



“PULMONARY EMBOLISM, ACUTE CORONARY SYNDROME, AND CHRONIC OBSTRUCTIVE PULMONARY DISEASE: A STUDY OF THE VARIATIONS OF D-DIMER IN VARIOUS CLINICAL CONDITIONS “

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Abstract

The use of D-dimer in the identification of acute aortic dissection, deep vein thrombosis, and pulmonary thromboembolism has been established [1]. The very first pathophysiological shifts in acute coronary syndrome, in line to a report, is an onset of coronary artery thrombus [2]. Plasmin lysis of cross-linked fibrin clots produces the enzymatic breakdown product known as D-dimer. For the purpose of determining the patient's fibrinolytic status, D-dimer concentration in plasma plays a key role. [3]. Risk stratification should be performed on pulmonary embolism patients to determine whether they may benefit from a D-dimer for advanced treatment, such as thrombolysis or embolectomy. D-dimer is a sign of endogenous fibrinolytic activity and is connected to the inflammatory process. Despite having a chronic inflammatory condition, chronic obstructive pulmonary disease (COPD) is characterized by a hypercoagulable state, especially D-dimer levels.

Keywords: D-dimer, Acute Coronary Syndrome, Pulmonary Embolism, Chronic Obstructive Pulmonary Disease, Thrombosis, Myocardial Infarction

INTRODUCTION

Cardiac biochemical markers, such as Troponin T or Heart type Fatty Acid Binding Protein H-FABP, are highly selective for myocardial injury, but not sensitive enough in the very early phase after the onset of Acute coronary syndrome (within a couple of hours after the onset).

Emergency departments (EDs) very often meet patients who have Acute Coronary Syndrome (ACS) [4]. Acute Myocardial Infarction (AMI) early diagnosis and quick rule out remain major concerns to mitigate mortality, morbidity, and the hospitalization costs and avoid executing further diagnostic and needless procedures in low-risk individuals [5]. Several hours after the onset of symptoms, cardiac enzyme levels begin to climb. A good sensitivity for the early detection of myocardial infarction (MI) has not been demonstrated for the ECG, however. [6] The cause of acute ischemic symptoms is coronary artery thrombosis. Fibrinogen, plasmin-2 antiplasmin, prothrombin, activated factor VII, and D-dimer are a few of the indicators that have been found to be involved in the formation and lysis of arterial thrombosis. It is anticipated that the level of incidence of coronary artery thrombosis will have an impact on these enzymes' values [7]. A few studies have recently shown the diagnostic utility of the D-dimer in the diagnosis of MI and ACS [8], however it was formerly utilized as a diagnostic marker for venous thromboembolism [9] and other conditions.

The agent a primary enzymatic breakdown outcome of plasmin-crosslinked fibrin is D-dimer. A single measurement may be sufficient to determine the fibrinolytic status because systemic values of D-dimer are a marker of the circulation's turnover of fibrin. Despite the usage of clinical norms, improper D-dimer testing stands a serious issue. Therefore, understanding the pathophysiological foundation and constraints of D-dimer testing is critical for emergency as well as intensive care physicians to ensure its appropriate usage in practice [10].

D-dimer concentrations were seen to stay high for several months following AMI, indicating that the patients with AMI had an improved coagulable condition [11]. Meanwhile, such a lifted coagulable state is connected to mortality and MI [12]. Thus, D-dimer as an indicator of coagulable state might be a useful biomarker in predicting cardiovascular ischemic events [13].

The amount of D-dimer in the blood rises in every physiological and clinical condition while plasmin has produced fibrin and has subsequently broken it down. Clinical circumstances that may raise D-dimer levels can cause chronic inflammatory disorders [14]. Systemic inflammation has been linked to Chronic Obstructive Pulmonary Disease (COPD), according to recent research [15], Pulmonary embolism (PE) is occurring more frequently in COPD patients who have recently experienced an acute exacerbation [16]. Furthermore, COPD has just been identified as a separate risk factor for PE [17].

Materials and Methods

A total of 60 patients with confirmed cases of acute coronary syndrome, pulmonary embolism, and chronic obstructive pulmonary disease (each with 20 patients) were sampled (10 patients each from the male and female groups). Those who have inflammation, malignancy, postsurgical treatment, liver disease, pregnancy, above 80 years of age and Computed Tomography Pulmonary Angiogram performed patients are excluded and above the age of 18 are included

2ml venous blood sample were collected by venipuncture in 3.2% sodium citrate vacutainer tube and the plasma is separated (within 60 minutes) by centrifugation at 3000 rpm for 5minutes. The dual monoclonal antibody (MAB) sandwich principle creates the foundation for the immunoassay known as the D-dimer Assay. With a gold sol particle label, the poly-streptavidin-biotin capture technique is put into use. The sample's D-dimer mixes with the antibodies attached to the gold sol particles to create a "sandwich" structure. The poly-streptavidin-biotin, which is immobilized in a line across the result window, then reacts with this. The speed and intensity of colour formation is related to the concentration of D-Dimer in the sample.

Observation and Result

Blood reports from 60 patients were collected, who are confirmed for acute coronary syndrome, pulmonary embolism, chronic obstructive pulmonary disease each with 20 patients, both male and female (10 patients each) respectively. The study was conducted by determining D-dimer in such patients.

D-DIMER AVERAGE VALUE IN ACUTE CORONARY SYNDROME (ACS)

The average mean D-dimer level in 20 patients with acute coronary syndrome

ACS	Normal value	Mean value
Total (20) patients	0-500 ng/ml	2749.5ng/ml

Gender wise distribution

Gender	Normal value	Mean value
Male (10) patients	0-500 ng/ml	3044.3 ng/ml
Female (10) patients	0-500 ng/ml	2454.7 ng/ml

AVERAGE VALUE OF D-DIMER IN PULMONARY EMBOLISM (PE)

The average mean value of D-dimer in 20 pulmonary embolism patients

PE	Normal value	Mean value
Total (20) patients	0-500 ng/ml	4290.75 ng/ml

Gender wise distribution

Gender	Normal value	Mean value
Male (10) patients	0-500 ng/ml	4628.7 ng/ml
Female (10) patients	0-500 ng/ml	3952.8 ng/ml

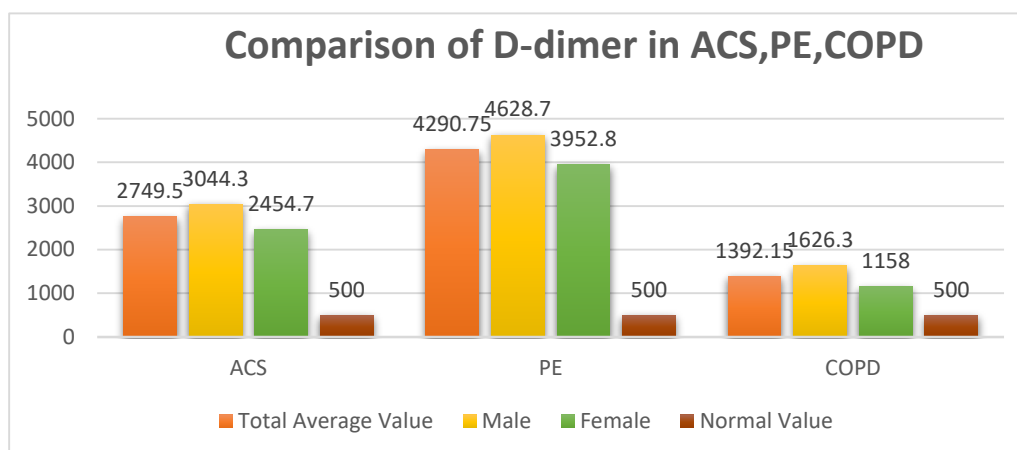
AVERAGE VALUE OF D-DIMER IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

20 people with chronic obstructive pulmonary disease's average mean D-dimer values

COPD	Normal value	Mean value
Total (20) patients	0-500 ng/ml	1392.15 ng/ml

Gender wise distribution

Gender	Normal value	Mean value
Male (10) patients	0-500 ng/ml	1626.3 ng/ml
Female (10) patients	0-500 ng/ml	1158 ng/ml



Comparison of D-dimer values in ACS, PE & COPD respectively

Discussion

As thrombus development is represented by the D-dimer level, it would be expected that in acute coronary syndrome, D-dimer level would rise earlier than cardiac biochemical indicators. In the very early period (within 2 hours) following the beginning of symptoms, D-dimer exhibited a higher positive test rate in ACS patients [18]. Ieko et al. [19] showed that D-dimer and plasmin-plasmin inhibitor complex levels in the acute phase of ACS were significantly higher than those in the recovery phase, suggesting intracoronary thrombosis. The D-dimer sensitivity and specificity for identifying ACS from non-ACS were stated as 95.4% and 83.7%, respectively, in Orak et al.'s study [20]. The D-dimer serum level in the case group with ACS was obtained at around 2.31-2.34. Furthermore, Tello-Montoliu's study [21] suggested employing an array of biomarkers, including D-dimer, for identifying ACS.

PE is the third most common acute cardiovascular disease, after myocardial infarction and stroke. The D-dimer test used in the current study appears to be an extremely useful in out-patients suspected of having PE: at D-dimer concentrations below a cut off of 500 $\mu\text{g/L}$, the assay provides for the safe exclusion of the disease in 30% of outpatients having an extremely low risk of thromboembolic events suspected of having it. Furthermore, in patients with a 50% or higher clinical probability of developing PE, D-dimer concentrations above a crucial level of 4,000 g/L may be suggestive of the condition. Bayes' rule [22] states that a patient with a 50% clinical likelihood of PE and a patient with an 80% clinical probability of PE would both have a probability of PE of 88% and 97%, respectively, with a D-dimer level above 4,000 g/L . According to Crawford et al.'s meta-analysis [23], the D-dimer's estimated sensitivity for PE was between 80 and 100%, but its specificity varied between 2 and 63%. D-dimer might be employed as the ideal PE screening method, according to Schutgens et al. [24], who discovered that due to its great sensitivity, PE could be excluded in the event of a negative result.

A sign of endogenous fibrinolytic activity, D-dimer is linked to the inflammatory reaction. The outcomes of this study indicate that D-dimer levels spike in COPD patients and suggest that this inflammatory marker may be used to monitor inflammation as COPD progresses. All COPD patients hospitalized due to an acute exacerbation should be assessed for the existence of Venous Thromboembolic Events (VTE), according to a proposal made by Günen et al. [25]. Due to the same presenting signs and symptoms of PE and other reasons of COPD exacerbation, it is hard to differentiate between the two. D-dimer testing is therefore more significant in ruling out the PE diagnosis in these patients. Sohne et al. demonstrated that low clinical likelihood and a normal D-dimer level provided comparable safety in excluding PE in COPD patients [26]. According to Akgün et al.'s study [27], patients with COPD exacerbation with VTE had higher D-dimer levels than COPD patients without VTE.

The present study shows the importance of D-dimer in various clinical circumstances. Acute coronary syndrome, pulmonary embolism, chronic obstructive pulmonary disease, pregnancy, malignancy, diffused intravascular coagulation, and other disorders are among those that cause an elevation in D-dimer. In our research, it was determined that people with acute coronary syndrome (ACS), pulmonary embolism (PE), and chronic obstructive pulmonary disease (COPD) had higher serum D-dimer levels.

Conclusion

The present study shows a single measurement may be sufficient to determine the fibrinolytic state based on systemic values of D-dimer as an indicator of fibrin turnover in the circulation. The study included 3 pathological and one non pathological condition to analyse the variations in D-dimer. D-dimer and coronary heart disease incidence or recurrence are significantly correlated. The value of plasma D-dimer could be an effective indicator of acute coronary syndrome. The inflammatory marker D-dimer is elevated in COPD patients, and it may be used to assess the role of inflammation in the development of the disease.

The prognosis of patients could be improved by the detection of plasma D-dimer levels, which could prevent unnecessary tests and make it easier to investigate PE treatments early on. The D-dimer test, which measures blood clotting activity, can be used to rule out a diagnosis or increase diagnostic yield in a variety of medical disorders in less time and at a lower cost. In conclusion, measuring the plasma D-dimer level may be a quick, easy, and affordable way to rule out ACS, PE, and COPD.

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