. Age of the patients ranged from 45-75 years. The control group consisted of 20 age, sex matched healthy volunteers. Inclusion criteria were patients with diagnosis of AMI. Patients with diabetes mellitus, renal insufficiency, hepatic disease or taking lipid lowering drugs or antioxidant vitamin supplements were excluded from the study. Normolipidemic status was judged by the following criteria: LDL-C d'160 mg/dl, HDL-C e'35 mg/dl, total cholesterol (TC) <200mg/dl, triglyceride (TG) level <150 mg/dl National Cholesterol Education Program, The Adult Treatment Panel III (NCEP,ATP-III,2001)<sup>[4]</sup>. Blood samples were collected after overnight fasting, serum was separated and used for estimation of lipid profile and bilirubin.

Lipid profile was done enzymatically using an Enzopak kit LDL-C was determined from the values of the TC and HDL-C using the following formula:

$$\text{LDL-C} = \frac{\text{TC}(\text{TG} - \text{HDL-C})}{5} \text{ and non HDL-C} = (\text{TC} - \text{HDL-C})$$

Cholesterol/ (HDL-C + bilirubin), (LDL-C)/ (HDL-C + bilirubin), LDL-C/HDL-C & TC/HDL-C ratios were also tested.

Serum total bilirubin was determined by Transasia kit. Statistical significance was analysed by students 't' test.

### RESULT

**TABLE 1 : Anthropometric data regarding controls and patients**

Variables	Controls (n=20)	AMI patients (n=20)	p-value
Age	58.90±10.63	60.65±10.03	0.30
Systolic blood pressure (mmHg)	100±4.08	156.0±15.22	<0.001
Diastolic blood pressure (mmHg)	75.91±5.66	94.38±38.91	<0.001
BMI (Kg/m <sup>2</sup> )	24.80±3.87	32.57±5.07	<0.001

**TABLE 2 : Lipid profile & serum bilirubin levels in AMI patients and controls**

Variables	Controls (n=20)	AMI patients (n=20)	p-value
TC mg/dl	138.55±19.74	161.6±51.57	0.036
TG mg/dl	121.95±35.41	133.15±70.75	0.266
HDL-C mg/dl	52.95±8.80	39.75±11.98	0.0001
VLDL mg/dl	24.25±7.12	26.64±14.14	0.252
LDL-C mg/dl	75.4±17.68	95.17±42.44	0.032
Non HDL-C mg/dl	85.60±19.73	120.35±55.36	0.007
Bilirubin mg/dl	0.64±0.25	1.09±0.59	0.002

**TABLE 3 : Distribution of lipid ratios in AMI patients and controls**

Variables	Controls (n=20)	AMI patients (n=20)	p-value
IDL-C/HDL-C	1.50±0.47	2.70±1.82	0.004
IDL-C/HDL-C+Bilirubin	1.43±0.41	2.59±1.66	0.003
TC/HDL-C	2.68±0.60	4.47±2.35	0.001
TC/HDL-C+Bilirubin	2.647±0.58	4.30±2.12	0.001

**TABLE 4 : Lipid profile & serum bilirubin levels in smokers with AMI and non-smokers with AMI**

Variables	Non-Smokers with AMI (n=9)	Smokers with AMI (n=11)	p-value
TC mg/dl	158 ± 49.03	170.90±61.41	0.30
TG mg/dl	114 ± 36.5	157±85.69	0.07
HDL-C mg/dl	40.89 ± 8.33	38.64±14.50	0.33
VLDL mg/dl	22.9 ± 7.3	31.42±17.12	0.07
IDL-C mg/dl	94.31± 44.38	100.78±47.81	0.38
Non-HDL-C mg/dl	117.11±53.14	129.54±55	0.32
Bilirubin mg/dl	1.2±0.78	0.74±0.40	0.01

### DISCUSSION

CAD is a major cause of morbidity and mortality in developed and developing countries. Dyslipidemia is a major modifiable risk factor for CAD<sup>[2]</sup>. In the present study, dyslipidemia was observed in AMI patients. This finding is in agreement of several studies (2, 5, 6).

LDL-C is widely considered to be the major atherogenic lipoprotein & the primary target of lipid lowering therapy. NonHDL-C, the sum of apolipoprotein-B containing lipoproteins i.e. LDL, VLDL, IDL, & lipoprotein (a) which measure as atherogenic cholesterol & has been identified as a secondary target of lipid lowering therapy by NCEP, ATP-III,2001(4). In this study nonHDL-C was highly significantly increased (p<0.001) as compared to LDL-C (p<0.05). Therefore, our find-

ings support the recommendation of NCEP-ATP-III to use nonHDL-C as a secondary target of therapy among AMI patients.

The systolic and diastolic BP of AMI cases were high at the time of study. The BMI was 32.7 Kg/m<sup>2</sup> = 3.87 in AMI and 24.80 Kg/m<sup>2</sup> = 3.57 in control group. BMI greater than 25Kg/m<sup>2</sup> is considered as the cut off for risk of health problems<sup>[7]</sup>.

The present study reveals the importance of assessing lipid ratios, even in normolipidemic subjects, since it is an atherogenic factor in development of AMI. The traditional ratios when combined with bilirubin were almost identical<sup>[8]</sup>.

Development of AMI involves lipid oxidation and formation of oxy radicals and that atherosclerosis and inflammation are associated with formation of oxy and peroxy radicals. Endogenous antioxidant serum bilirubin was in the upper portion of the reference interval in AMI as compared to control. The antioxidant capacity of bilirubin and its ability to provide potent scavenging of peroxy radicals have led to suggestion that mildly increased circulatory bilirubin may have physiologic functions to protect against disease processes that involve oxy and peroxy radicals<sup>[3]</sup>.

It has been reported that serum bilirubin correlates inversely with several established risk factors for AMI, including smoking, increased LDL-C, obesity and directly proportional to the protective factor HDL-C<sup>[9,10]</sup>. In the present study serum bilirubin level decreased in smokers with AMI as compared to non smokers AMI patients. Thus this finding supports those of other studies that show smoking affects serum bilirubin level.

## CONCLUSION

Endogenous antioxidant serum bilirubin was elevated in AMI in 24 hours which may suggest that there is excess formation of free radicals. Smoking status may also affect serum bilirubin levels. Dyslipidemia was observed in AMI patients which may suggest that these patients may be prone to AMI due to increased free radical generation. Serum LDL-C levels are the main target for lipid management in the majority of guidelines. In our study non-HDL-C is highly significantly increased than LDL-C. The NCEP-ATP III recently recommended that non-HDL-C be used as a second-

ary target of therapy. Our study also recommends the same in patients with AMI as it is less expensive and simpler to use.

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