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ESTIMATION OF APOLIPOPROTEIN(B) IN EARLY ONSET ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT

Background: Cases of early onset ST segment elevation myocardial infarction (STEMI) is increasing worldwide including our region. In recent years the scientific community paid attention to non-classical risk factors like apolipoproteins. We aimed to assess the relation of apolipoprotein B (apoB) with early onset STEMI in Duhok, Kurdistan region of Iraq. **Methods:** This case-control study was performed at the Azadi Teaching Hospital and Azadi heart center in Duhok, Iraq, between May 2021 and December 2021. The study included 41 young patients (<50 Years) presented with acute STEMI and 48 healthy individuals with the matched age and sex and without a history of coronary artery disease (CAD) or ischemic stroke. Demographic, clinical and cardiovascular risk factors of the sample were studied. The apoB was measured and its relation to STEMI was assessed. **Results:** Cases had clustered of conventional risk factors and had significantly higher rates of these risk factors. Smoking was evidently more prevalent amid other risk factors. The apoB levels were higher among cases, but did not achieved a significant association with early onset STEMI (P>0.05). **Conclusions:** Despite of lacking direct association of apoB with early onset STEMI in this pilot study, the apoB has to be corelated with other CVS risk factors in the risk stratification for STEMI. Further cohort studies are warranted to assess the effect of apoB in the occurrence of STEMI with and without conventional CVS risk factors and to evaluate the sensitivity of this indicator in predicting ischemia.

KEYWORDS: Apolipoprotein B, ST-segment elevation myocardial infarction, Dyslipidemia, Cardiovascular risk factors.

INTRODUCTION

Identifying individuals at high risk for ST-segment elevation myocardial infarction (STEMI) remains a critical aspect of cardiovascular prevention and management. Among the key modifiable risk factors, dyslipidemia has a well-established role in the pathogenesis and progression of coronary artery disease (CAD), ultimately influencing clinical outcomes in patients with STEMI.[1,2] Traditional assessment of cardiovascular disease risk includes measuring serum levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG). While statin therapy effectively lowers LDL-C and reduces cardiovascular events, a substantial residual cardiovascular risk—estimated at around 70%—persists even after achieving lipid targets.[3,4] Recent evidence suggests that focusing solely on lipid concentrations may not fully capture the atherogenic potential of circulating lipoproteins. Instead, evaluating the structural and functional protein components, such as apolipoproteins, may enhance risk stratification. Among them, apolipoprotein B (apoB) has emerged as a superior marker of atherogenic particle burden. Each atherogenic lipoprotein particle (VLDL, IDL, and LDL) contains one molecule of apoB, making serum apoB concentration a direct indicator of the number of circulating atherogenic particles.[5,6] This property makes apoB especially valuable in patients with elevated TG levels, where LDL-C may underestimate cardiovascular risk. As such, many experts now advocate measuring non-HDL-C or apoB levels instead of relying solely on LDL-C for a more comprehensive risk assessment.[7] Dyslipidemia itself is characterized by increased serum TC (>200 mg/dL), LDL-C (>100 mg/dL), TG (>150 mg/dL), and decreased HDL-C (<40 mg/dL), creating a pro-atherogenic environment

conducive to plaque formation and rupture.[8] ApoB-100, synthesized in the liver, is the predominant isoform in plasma and reflects the lipoproteins most responsible for atherosclerosis.[9] Myocardial infarction (MI), especially STEMI, results from a complete and sustained occlusion of a coronary artery, most often due to atherosclerotic plaque rupture and subsequent thrombus formation.[10-11] It is a clinical emergency that manifests with chest pain in the vast majority of cases and is most commonly located in the anterior wall of the heart.[12] In the U.S. alone, approximately 750,000 people suffer a myocardial infarction annually, with a significant portion being STEMI presentations.[13] The diagnosis of STEMI is confirmed by ECG criteria defined by the American College of Cardiology and related bodies, which include specific thresholds for ST-segment elevation in contiguous leads.[14] For patients with baseline conduction abnormalities, such as left bundle branch block, Sgarbossa's criteria help confirm STEMI.[15] By integrating traditional lipid profiles with apoB measurements, clinicians may better identify those at risk of future coronary events, enhancing the prevention and early management of STEMI. The aimed to assess the relation of apolipoprotein B (apoB) with early onset STEMI in Duhok, Kurdistan region of Iraq.

METHOD

This case-control study was conducted at Azadi Teaching Hospital and the Duhok Cardiac Center in Duhok Province, Kurdistan Region, Iraq, between May and December 2021. The study included 41 young patients (aged <50 years) who presented with ST-segment elevation myocardial infarction (STEMI), and 48 healthy controls matched for age and sex, without a history of coronary artery disease (CAD) or ischemic stroke. Individuals older than 50 years or those diagnosed with non-STEMI were excluded from the study. Demographic information, cardiovascular risk factors—including diabetes mellitus, hypertension, smoking, dyslipidemia, and family history of premature CAD—were collected using structured questionnaires. Risk factors were defined based on established criteria.[16,17] Blood pressure ≥140/90 mmHg or use of antihypertensives was considered hypertension. Diabetes mellitus was defined by fasting plasma glucose >126 mg/dl on two occasions or the use of antidiabetic medications. Dyslipidemia was defined as serum total cholesterol >200 mg/dl, LDL >100 mg/dl, HDL <40 mg/dl, triglycerides >150 mg/dl, or statin use. Chronic kidney disease was considered if serum creatinine exceeded 1.2 mg/dl in males or 1.0 mg/dl in females. A positive family history of CAD referred to events occurring in male relatives ≤45 years or female relatives ≤55 years. For both groups, 5 ml of fasting venous blood was collected for analysis of apolipoprotein B (apoB), lipid profile, glucose, and creatinine. ApoB was measured using immunoturbidimetric assay, with a reference range of 0.6–1.3 g/l. Other biochemical parameters were analyzed using a fully automated Cobas C 311 analyzer (Roche Diagnostics, Germany). Diagnosis of STEMI was based on clinical symptoms (e.g., chest pain >30 minutes), ECG changes (ST elevation ≥0.2 mV in leads V1–V3 or ≥0.1 mV in other leads), and elevated high-sensitivity cardiac troponins, consistent with current international guidelines.[30,31]

Data were analyzed using JMP Pro 14.3. Continuous variables were expressed as means ± standard deviation, and categorical variables as numbers and percentages. Comparisons between groups were performed using independent t-tests or Chi-squared tests. A p-value of <0.05 was considered statistically significant. Ethical approvals were obtained from the Iraqi Board of Medical Specializations and the Duhok Health Directorate. Written informed consent was secured from all participants.

RESULTS

In this study we made the homogeneity for age and gender between the cases and controls. In this regard, of the total 48 cases and 66 controls, 41 cases and 48 controls were included in this study. Compared to control arm the STEMI significantly was more among smokers (73.17% vs. 26.83%; P<0.001), in patients with prior CAD (19.51% vs. 2.08%; P=0.0066), in hypertension (39.02% vs. 10.42%; P=0.0015), and in patients with family history of IHD (34.15% vs. 14.58%; P=0.0303) as in (Table 1).

Table 1: Comparisons of clinical characteristics between patients with STEMI and healthy controls.

Characteristics	Study group no (%)		P-value (two-sided)
	Case (n=41)	Control (n=48)	
Age (years)	43.6 (5.2) Range: 34-50	41.7 (5.8) Range: 30-50	0.1135
Gender Female Male	 7 (17.07 34 (82.93	 16 (33.33 32 (66.67	0.0807
Smoking No Yes	 11 (26.83 30 (73.17	 35 (72.92 13 (27.08	**<0.0001**
BMI Normal Overweight Obese	 6 (14.63 20 (48.78 15 (36.59	 10 (20.83 28 (58.33 10 (20.83	**0.2466**

Diabetes			
No	23 (56.10	41 (85.42	**0.0022**
Yes	18 (43.90	7 (14.58	
Prior CAD			
No	33 (80.49	47 (97.92	**0.0066**
Yes	8 (19.51	1 (2.08	
Hypertension			
No	25 (60.98	43 (89.58	**0.0015**
Yes	16 (39.02	5 (10.42	
Family history of IHD			
No	27 (65.85	41 (85.42	**0.0303**
Yes	14 (34.15	7 (14.58	
Chronic drugs use			
No	31 (75.61	47 (97.92	**0.0014**
Yes	10 (24.39	1 (2.08	
Anti-lipid drugs			
No	33 (80.49	48 (100.00	**0.0013**
Yes	8 (19.51	0 (0.00	
Dyslipidemia			
No	13 (31.71	20 (41.67	**0.3323**
Yes	28 (68.29	28 (58.33	
[a] an independent t-test and [b] Pearson chi-squared test was performed for statistical analyses.			

The study showed that the prevalence of elevated ABO-B was not statistically significantly between the cases and controls (14.63% vs. 10.42%; p=0.5468). In addition, the patients and controls had no significant difference in the prevalence of renal impairment (2.44% vs. 0.00%; p=0.2765). The anterior (39.02%) and inferior STEMI (24.39%) were the most prevalence types of STEMI. The study found that the rate of in-hospital complications was 17.07% in patients with STEMI (Table 2).

Table 2: Comparisons of ABO-B level between the patients with STEMI and their healthy controls.

Outcomes	Study group no (%)		P-value (two-sided)
	Case	Control	
ABO-B level			
Elevated	6 (14.63	5 (10.42	0.5468
Normal	35 (85.37	43 (89.58	
Concentration	1.1 (0.3	1.0 (0.3	0.0634
Types of STEMI			
Anterior STEMI	16 (39.02		
Inferior STEMI	10 (24.39		
Lateral STEMI	6 (14.63	NA	NA
Multi-type	3 (7.32		
Posterior STEMI	1 (2.44		
Septal STEMI	5 (12.20		
In-hospital complications			
No	34 (82.93	14 (100	0.0979
Yes	7 (17.07	0 (0.00	
Renal impairment			
No	40 (97.56	48 (100	0.2765
Yes	1 (2.44	0 (0.00	
Pearson chi-squared test was performed for statistical analyses.			

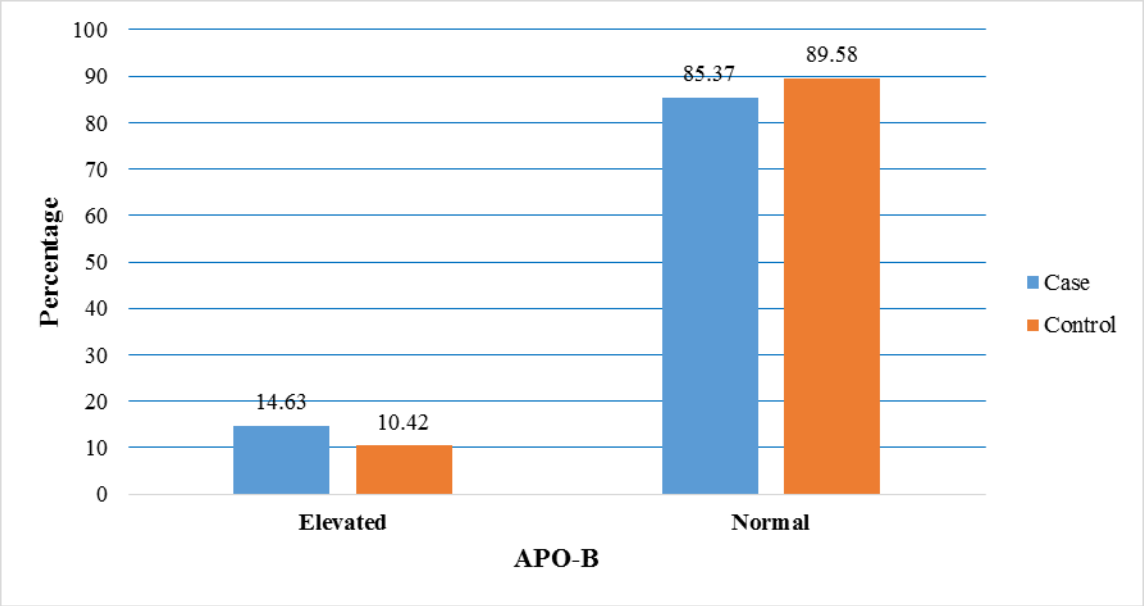

Fig 1: Prevalence of elevated and normal APO-B between the patients with STEMI and healthy controls.

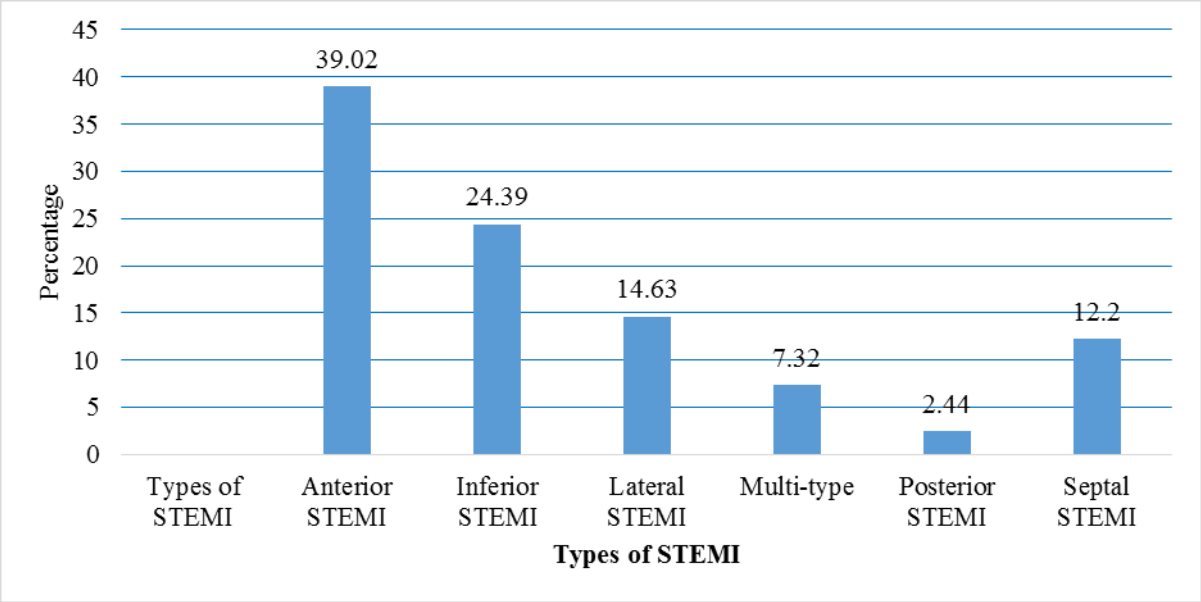

Fig 2: Types of STEMI in patients with STEMI.

DISCUSSION

The principal finding of this study was that there was no statistically significant difference in apoB levels between young patients with early-onset ST-segment elevation myocardial infarction (STEMI) and their age- and sex-matched healthy controls. While this contrasts with much of the existing literature, several factors may explain this observation. Notably, the small sample size and pilot nature of the study likely limited the statistical power needed to detect a significant association. Multiple prior studies have demonstrated a strong relationship between elevated apoB and cardiovascular disease risk. Avogaro and Sniderman et al. showed that high apoB levels, even in the presence of normal cholesterol levels, were associated with coronary atherosclerosis.[18,19] Similarly, in the Quebec Cardiovascular Study, Lamarche et al. found that apoB was independently predictive of coronary heart disease over a 5-year follow-up, even after adjusting for triglycerides, HDL-C, and the TC/HDL-C ratio.[20] Large-scale studies such as AMORIS[21], the Thrombo Metabolic Syndrome Study[22], the Northwick Park Heart Study[23], the Nurses' Health Study[24], and the Health Professionals Follow-up Study[25] consistently reported that apoB was more strongly associated with cardiovascular risk than LDL-C. In the AMORIS study, which followed over 175,000 individuals, apoB demonstrated higher sensitivity and specificity for predicting fatal myocardial infarction than LDL-C in both men and women.[21] The apparent lack of significance in our study may reflect both sample size limitations and measurement variability. For example, the ARIC study[26], despite a strong univariate association between apoB and cardiovascular outcomes, found that apoB's predictive power diminished in certain

subgroups due to a relatively high error rate in apoB measurement. We also observed a high prevalence of smoking among STEMI patients, consistent with findings from Ameen et al., who reported clustering of traditional risk factors such as smoking, male sex, and dyslipidemia among early-onset STEMI cases in Iraqi Kurdistan.[27] Additional support for apoB as a predictor of ischemic events comes from the Copenhagen City Heart Study, which showed apoB to be a better predictor than LDL-C for ischemic heart disease, stroke, and other vascular events.[28] Likewise, in the INTERHEART[29], ISIS[30], and ERFC[31] studies, apoB consistently outperformed LDL-C in predicting myocardial infarction across diverse populations. Nevertheless, some studies such as the Emerging Risk Factor Collaboration[32] and the Copenhagen Heart Study[33] suggested that apoB and non-HDL-C are equivalent predictors. Recognizing its predictive value, the 2018 National Cholesterol Education Program guidelines now recommend apoB measurement in primary prevention, especially when levels exceed 130 mg/dL.[34] While our findings did not show a statistically significant difference in apoB levels between STEMI cases and controls, the overall body of evidence underscores apoB's important role in cardiovascular risk stratification. Larger, well-powered studies are warranted to validate these findings in younger populations.

CONCLUSION

Even though we found higher levels of apoB in people with early-onset STEMI, our pilot study showed that apoB levels are not significantly linked to early-onset myocardial infarction, mainly because our small sample size limits the ability to apply our findings more broadly. The apoB should be correlated with other CV risk factors in the risk stratification of STEMI. Further cohort studies are_warranted to see the direct effect of apoB in the occurrence of early-onset STEMI in patients with and without conventional CV risk factors and to evaluate the sensitivity of this indicator in predicting ischaemia, particularly in young patients with normal or near-normal serum lipid levels.

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