The role of triglycerides as a risk factor of ischemic stroke remains controversial, however some studies reported a strong association between elevated levels of postprandial triglycerides and increased risk of myocardial infarction, ischemic heart disease, and ischemic stroke [5-7]. The atherosclerosis has long been hypothesized to be a disorder influenced by postprandial effects of TG as early as 1950, when Moreton, suggested a linkage between chylo-micronemia, fat tolerance, and atherosclerosis by affecting endothelial function and producing the atherogenic small LDL particles [8].

Thus measurement of postprandial triglycerides, particularly because they peak 3-4 h after ingestion of a fat-rich meal, might provide more relevant information on vascular risk than measurements based on fasting concentrations [9]. Recent studies found a strong association between elevated levels of postprandial triglycerides, and increased risk of ischemic heart disease [10].

Low serum testosterone levels have been associated with several components of metabolic syndrome, including CVD, hypertension, abdominal obesity, insulin resistance, and inflammatory markers in male individuals independent of age [11,12].

Also, it has been shown that low endogenous testosterone levels are associated with increased risk for both all-cause and cardiovascular mortality [13,14]. Testosterone is a muscle-building hormone, and there are many testosterone-receptor sites in the heart. The weakening of the heart muscle can sometimes be attributed to testosterone deficiency [15].

Testosterone is not only responsible for maintaining heart muscle protein synthesis; it is also a promoter of coronary artery dilation and helps to maintain healthy cholesterol levels [16-18].

The aim of the present study was to stress on the importance of postprandial lipids, in general, and triglycerides, in particular, and its relation to serum testosterone level in evaluating the risk of CVD in males.

Methods

This study included 40 male patients with CVD of age range between 30-60 years and disease duration of 2-15 months, who were attending the Coronary Care Unit (CCU) at Baghdad medical city during the period from December 2011 to June 2012 between 9.00 and 12.00 am. Patients with diabetes mellitus and thyroid disease were excluded from the study. The study also included 46 normal male volunteers of matching age and BMI, Who were non-smokers; non alcoholics and none, had dyslipidemia as revealed from previous laboratory tests.

Ten milliliters (10 ml) of venous blood were withdrawn from both patients and controls, collected in plain tube and centrifuged for 15 minutes at 3000rpm after being allowed to clot at room temperature for 30 minutes. The separated sera were divided into aliquots and stored frozen at -20 ^{o}C to be used for hormonal assays. Postprandial blood glucose, lipid profile, urea and creatinine were measured immediately after separation of the serum. In this study the determination of patients' androgen sex hormones; luteinizing hormone (LH) [19,20], follicle stimulating hormone (FSH) [21], testosterone [22], sex hormone binding globulin (SHBG) [23] were measured by enzyme linked Enzyme-Linked Immune Sorbent Assay (ELISA, Sandwich Assay). Body mass index was calculated as body weight (in Kg)/Sq. height (in meter).

Statistical study

All values were expressed as mean ± standard deviation (mean ± SD). All Statistical analysis was performed using Social process statistical system (SPSS version 15.0). Independent student t-test was performed to assess differences between two means. Pearson correlation coefficient was used to determine the correlation between quantitative data. P value < 0.05 was considered significant.

Results

As shown in table 1, there is a significant difference in total testosterone between the two