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Study Effect of Fibrinogen on Patients with Cardiovascular Disease

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Abstract: Cardio vascular disease has become a great problem throughout the world. It is connected to increase morbidity and mobility and decrease quality of life in patients compared to the general population. many cases of disease has been described in cardiovascular disease. The study conducted on too Iraqi patient with chronic atherosclerosis at age rang (29-70years) in Baquba teaching hospital in (center care unit) during the period from 25 September 2020to may 2021. The patients divided in (55 mans and 45 patients) the total patient divided in tow groups according the treatment (30) of total patient under go to treatment for two to three days after atherosclerosis and 70 patient in same day of diagnosis of atherosclerosis. Fibrinogen value increase and we can use this as biomarkers with Atherosclerosis.

Introduction

Our knowledge of 'accepted' cardiovascular risk factors has become widespread. In several countries its application to clinical practice has started to bring about a considerable reduction of cardiovascular mortality and morbidity. Yet our understanding of such risk factors is still incomplete. Only about 30% of all cardiovascular events can be 'explained' on the basis of 'accepted' risk factors.[1]. Additional risk factors are therefore likely to exist. An increasing body of evidence suggests that fibrinogen is a prime candidate.[2].This review is aimed at summarizing recent, important data linking fibrinogen with cardiovascular risk in clinical and epidemiological research.

Physiology Human fibrinogen is a long, dimeric protein of 340 000 daltons molecular mass. It consists of three different pairs of polypeptide chains that are held together by disulphide bonds to form a trinodular structure. Thus the molecule comprises 2 main subunits, each consisting of 3 polypeptide chains. These are encoded by different genes (FOA, FOB, FOe) located on chromosome 4. The synthesis of the B chain is the rate-limiting process that is also influenced by interleukin-6. Thus, interactions between genetic and external factors are conceivable. Fibrinogen is synthesized by parenchymal liver cells from where it enters the circulation. Eighty to ninety per cent of the body's fibrinogen circulates in the blood plasma. 'Normal' plasma levels range from 2 to 4.5 gil. The molecule's half-life is 3-6 days. The synthesis of fibrinogen is believed to be under the feedback control of its plasma degradation products and cytokines, particularly interleukin-I, produced by activated macrophages. Fibrinogen is best appreciated as an acute phase protein and a clotting factor but it also has numerous other functions [3,4]. It is an essential cofactor for platelet aggregation, a powerful determinant of the rheological behaviour of blood, and a stimulant of smooth muscle cell migration and proliferation. These and other phenomena may be involved in its ability to be a cardiovascular risk factor.

Experimental part

PRINCIPLE OF THE ASS AY:

This FGLI enzyme linked immunosorbent assay applies a technique called a quantitative sandwich immunoassay. The microtiter plate provided in this kit has been pre-coated with a monoclonal antibody specific for FGLI. Standards or samples are then added to the microtiter plate wells andFGL1 if present, will bind to the antibody pre-coated wells. In order to quantitatively determine the amount of FGLI present in the sample, a standardized preparation of horseradish peroxidase (HRP)-conjugated polyclonal antibody, specific for FGLI are added to each well to "sandwich" the FGLI immobilized on the plate. The microtiter plate undergoes incubation, and then the wells are thoroughly washed to remove all unbound components. Next, A and B substrate solution is added to each well. The enzyme (HRP) and substrate are allowed to react over a short incubation period. Only those wells that containFGL1 and enzyme-conjugated antibody will exhibit a change in colour. The enzyme-substrate reaction is terminated by the addition of a sulp huric acid solution and the colour change is measured spectrophotometrically at a wavelength of 450 nm.

REAGENT PREPARATION:

1. Bring all kit components and samples to room temperature (18-25 C) before use.
2. Dispense 10u 1 of LYSIS BUFFER SOLUTION into 100u 1 specimens, mix and stand for hour (The proportion of LYSIS BUFFER and Specimens shall be no less than 1:10.
3. Wash Solution - Dilute 10mL of Wash Solution concentrate (10x) with 990mL of deionized or distilled water to prepare 1000mL of Wash Solution (1x).

ASS AY PROCEDURE:

Prepare all Standards before starting assay procedure (Please read Reagents Preparation). It is recommended that all Standards and Samples be added in duplicate to the Microtiter Plate.

1. Secure the desired number of coated wells in the holder then add 50 u L of Standards or Samples to the appropriate well of the antibody pre-coated Microtiter Plate.
2. Add 100 u L of Conjugate to each well Mix well. Complete mixing in this step is important. Cover and incubate for 1 hour at 37°C.
3. Wash the Microtiter Plate using one of the specified methods indicated below.
4. Manual Washing: Remove incubation mixture by aspirating contents of the plate into a sink or proper waste container. Using a squirt bottle, fill each well completely with wash solution, then aspirate contents of the plate into a sink or proper waste container. Repeat this procedure four more times for a total of FIVE washes. After final wash, invert plate, and blot dry by hitting plate onto absorbent paper or paper towels until no moisture appears. Note: Hold the sides of the plate frame firmly hen washing the plate to assure that all strips remain securely in frame.
5. Automated Washing: Aspirate all wells, and then wash plate FIVE times using wash solution. Always adjust your washer to aspirate as much liquid as possible and set fill volume at 350 u L/well/wash (range: 350-400 u L). After final wash, invert plate, and blot dry by hitting plate onto absorbent paper or paper towels until no moisture appears. It is recommended that the washer be set for a soaking time of 10 seconds or shaking time of 5 seconds between washes.
6. Add 50 u L Substrate A to each well.
7. Add 50 u L Substrate B to each well. Cover and incubate for 15 minutes at 20-25°C. (avoid sunlight)
8. Add 50 u L of Stop Solution to each well. Mix well.
9. Read the Optical Density (O.D.) at 450 nm using a microtiter plate reader immediately.

Result and discussion

The Fibrinogen level showed increased significant in patients (277.06 ± 11.73) when compared with control (58.05 ± 6.72) p-value <0.05 **,** this result was agree with many studies which prove that elevated fibrinogen level in patient with coronary heart disease (CHD) like Chinese study by Jung Wang *et.al* (2019)[5] were observed Elevated fibrinogen levels in patients who are at higher risk for CHD, such as those who smoke and have diabetes, hypertension, obesity, lipid metabolism disorders, menopause, and depression. In contrast, factors that reduce CHD risk, such as regular exercise, also reduce fbrinogen levels. Experimental studies have also suggested that fibrinogen and fbrin degradation products may increase coronary plaque vulnerability by stimulating coagulation, platelet aggregation, and vascular endothelial dysfunction and demonstrated that fibrinogen is correlated with atherosclerosis severity, another study *Ping Jiang et.al*.(2019) [6] observed correlation between fibrinogen levels and traditional factors in patients with coronary artery disease (CAD) were found Fibrinogen plays important roles in platelet aggregation, plasma viscosity, and fibrin formation. It is an acute-phase reactant that is increased in inflammatory states. In accordance with the hypothesis that inflammation plays an important role in plaque rupture and thrombosis, elevated fibrinogen levels have been identified in patients with unstable angina.

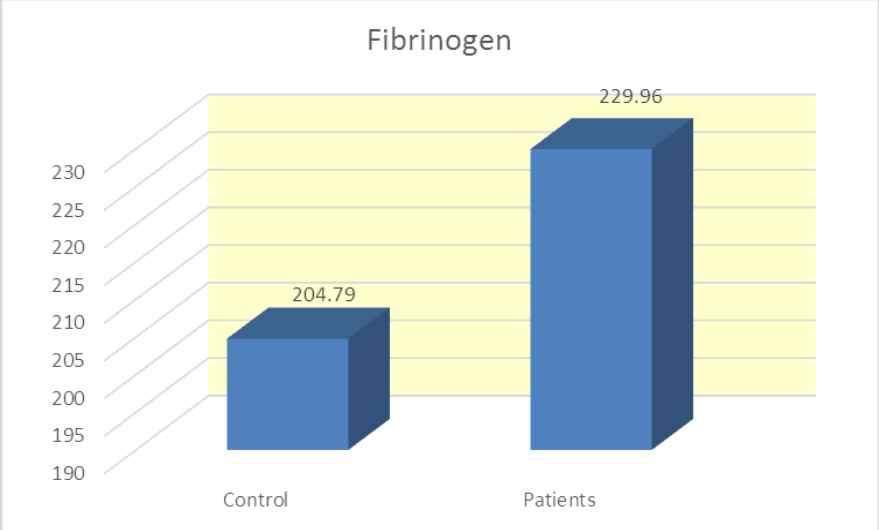

Figure (1) serum Fibrinogen level in study groups

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