



Egyptian Society of Cardiology
The Egyptian Heart Journal

www.elsevier.com/locate/ehj
www.sciencedirect.com



ORIGINAL ARTICLE

Lipid profile in Egyptian patients with coronary artery disease

M. Mohsen Ibrahim ^{a,*}, Ahmed Ibrahim ^b, Khaldoon Shaheen ^b, Mona A. Nour ^c

^a Cardiology Department, Cairo University, Faculty of Medicine, Egypt

^b Case Western Reserve University/St. Vincent Charity Hospital, USA

^c Tropical Medicine Department, Cairo University, Faculty of Medicine, Egypt

Received 18 June 2012; accepted 16 August 2012

Available online 13 October 2012

KEYWORDS

Lipid profile;
Coronary disease;
Risk factors;
Egypt

Abstract *Background:* No data are available about plasma lipid profile in Egyptians with coronary artery disease (CAD). Plasma lipid profile may differ according to ethnic origin and geographic area.

Objectives: Identify plasma lipid abnormalities in Egyptians with CAD and define the role of age, type of CAD, and the presence of hypertension (HT) on lipid profile.

Methods: Retrospective consecutive sampling of lipid profile of 1000 patients with CAD. Results were compared to a control group of 1920 non-coronary individuals.

Results: Patients' age range was 19–90 years. HT was present in 56.7% of patients. The commonest isolated lipid abnormality was a reduced HDL-C in men and increased plasma triglycerides (TG) in women. Patients with myocardial infarction (MI) had a lower HDL-C than those with angina pectoris (AP). Abnormalities were more severe and more prevalent in the young age group. No significant difference in lipid profile was present between normotensive (NT) and hypertensive (HT) CAD patients.

Conclusion: Dyslipidemia is common among Egyptians with CAD. Lipid profile was influenced by age, gender, type of CAD, but not by the presence of HT. The high prevalence rate of risk factors particularly among young Egyptians is remarkable and can explain the epidemic of CAD among Egyptians.

© 2012 Egyptian Society of Cardiology. Production and hosting by Elsevier B.V. All rights reserved.

1. Introduction

It is projected that CAD will be the leading cause of death in developing countries by the year 2020.^{1,2} In Egypt, mortality secondary to CAD is rapidly rising. According to WHO statistics³ the age-standardized mortality rates from CAD are one of the highest worldwide. One possible explanation is the high prevalence rate of CAD risk factors. Hypertension, dyslipidemia and obesity are common among Egyptians.⁴ Unfortunately, systematically documented data on CAD prevalence,

* Corresponding author.

E-mail addresses: ehs@link.net (M. Mohsen Ibrahim), ahmedibrahimucsd@gmail.com (A. Ibrahim), khaldoonshaheen@yahoo.com (K. Shaheen), mibrahim_02@yahoo.com (M.A. Nour).

Peer review under responsibility of Egyptian Society of Cardiology.



Production and hosting by Elsevier

incidence and rate of cardiovascular risk factors in developing countries are scarce.^{5,6} There is geographic and genetic variability in the prevalence of CV risk factors and in their contribution to the development of CAD.^{7,8} When investigating the relationship between CAD and lipid disturbances it is necessary to use local data on blood lipid profile in each region. The prevalence, type of lipid abnormalities and its association with CAD were not reported among Egyptians. Knowledge about the determinants of disease in persons within populations and of lipid profile in this group of relatively high-risk patients could be used to make recommendations on lipid management. Prevention programs will give priority to the most common risk factors and possibly the predominant type of dyslipidemia.

The present study has two objectives. First: to define the lipid profile in Egyptian patients with CAD who are not on lipid-lowering therapy. Second: to examine the effects of age, gender, type of CAD and the presence of hypertension on changes in lipid profile.

2. Methods

The study design was a retrospective consecutive sampling of all patients with CAD who satisfied the inclusion criteria. The data were collected from specialized cardiac clinic records during the period 1998–2006. Included in the study were patients with complete records showing detailed lipid profiles on 12 h fasting plasma samples who had a diagnosis of chronic stable angina or remote (at least 3 months) myocardial infarction and were not receiving statins or lipid lowering drugs on their initial clinic visit.

Plasma concentrations of total cholesterol and triglycerides were determined by enzymatic methods (Boehringer–Mannheim). HDL cholesterol was measured after precipitations of VLDL and LDL by the phosphotungstate method (Boehringer–Mannheim). LDL was estimated using the Friedwald formula when TG levels did not exceed 300 mg/dl and otherwise using direct quantitative homogenous enzymatic assays.

3. Control subjects

Data were collected during the Egyptian National Hypertension project (NHP), a national hypertension survey in Egypt. NHP was a multistage, national survey conducted between 1991 and 1993. The design and rationale of the NHP have been reported.^{4,9,10} The survey consisted of two phases. During phase I of the survey, hypertensive (HT) patients were identified. In phase II, clinical and laboratory evaluations were made on HT and gender matched normotensive (NT). A total of 2313 individuals were examined. After exclusion of CAD patients, there were 1920 patients with complete laboratory and clinical data, 716 were normotensive and 1404 hypertensive. Blood was collected after 12 h of fasting. Samples were processed locally, frozen at -30°C and then transferred to the basic central laboratory for batch analysis.

4. Ethics

No information that could identify a subject directly or indirectly was requested. Ethical approval of the study was, therefore, not required.

5. Definitions

High blood pressure (BP) was diagnosed based on at least two separate clinic visits (average of the last two of the 3 measurements during each visit) if mean SBP was ≥ 140 mmHg or DBP ≥ 90 mmHg or if patients were receiving treatment for hypertension.

5.1. Chronic stable angina

Classic history of typical anginal pain with evidence of myocardial ischemia on stress testing or an abnormal coronary angiogram.

5.2. Myocardial infarction

History and results of hospital records (ECG changes and elevated cardiac enzymes) or history and ECG changes showing pathologic Q waves. Only patients with remote MI (at least 3 months) were included in the study.

5.3. Overweight

Overweight was defined as BMI between 25–29.9 kg/m². Obesity, defined as ≥ 30 kg/m².

5.4. Dyslipidemia

Three different cutpoints were used to analyze the prevalence of high LDL-C and low HDL-C based on the different existing consensus recommendations. The selected cutpoints for high LDL-C were 100, 130 and 160 mg/dl and for low HDL were 35, 40 and 50 mg/dl. Cutpoint used to analyze prevalence of hypertriglyceridemia was (> 150 mg/dl). A total plasma cholesterol level ≥ 200 mg/dl was considered abnormal.

Diabetes mellitus was diagnosed if fasting plasma glucose was ≥ 126 mg/dl on two laboratory results or if the patient was on antihyperglycemic therapy.

6. Statistical analysis

The following data were entered into a computer program with SPSS statistical package for detailed statistical analysis:

- (1) Demographic characteristics: age and gender.
- (2) Lipid profile: total cholesterol (TC), LDL-C, HDL-C, triglycerides and TC/HDL-C ratio.
- (3) BMI.
- (4) Hypertension state.

Mean values were reported for continuous variables. Prevalence and frequencies are expressed in terms of percentage. Comparison between groups was performed using the chi-squared test to compare frequencies, the student t-test to compare mean values of the 2 groups.

Results were classified into 3 age groups for normotensive (NT) and hypertensive (HT) subjects and for men and women when the numbers in each gender type were adequate.

7. Results

A thousand consecutive patients with CAD satisfied the inclusion criteria. The results were compared with findings in 716 NT and 1404 HT subjects without coronary disease who served as a control group.

7.1. Population Characteristics

The age of the patients ranged from 19–90 years old with a mean age of 54 years, 77.6% of the patients were males, 543 patients had stable CAD (angina-AP) while 457 had a history of myocardial infarction (MI). Hypertension was present in 56.7% of patients, obesity (BMI \geq 30 kg/m²) in 25.8%, and diabetes mellitus in 34.4%.

7.2. Prevalence of lipid abnormalities in patients with CAD

Mean values (mg/dl) for LDL-C, HDL-C, TC and triglycerides (TG) were 140.6, 41.5, 217, and 160.9, respectively. Low HDL-C (< 40 mg/dl) was present in 49.2% of CAD patients, increased LDL-C (> 160 mg/dl) in 30.2%, increased triglycerides (> 150 mg/dl) were present in 45% of patients. The pattern of lipid abnormalities differed according to gender (Fig. 1) and type of CAD. In males, the most frequent abnormality was a low HDL-C (55.4%). In females, the commonest abnormality was increased LDL-C (41.1%). Fig. 2 is a pie chart showing the distribution of isolated and combined lipid abnormalities in CAD patients for men and women. Table 1 shows the prevalence of lipid abnormalities at different cutoff points.

Patients with MI had a lower level of HDL-C (HDL-C < 40 mg/dl) than AP patients (62.2% versus 40%, respectively). Females with MI have the highest prevalence rate of increased LDL-C present in 51.7%. Male patients with MI have the highest rates of increased TG (49.4%). When cutoff points for low HDL-C were based upon the National Cholesterol Education Program (< 40 mg/dl for men and < 50 mg/dl for women) 55.4% of male patients and 65.6% of female patients had abnormal low HDL-C.

Among patients with CAD, a completely normal blood lipid profile was present in 20.2% of male patients and 29.9% of females. Low HDL-C (< 40 mg/dl) as the only lipid abnormality was present in 21.4% of males and 7.6% of females. In-

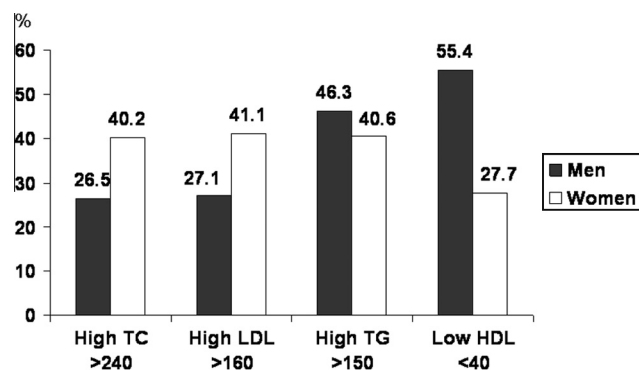


Figure 1 Frequency of lipid abnormalities in Egyptian men and women with CAD.

creased LDL-C > 100 mg/dl was present in more than 80% of patients. Low HDL-C with normal LDL-C was present in 34.6% of CAD patients.

7.3. Effect of age

Table 2 shows the lipid profile of CAD patients in the three age groups. Plasma levels of TC, LDL-C and TG were higher in the young compared to middle age and elderly patients. There was a trend for a decrease in plasma levels with advancing age. There was no difference in the prevalence of hypertriglyceridemia and very low HDL-C (< 35 mg/dl) between the 3 age groups. TC/HDL-C ratio was higher in the young compared to the middle age group. Obesity was more prevalent in young CAD patients than in middle age and elderly patients.

7.4. Comparison of NT and HT CAD patients

Table 3 shows a comparison of plasma lipids, prevalence of obesity and diabetes in normotensive and hypertensive CAD patients. Hypertensive patients were older than normotensive patients. There were no significant differences between the two groups as a whole in plasma lipid levels. The only significant difference between normotensive and hypertensive patients was in the TG levels which were higher in young hypertensive patients and in LDL-C levels which were higher in elderly hypertensives. A higher prevalence of low HDL-C (\leq 35 mg/dl) was present among the young hypertensives. There was a tendency of a higher TC/HDL-C ratio among hypertensive patients. Obesity was more common in middle age hypertensives. Prevalence of diabetes did not differ between NT and HT CAD patients.

7.5. Comparison of NT CAD patients and NT controls

In all age groups, levels of plasma TC, LDL-C and TG were higher in NT CAD patients compared to the NT controls. Prevalence of abnormalities in lipid levels (increased TC, LDL-C and TG) was significantly greater only in young and middle age groups. Plasma low HDL-C level and its prevalence were higher only in middle age patients. Diabetes was more prevalent in all age groups of CAD patients. Obesity was more common in the middle age control group than in CAD patients. Table 4 shows lipid abnormalities and prevalence of diabetes and obesity in normotensive CAD patients and normotensive control subjects at different age groups.

Advancing age was associated with a tendency to increased LDL-C plasma levels in normal subjects while the reverse was present in CAD patients. Fig. 3 shows that the trend in normotensive controls was an increase in LDL-C (\geq 160 mg/dl) till the age of 60 years; while the reverse was present in CAD patients. Also, there was a trend for lower TG levels with advancing age in CAD patients. HDL-C plasma levels were not influenced by age in controls and CAD patients.

8. Discussion

This is the first report of plasma lipid profile among Egyptian patients with CAD. It is one of the few studies in a developing country involving a large number of CAD patients over a wide age range. The effects of type of CAD, MI vs. AP, age, gender,

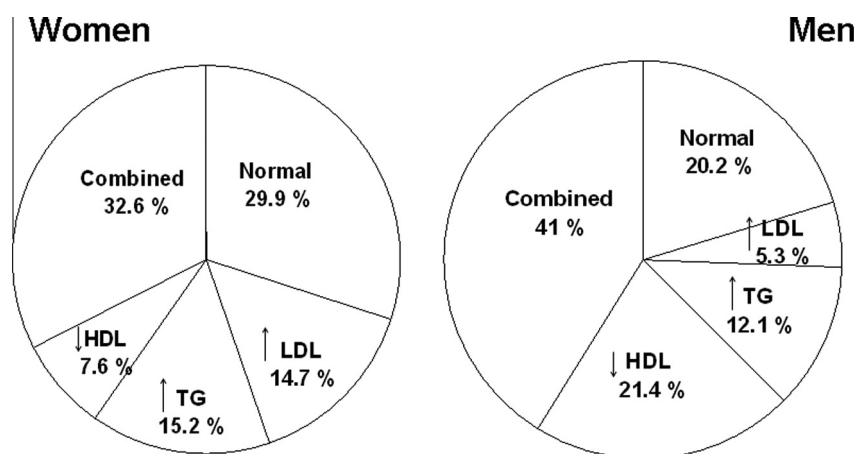


Figure 2 Frequency of isolated and combined lipid abnormalities in Egyptian men and women with CAD.

Table 1 Prevalence (%) of lipid abnormalities in 1000 CAD patients.

	No.	TC (> 200 mg/dl)	TG (> 150 mg/dl)	LDL (> 160 mg/dl)	LDL (> 130 mg/dl)	LDL (> 100 mg/dl)	HDL (< 35 mg/dl)	HDL (< 40 mg/dl)	HDL Males (< 50 mg/dl) females
Total	1000	58.7	45.0	30.2	54.5	82.4	28.9	—	—
M.	776	55.3	46.3	27.1	51.8	81.4	33.1	55.4	—
F.	224	70.5	40.6	41.1	63.8	85.7	14.3	—	65.6
HT.	567	61.6	47.4	31.2	56.1	83.1	29.1	56.4	64.2
NT.	433	55	41.8	28.9	52.4	81.5	28.6	54.3	69.2
MI	457	57.5	48.8	32.2	55.8	81.4	36.8	—	—
AP	543	59.7	41.8	28.5	53.4	83.2	22.3	—	—
MI M.	399	55.9	49.4	29.3	53.6	80.7	38.3	62.4	—
MI F.	58	70	44.8	51.7	70.7	86.2	25.9	—	77.6
AP M.	377	54.6	42.9	24.7	49.9	82.2	27.6	48	—
AP F.	166	71.1	39.2	37.3	61.4	85.5	10.2	—	61.4
DM.	344	59.6	48.3	30.2	54.7	82.3	29.9	58.2	65.3
Obesity	522	65.3	51.2	32.6	59.7	85.2	27.1	58.8	62.6

M: males F: females.

MI: myocardial infarction AP: stable angina pectoris.

DM: diabetes mellitus Obesity: BMI \geq 30 kg/m².

high BP and body weight are described. The fact that only a minority of Egyptian CAD patients receive statin therapy¹¹ allowed the recruitment of a large number of patients not on lipid modifying therapy. The results were compared to a control population of normotensive and hypertensive Egyptians without CAD in the same age groups. The commonest isolated lipid abnormality was a reduced HDL-C in men (21.4%) and increased plasma TG (> 150 mg/dl) in women (15.2%). A completely normal plasma lipid profile was present in 20.2% of male and 29.9% of female patients.

The overall prevalence of dyslipidemia in our CAD patients was greater than previously reported.^{12–15} Mohan et al.¹⁶ found no difference in mean HDL-C levels between CAD and non CAD Asian Indians. On the other hand, Rubins et al.¹⁷ while comparing their population with CAD with the general US men found a striking difference between the 2 populations in all age groups, lower levels of HDL-C and higher level of TG in men with CAD. Ethnic differences, dietary habits and lifestyle may explain differences between different populations.

A low HDL-C (< 35 mg/dl) was reported in 38% of CAD male patients,¹⁵ while HDL-C < 40 mg/dl was present in 49% of our CAD patients and in 45% of Jordanian CAD patients.¹⁴ Data from a number of epidemiologic studies showed that low HDL-C is not uncommon in the general population.^{17–20} A high prevalence of low HDL-C (< 35 mg/dl) was present in normotensive Egyptian men and women (32.2% and 20.6%, respectively).⁴

Increased triglycerides (> 150 mg/dl) were present in 45% of our CAD patients while it ranged between 23% and 44.5% in control subjects depending upon age, presence or absence of hypertension. Other studies from our region found that CAD patients had significantly higher TG and TC and lower HDL-C levels than individuals with no CAD.^{14,21}

Our young CAD patients (< 40 years) had higher prevalence of plasma lipid abnormalities compared to middle age and elderly patients. The tendency for increased LDL-C with advancing age in our control group and previously reported in normal subjects^{4,22,23} was not present and even reversed in our CAD patients. Aging appeared to attenuate the

Table 2 Lipid profile in the three age groups.

Age (years)				<i>P</i>		
	< 40 (1)	40–60 (2)	> 60 (3)	(1) vs (2)	(2) vs (3)	(1) vs (3)
No.	62	674	264			
TC (mg/dl)	231.0	217.8	213.4	0.05	0.2	0.01
LDL-C (mg/dl)	152.8	141.4	135.8	0.04	0.07	0.005
HDL-C (mg/dl)	41.5	40.5	43.8	0.4	0.000	0.1
TG (mg/dl)	178.5	163.3	150.8	0.1	0.02	0.004
↑TC (> 200 mg/dl) (%)	71	60.4	58.2	0.3	0.3	0.04
↑LDL-C (%)						
> 100 (mg/dl)	88.9	83.7	76.9	0.1	0.6	0.08
> 130 (mg/dl)	72.6	54.7	49.6	0.9	0.7	0.001
> 160 (mg/dl)	40.3	30.4	27.3	0.1	0.1	0.03
Low HDL-C (%)						
< 35 (mg/dl)	27.4	32.6	19.7	0.4	0.6	0.4
< 40 (mg/dl)	45.2	53.0	40.5	0.5	0.06	0.1
< 50 (mg/dl)	80.6	83.2	73.9	0.2	0.001	0.5
↑TG (%)						
> 150 (mg/dl)	45.2	53.7	60.6	0.2	0.3	0.7
TC/HDL-C	5.8	5.6	5.0	0.5	0.000	0.001
↑BMI (≥ 30 kg/m ²)%	21.0	12.9	11.0	0.02	0.02	0.01

Table 3 Comparison of NT and HT CAD patients at different age groups.

Age	All			< 40			40–60			> 60		
	NT	HT	<i>P</i>	NT	HT	<i>P</i>	NT	HT	<i>P</i>	NT	HT	<i>P</i>
No.	433	567		39	23		309	365		85	179	
Age mean	51.96	55.78	< 0.001	35.1	34.9	0.8	49.9	51.7	0.000	66.9	66.8	0.9
TC (mg/dl)	214.45	219.78	0.09	231.9	229.5	0.8	213.9	221.1	0.06	208.4	215.7	0.2
LDL-C (mg/dl)	138.84	141.99	0.2	143.2	145.9	0.6	139.6	143.8	0.1	134.1	144.4	0.05
HDL-C (mg/dl)	41.20	41.68	0.4	43.6	41.8	0.2	39.8	42.0	0.01	41.8	40.8	0.1
TG (mg/dl)	156.49	164.38	0.1	146.3	170.0	0.01	163.9	162.4	0.8	150.6	164.3	0.09
↑TC (> 200 mg/dl)(%)	57.3	63.1	0.06	69.2	73.9	0.4	56.6	63.6	0.04	54.1	60.9	0.1
↑LDL-C (%)												
≥ 100 mg/dl	81.5	82.7	0.3	89.7	87.0	0.5	81.2	85.8	0.07	78.8	76.0	0.3
≥ 130 mg/dl	52.4	56.1	0.1	71.8	73.9	0.5	52.8	56.4	0.1	42.4	53.1	0.06
≥ 160 mg/dl	28.9	31.2	0.2	35.2	36.7	0.4	30.5	29.0	0.3	21.3	31.8	0.03
LOW HDL-C (%)												
< 35 mg/dl	28.6	29.1	0.8	18.7	30.3	0.04	33.6	28.4	0.1	27.0	29.7	0.3
< 40 mg/dl	50.3	48.3	0.2	43.6	47.8	0.4	52.4	53.4	0.4	45.9	38.0	0.1
< 50 mg/dl	83.1	78.7	0.07	79.5	82.6	0.5	85.1	81.6	0.1	77.6	72.1	0.2
↑TG(%)												
≥ 150 mg/dl	41.8	47.4	0.07	64.8	47.7	0.01	55.5	53.9	0.3	58.2	53.4	0.2
TC/HDL-C	5.4	5.5	0.3	5.3	5.7	0.06	5.6	5.4	0.2	5.3	5.6	0.08
Diabetes (%)	30.9	37.0	0.04	28.6	33.0	0.3	28.6	38.4	0.1	36.9	37.0	0.5
BMI (kg/m ²)	25.4	25.9	0.1	25.5	26.2	0.5	25.1	26.3	0.000	26.2	25.1	0.3
↑BMI(≥ 30 kg/m ²)%	12.0	13.4	0.06	15.4	13.8	0.7	10.5	15.2	0.01	13.1	9.5	0.5

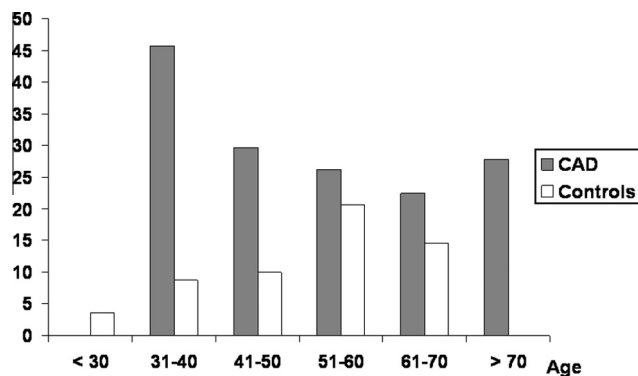
differences between our CAD patients and controls. Chodorowski et al.²⁴ found in patients with acute MI a decline in LDL-C until the age of 68, HDL-C levels did not change with age. Even though total cholesterol concentration represents a significant risk factor in the elderly, there is evidence that this relationship weakens progressively with advancing age to the point where TC levels do not appear

to contribute to the risk of CAD or overall mortality beyond the age of 70 years.^{25,26}

Abnormalities in plasma lipids and blood sugar were reported to be more frequent in hypertensive patients compared to normotensives.^{4,20,27} This was not present in our CAD patients except for higher TG levels in young and higher LDL-C in elderly hypertensive patients. Hammoudah et al. found

Table 4 Comparison of lipid profile in normotensive CAD and normotensive controls.

	< 40			40–60			> 60		
	CAD	Controls	<i>P</i>	CAD	Controls	<i>P</i>	CAD	Controls	<i>P</i>
No.	39	299		309	341		85	76	
Age mean	35.1	32.1	0.000	49.9	48.2	0.000	66.9	67.6	0.4
TC (mg/dl)	231.9	175.7	0.000	213.9	191.1	0.000	208.4	190.05	0.009
LDL (mg/dl)	156.6	109.8	0.000	138.6	120.6	0.000	131.6	121.9	0.1
HDL (mg/dl)	42.3	42.3	0.9	40.6	42.0	0.04	43.1	42.5	0.4
TG (mg/dl)	160.4	118.9	0.000	158.8	145.2	0.01	146.2	127.2	0.03
↑ TC (> 200 mg/dl) (%)	69.2	22.6	0.000	56.6	36.4	0.000	54.1	37.8	0.02
↑ LDL-C (%)									
≥ 100 (mg/dl)	89.7	62.0	0.000	81.2	68.9	0.000	78.8	79.5	0.5
≥ 130 (mg/dl)	71.8	24.7	0.000	52.8	36.9	0.000	42.4	42.5	0.5
≥ 160 (mg/dl)	43.6	6.2	0.000	28.5	13.8	0.000	23.5	11.0	0.03
Low HDL-C (%)									
< 35 (mg/dl)	23.1	24.6	0.5	31.1	26.2	0.07	22.4	25.7	0.2
< 40 (mg/dl)	43.6	42.7	0.5	52.4	44.9	0.02	45.9	43.2	0.5
< 50 (mg/dl)	79.5	76.1	0.3	85.1	76.5	0.003	77.6	67.6	0.1
↑ TG (%)									
≥ 150 (mg/dl)	56.4	20.3	0.000	57.9	35.5	0.000	60.0	23.0	0.000
TC/HDL-C	5.7	4.4	0.000	5.5	4.8	0.000	5.1	4.7	0.2
Diabetes (%)	12.8	3.8	0.02	32.0	8.5	0.000	35.3	12.2	0.000
BMI (kg/m ²)	25.5	27.7	0.03	25.1	28.4	0.000	26.2	26.0	0.8
↑BMI (≥ 30 kg/m ²)%	23.1	23.7	0.9	10.7	30.1	0.000	11.8	19.5	0.3

**Figure 3** Prevalence of increased LDL-C (≥ 160 and ≥ 100 mg/dl) in NT CADs and NT controls in different age decades.

that hypertension had no effect on serum lipid levels.¹⁴ It is possible that lipid abnormalities present in CAD patients mask the difference between normotensive and hypertensive subjects.

A comparison of normotensive CAD and normotensive controls in the same age group showed significant differences in plasma lipid abnormalities of TC, LDL-C and TG which were more prevalent in CAD patients. Diabetes was more than 3 times frequent in normotensive CAD patients compared to normotensive controls in the same age group, while there was no difference in the prevalence of diabetes between normotensive and hypertensive CAD patients.

The prevalence of obesity in our normotensive subjects was greater in the control group than CAD patients particularly in the middle age. This was an unexpected observation, since obesity is considered an independent risk factor for CAD²⁸ and was associated with more frequent lipid abnormalities. One explanation is the type of body fat distribution, since visceral abdom-

inal obesity is the one associated with increased cardiometabolic risk.²⁹ Information about waist circumference and type of obesity-visceral vs. subcutaneous is not available in our study.

We found difference in lipid profile between MI and AP patients, which was gender dependent. In general, patients with MI had a lower level of HDL-C than AP patients. ACS in Jordanian patients had lower total cholesterol, triglycerides and LDL-C levels compared with those with chronic CAD.¹⁴

The study population was recruited from a single cardiac referral center. Therefore, the data are not representative of the whole nation and are liable to selection bias. Because of the relatively large number and inclusion of both genders over a wide age spectrum they possibly represent a large segment of the Egyptian CAD patients. The absence of information about other risk factors e.g. cigarette smoking might have influenced some of the results. Angiographic documentation of CAD was not available in all patients. The use of different cutoff points diagnostic of lipid abnormalities, though confusing, makes possible a comparison of our prevalence data with other reported studies which use different diagnostic thresholds.

9. Conclusion

This is the first report of plasma lipid abnormalities in Egyptians with CAD. Lipid abnormalities were present in about 80% of patients. The type of plasma lipid abnormality differed according to age, gender and mode of presentation of CAD, while the presence of hypertension had a limited effect on the changes in lipid profile. Dyslipidemia was especially common among young CAD patients and with aging the prevalence of lipid abnormalities was gradually attenuated.

This high prevalence of plasma lipid abnormalities in our Egyptian patients underscores the need for a nationwide public

awareness campaign promoting healthy diet and physical activity. Routine screening in high risk groups for dyslipidemia and other risk factors should be encouraged.

Acknowledgments

The authors acknowledge the excellent secretarial work of Mrs. Rehab M. El-Ashkar who helped in the editing and typing of the manuscript.

References

- Murray CJL, Lopez AD. *The global burden of disease: a comprehensive assessment of mortality and disability from disease, injuries and risk factors in 1990 and projected to 2020*. Boston (Mass): Harvard School of Health; 1996.
- The World Health Report 1999: The double burden: Emerging epidemics and persistent problems. WHO 1999. Available at: <<http://www.who.org>>.
- World Health Statistics 2008: WHO. Available at: <http://www.who.int/whosis/whostat/EN_WHS08_TOCintro.pdf>.
- Ibrahim MM, Appel LJ, Rizk HH, Helmy S, Mosley J, Ashour Z, et al. Cardiovascular risk factors in normotensive and hypertensive Egyptians. *J Hypertens* 2001;**19**:1933–40.
- Beaglehole R. International trends in coronary heart disease mortality and incidence rates. *J Cardiovasc Risk* 1999;**6**:63–8.
- Okraïnec K, Banerjee DK, Einsenberg MJ. Coronary artery disease in the developing world. *Am Heart J* 2004;**148**:7–15.
- Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanus F, et al. INTERHEART study investigators. Effects of potentially modifiable risk factors associated with myocardial infarction in 52 countries (The INTERHEART Study): case-control study. *Lancet* 2004;**364**:937–42.
- Johnson CL, Rifkind BM, Sempos CT, Carroll MD, Bachorik PS, Briefel RR, et al. Declining serum total cholesterol levels among US adults. The National Health and Nutrition Examination Surveys. *JAMA*. 1993;**269**(23):3002–8.
- Ashour Z, Ibrahim MM, Appel LJ, Ibrahim AS, Whelton PK. For the NHP investigative team. The Egyptian National Hypertension Project (NHP): design and rationale. *J Hypertens* 1992;**26**:880–5.
- Ibrahim MM. The Egyptian National Hypertension Project (NHP): preliminary results. *J Human Hypertens* 1996;**10**(suppl):S39–41.
- Mendis S, Abegunde D, Yusuf S, Ebrahim S, Shaper G, Ghannem H. WHO-PREMISE (phase I) study Group). WHO study on prevention of recurrences of myocardial infarction and stroke (WHO-PREMISE. *Bull World Health Organ* 2005;**83**:820–8.
- Lamarche B, Despre J-S, Moorjani S, Cantin B, Dagenais G, Lupien P, et al. Prevalence of dyslipidemic phenotypes in ischemic heart disease (prospective results from the Quebec cardiovascular study). *Am J Cardiol* 1995;**75**:1189–95.
- Genest JJ, McNamara JR, Ordovas JM, Jenner JL, Silberman SR, Anderson KM, et al. Lipoprotein cholesterol, apolipoprotein A-I and B and lipoprotein(a) abnormalities in men with premature coronary artery disease. *J Am Coll Cardiol* 1992;**19**:792–802.
- Hammoudeh AJ, Izraïq M, Al-Mousa E, Al-Tarawneh H, Elharassis A, Mahadeen Z, et al. Serum lipid profiles with and without CAD: Jordan hyperlipidemia and related targets study (JoHARTS-1). *East Mediterr Health J* 2008;**14**:24–32.
- Rubins H, Robins S, Collins D, Iranmanesh A, Wilt T, Mann D, et al. Distribution of lipids in 8,500 men with coronary artery disease. *Am J Cardiol* 1995;**75**:1196–201.
- Mohan V, Deepa R, Rani SS, Premolatha G. Prevalence of coronary artery disease and its relationship to lipids in a selected population in South India: the Chennai Urban Population Study (CUPS No.S). *J Am Coll Cardiol* 2001;**38**:682–7.
- Schaefer EJ, Lamon-Fava S, Ordovas JM, Cohn SD, Schaefer WP, Castelli WP, et al. Factors associated with low and elevated plasma high density lipoprotein cholesterol and apolipoprotein A-I levels in the Framingham offspring study. *J Lipid Res* 1994;**35**:871–82.
- Health Survey of England 1998: Cardiovascular disease <<http://www.dh.gov.uk/PublicationsandStatistics/PublishedSurvey/HealthSurveyforEngland/HealthSurveyResults/Fa/en>>.
- Assmann G, Schulte H, Eckardstein von, Huang Y Y. High density lipoprotein cholesterol as a predictor of coronary heart disease risk. The PROCAM experience and pathophysiological implications for reverse cholesterol transport. *Atherosclerosis* 1996;**124**(Suppl):S11–20.
- Aguilar-Salinas CA, Olaïz G, Valles V, Torres JM, Gómez Pérez JA, Rull JA, et al. High prevalence of low HDL cholesterol concentrations and mixed hyperlipidemia in a Mexican nationwide survey. *J Lipid Res* 2001;**42**(8):1298–307.
- Al-Kateb H, Marzour W, Shameah M, Juoma M. Coronary risk factors of angiographically assessed patients from Syria. *J Cardiovasc Risk* 1998;**5**:31–5.
- Kreisberg RA, Kasim S. Cholesterol metabolism and aging. *Am J Med* 1987;**82**:54–60.
- Ericsson S, Eriksson M, Vitols S, Einarsson K, Berglund L, Angelin B. Influence of age on the metabolism of plasma low density lipoproteins in healthy males. *J Clin Invest* 1991;**87**(2):591–6.
- Chodorowski Z, Anand JS, Foerster J, Gruchaa M, Chlebus K. Differences in lipid profile in patients with first myocardial infarction occurring at different ages. *Borgis – New Medicine* 2004;**2**:48–51.
- Kronmal RA, Cain KC, Ye Z, Omenn GS. Total serum cholesterol levels and mortality risk as a function of age. A report based on the Framingham data. *Arch Intern Med* 1993;**153**:1065–73.
- Krumholz HM, Seeman TE, Merrill SS, Mendes de Leon CF, Vaccarino V, Silverman DI, et al. Lack of association between cholesterol and coronary heart disease mortality and morbidity and all cause mortality in persons older than 70 years. *JAMA* 1994;**272**(17):1335–40.
- Ferrannini E. Metabolic abnormalities in hypertension: a lesson on complexity. *Hypertension* 1991;**18**:636–9.
- Allison DB, Fontaine KR, Manson JE, Stevens J, VanItallie TB. Annual death attributable to obesity in the United States. *JAMA* 1999;**282**:1530–8.
- Ibrahim MM. Subcutaneous and visceral adipose tissue: structural and functional differences. *Obes Rev* 2009;**11**(1):11–8.